magnus
Group Share, Receive & Grow !

5th *Edition of International Conference on*

NEUROLOGY AND
NEUROLOGICAL DISORDERS

VIRTUAL EVENT

JUNE 2022

15-16

BOOK OF
ABSTRACTS

5TH EDITION OF
INTERNATIONAL CONFERENCE ON

NEUROLOGY AND
NEUROLOGICAL DISORDERS

15-16 JUNE

INDEX

Contents

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 NEUROLOGY 2022

Magnus Group is pleased to invite you to participate in the '5th Edition of International Conference on Neurology and Neurological Disorders' (Neurology 2022) Virtual Event during June 15-16, 2022 with the theme "Sensing New Horizons and Bridging Synaptic Gaps in Neurology" following the victorious completion of four editions.

This Neurology 2022 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.

We are confident that our conference will provide you with an incredible chance to explore new horizons in your field and we hope to see you at our upcoming INBC 2022 conference during October 24-26, 2022.

KEYNOTE FORUM

DAY 01

5TH EDITION OF
INTERNATIONAL CONFERENCE ON

NEUROLOGY AND NEUROLOGICAL DISORDERS

15-16 JUNE

Ken Ware

Founder and head of research and development at Neurotricional Sciences and NeuroPhysics Therapy Institute, Australia

Exploring the psychophysical phenomena; Fibromyalgia, Myalgic Encephalomyelitis/chronic fatigue syndrome (ME), Postural Orthostatic Tachycardia Syndrome (POTS) and introducing novel approaches for effective neurophysics treatment for these physical and mental health compromising conditions

There is an ever-increasing number of people around the world who reportedly suffer from one or more of the psychophysical conditions Fibromyalgia, Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME), Postural orthostatic tachycardia syndrome (POTS). In the USA there are presently more than 5 million suffers of Fibromyalgia and in the Netherlands, there are more than 400,000. 80 to 90% of suffers are women. People suffering from the said psychophysical conditions commonly complain of other concerning debilitating conditions. For example, of the 400,000 Dutch people suffer from fibromyalgia, 70% suffer from sleep problems, 50% have difficulties with walking and social activities, and 55% suffer from depression. The present global medical consensus states that the cause of these conditions is unknown, but risk factors include traumatic injury, rheumatoid arthritis and other autoimmune disorders, such as lupus, and genetic factors. There is no cure, but medications, exercise, acupuncture, and behavioural therapy can help relieve symptoms and improve sleep quality.

NeuroPhysics Therapy (NPT) has proven to be a highly effective self-initiated treatment for the said psychophysical conditions. This presentation reviews various patient outcomes and delves into the novel approaches and rationale employed, and describing the new lens used to view these conditions through. The general understanding is that 'the human' complex adaptive system can be extremely sensitive to initial conditions. Through the development of specialized techniques that perturb to expose rogue sensitivities to initial conditions in a controlled environment and the underlying philosophy of NPT being to treat the person not the disorder, the patient is able to learn new control skills over how their systems are perceiving and responding to their environments. This is a top-down bottom up, perception, action, cognition approach that goes beyond simply assisting to manage the symptoms associated with these conditions but to enable patients to move on and live a symptom free life that they have control and authority over.

Keywords: Fibromyalgia; ME; POTS; NeuroPhysics Therapy; Complexity; Chaos Theory; Perception; Psychophysical;

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 Bodybuilding 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.

Wagih Shafik El Masri[1,2]

[1]Hon. Clinical Professor of Spinal Injuries Keele University, United Kingdom

[2]Emeritus Consultant Surgeon in Spinal Injuries, Midland Centre for Spinal Injuries, The Robert Jones & Agnes Hunt Orthopaedic Hospital, Oswestry

Spontaneous Neurological Recovery (SNR) following Traumatic Spinal Cord Injuries (TSCI)

Prior to the 2nd world war and for thousands of years TSCI challenged, frustrated and defeated the medical profession and the majority of patients died from a range of complications within two years of injury.

During the 2nd world war Ludwig Guttmann (Neurosurgeon) in the UK demonstrated that patients who undergo simultaneous Active Physiological Conservative Management (APCM) of the injured spine of patients with paralysis/ paresis, multi-system physiological impairment and malfunction and the range of psycho-social problems from the first few hours or days of injury can not only survive and live long, healthy, enjoyable, dignified productive and often competitive but also many recover neurologically spontaneously without surgical, cellular of or other interventions on the spine or the spinal cord. He also observed that this SNR is achieved irrespective of the radiological presentation of the spinal injury on Xrays.

Frankel et al in 1965 studied the SNR in 612 patients with cord damage admitted to Stoke Mandeville Hospital within 15 days from injury and treated with APCM. They published these findings in 1969 having observed that about 60% of patients who present with long sensory tract sparing (without motor sparing), 70% of those who retain pin prick sensation and over 75% of patients presenting with sensory-motor sparing (however insignificant the motor power is clinically) will recover motor function and many are able to stand and walk without any intervention and irrespective of the radiological presentation on Xrays. These outcomes have been repeatedly and consistently confirmed and published over the last century since.

The development of CT & MRI scan encouraged reliance on the radiological appearances to promote surgery based on an unproven concept in humans and a strong belief extrapolated from the laboratory animal that there is a "window of opportunity" during which surgery and in particular decompression can halt many of the detrimental 2ry detrimental cellular and cell membrane disturbances; vascular, chemical, metabolic, inflammatory and enzymatic changes and possibly disruption of blood the brain barrier and the loss of auto- regulatory functions in the injured cord to improve neurological outcomes. This window was found to be four hours from injury in laboratory animal during which was impossible to test in humans. A number of well-advertised studies in an attempt to demonstrate that decompression of the cord within 24 hours of injury yields numerically better outcomes than late surgery were published initially suggesting and subsequently claiming that early surgical decompression should become the Standard of Care in the management of TSCI.

Unfortunately the literature on SNR and its prognostic indicators was ignored; no attempt was made to explore or demonstrate equality never mind superiority of early or late surgical outcomes over those of APCM or entertain the possibility that the described improvement following early surgery only reflects the SNR or could indeed be inferior to the SNR that achieved with APCM. I will discuss the Rationale and outcomes of APCM and Surgical Management, the predictors, positive and negative factors that can influence SNR and the possible interpretations of the current findings in the literature.

Audience Takeaway

- Become familiar with the extent of spontaneous neurological recovery and its clinical predictors following TSCI with or without surgical, cellular, hormonal or other intervention on the injured spine or spinal cord.

- Become familiar with the current clinical controversy in managing the injured spine with spinal cord injury.

- Enable you to better assess the literature on neurological & functional outcomes of management.

- Highlight the limitations of the currently promoted method of assessment and documentation of the neurological effects of TSCI and reflect on the validity of current claims of added value of interventions.

- Encourage you to be involved as an independent neurological assessor and interpreter of outcome studies.

Biography

WSEM was accredited in Traumatic Spinal Injuries (TSI) following training in this field and its allied surgical specialties at Stoke Mandeville, Oxford, Guys Hospitals & the USA between 1971&1982, appointed Director of the Spinal Injury Centre (44 beds) in the RJAH Orthopaedic Hospital-Oswestry between 1983 & 2014. He treated 10,000 patients with cord damage and took full responsibility of 3000 patients from the first few hours/days of injury to end of life. He published over 146 manuscript, lectured worldwide and received National & International Awards and Recognition from a range of Institutions including the House of Lords in the UK.

Stephen Grossberg

Boston University, United States

Towards solving the hard problem of consciousness : The varieties of brain resonances and the conscious experiences that they support

The hard problem of consciousness is the problem of explaining how we experience qualia or phenomenal experiences, such as seeing, hearing, and feeling, and knowing what they are. To solve this problem, a theory of consciousness needs to link brain to mind by modeling how emergent properties of several brain mechanisms interacting together embody detailed properties of individual conscious psychological experiences. This article summarizes evidence that Adaptive Resonance Theory, or ART, accomplishes this goal. ART is a cognitive and neural theory of how advanced brains autonomously learn to attend, recognize, and predict objects and events in a changing world. ART has predicted that "all conscious states are resonant states" as part of its specification of mechanistic links between processes of consciousness, learning, expectation, attention, resonance, and synchrony. It hereby provides functional and mechanistic explanations of data ranging from individual spikes and their synchronization to the dynamics of conscious perceptual, cognitive, and cognitive-emotional experiences.

ART has reached sufficient maturity to begin classifying the brain resonances that support conscious experiences of seeing, hearing, feeling, and knowing. Psychological and neurobiological data in both normal individuals and clinical patients are clarified by this classification. This analysis also explains why not all resonances become conscious, and why not all brain dynamics are resonant. The global organization of the brain into computationally complementary cortical processing streams (complementary computing), and the organization of the cerebral cortex into characteristic layers of cells (laminar computing), figure prominently in these explanations of conscious and unconscious processes. Alternative models of consciousness are also discussed.

Keywords: Adaptive resonance; Attention; Audition; Consciousness; Emotion; Vision

Biography

Grossberg is a founder of the fields of computational neuroscience, connectionist cognitive science, and neuromorphic technology. His work focuses upon the design principles and mechanisms that enable the behavior of individuals, or machines, to adapt autonomously in real time to unexpected environmental challenges. This research has included neural models of vision and image processing; object, scene, and event learning, pattern recognition, and search; audition, speech and language; cognitive information processing and planning; reinforcement learning and cognitive-emotional interactions; autonomous navigation; adaptive sensory-motor control and robotics; self-organizing neurodynamics; and mental disorders. Grossberg also collaborates with experimentalists to design experiments that test theoretical predictions and fill in conceptually important gaps in the experimental literature, carries out analyses of the mathematical dynamics of neural systems, and transfers biological neural models to applications in engineering and technology. He has published seventeen books or journal special issues, over 500 research articles, and has seven patents.

SPEAKERS
DAY 01

5TH EDITION OF
INTERNATIONAL CONFERENCE ON

NEUROLOGY AND
NEUROLOGICAL DISORDERS

15–16 JUNE

Chonghe Jiang*, Jiangwen Zeng

Department of Urology, The Sixth Affiliated Hospital of Guangzhou Medical University, China

The bladder cooling reflex and its clinical application

Objects: The ice-water test is a simple supplementary urodynamic test that is useful in differenting neurogenic from non-neurogenic bladder dysfunctions. Bors and Blinn some 50 years ago reported that rapid filling of the bladder with a small volume of ice water caused an immediate reflex detrusor contraction in patients with spinal upper motor neuron lesion but no response in those with lower motor neuron lesion or in neurologically normal subjects. A similar bladder cooling reflex has recently been characterised in the cat and was found to originate from specific cold receptors in the bladder wall. This presentation is a review of the physiology of the bladder cooling reflex and its clinical applications.

Materials and Methods: Adult cats under alpha-chloralose anesthesia were used for experiments. Two thin catheters were inserted into the bladder through a slit in the proximal urethra, one for bladder pressure recording, the other for bladder infusions of saline at different temperatures. Infusion evoked afferent and efferent nerve activity was recorded simultaneously from pelvic nerve branches to the bladder.

Clinically, the bladder cooling test was applied as a complement to routine cystometry both in children and adults. The outcome was evaluated retrospectively by members of our group. A total of 284 children with nonneurogenic and 108 with known or suspected neurogenic bladder dysfunction, ranging in age from 1 month to 18 years, were included together with 557 adults with overactive bladders, lower motor neuron lesions or pure stress incontinence. In adults, the bladder cooling test was performed as a rapid manual infusion of 100 ml cold saline (3 - 10O C), while for children about half the cystometric capacity was used after pretesting with an equal volume of body-warm fluid. The test was considered positive if the fluid was expelled from the bladder within 1 min or if a detrusor contraction greater than 30 cm H_2O was evoked by the cold but not warm fluid.

Results: In the cat, reflex detrusor contractions were more readily evoked by infusions of cold than body-warm saline into the bladder. The threshold volume was lower and the efferent activity poorly correlated to the mechanoreceptor response showing prolonged after activity on bladder unloading. The cooling reflex was greatly exaggerated by intraluminal exposure of the bladder or the urethra to menthol (0.6 mM), which shifted the temperature-response curve towards higher temperatures. Afferent recordings demonstrated that cooling or menthol exposure activated a subgroup of unmyelinated C-afferents in the bladder pelvic nerve. Electrical stimulation at appropriate intensity revealed a corresponding bladder C-fibre reflex that survived an acute spinalization at a low thoracic level. Thus, the bladder cooling reflex is mediated by a segmental spinal pathway, partly separate from the ordinary micturition reflex.

Clinical studies in young children revealed that the bladder cooling test is positive during the first 2 years of life but typically becomes negative when the child gains voluntary control of its bladder. In neurologically intact adults the test is negative. It was positive in more than 90 % of patients with complete or incomplete upper motor neuron lesions and in about 75% of patients with multiple sclerosis, Parkinson's disease or previous cerebrovascular accident resulting in bladder hyperreflexia. Neither infusion speed nor infused volume was critical for the outcome of the test in such patients, provided that the bladder wall was sufficiently cooled. Thus, in adults, a positive test indicates overt or occult neuropathy.

Conclusions: The urinary bladder is equipped with a reflex system originating from cold receptors in the bladder wall and having a reflex pathway partly separate from that of the normal micturition reflex. The human bladder cooling reflex is in principle organized in the same way as that of laboratory animals. A positive bladder cooling test is an infant reflex response that becomes suppressed by descending signals from higher centers during maturation of the central nervous

system. Excluding the age of factor, a positive test in patients with voiding dysfunction is an indicator of a latent or overt neurological disorder.

The bladder cooling test ("ice water test") is a rapid and simple test, easy to interpret and well tolerated by patients. The test increases the precision of urodynamic evaluations.

Objects: The ice-water test is a simple supplementary urodynamic test that is useful in differing neurogenic from non-neurogenic bladder dysfunctions. Bors and Blinn some 50 years ago reported that rapid filling of the bladder with a small volume of ice water caused an immediate reflex detrusor contraction in patients with spinal upper motor neuron lesion but no response in those with lower motor neuron lesion or in neurologically normal subjects. A similar bladder cooling reflex has recently been characte

**Audience Takeaway**

To learn bladder colling reflex:

• What´s the triggering stimlus?   Bladder tension, or bladder cooling?

• Where are the bladder cooling receptors? Bladder and urethral walls, or structures around the bladder and urethra?

• What type of receptors are involved? Ad fiber or C fiber cold receptors, or nociceptors (pain receptors)?

The benefits of bladder cooling test (BCT):

• The BCT is a simple and rapid add-on test.

• Its outcome is easy to interpret.

• It is well tolerated by patients.

• The underlying physiology is reasonably well known.

• It can in doubtful cases differentiate upper from lower motor neuron lesions.

• It can varify a neurogenic etiology in certain types of urge incontinence.

Biography

Dr. Chonghe Jiang, MD, PhD, first research engineer of Linkoping University, Sweden, now is working in the Sixth Affiliated Hospital of Guangzhou Medical University, China, as a professor, urologist and director of Kidney Center. He is major in research work on voiding dysfunctions, and clinical work on urinary continence. All the publications are involved in neuroncontrol of lower urinary tract. Identifying and clarifying the bladder cooling reflex and applying neuromodulatory technique in treatment of urinary incontinence by using electrical stimulation are main contributions in this area.
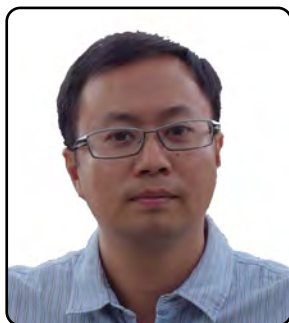
Liang Zhu[1,2], Mengqi Wang[1], Yin Liu[1], Weijie Zhang[2], Hequn Zhang[1], Anna Wang Roe[1], Wang Xi[1]*

Interdisciplinary Institute of Neuroscience and Technology (ZIINT), Zhejiang University, China

Single microvessel occlusion technology (PLP) produces lamina-specific microvascular flow effects and neuronal degeneration

There is increasing recognition that dysfunctions of microvasculature in the brain may underlie neurodegenerative disease. To understand the functional architecture of cortical microvasculature, we developed a highly precise photothrombosis method (ULPT) to occlude single microcapillaries in distinct cortical layers, and then quantitatively studied effects on vasodynamics in the upstream and downstream branchlets of the micro-occlusion. Our results showed that such occlusion led to rapid regional flow redistribution and local downstream BBB leakage, as well as dramatic changes in neuronal dendritic architecture and neuronal degeneration. Interestingly, compared to layer 4, layers 2/3 exhibited significantly less flow redistribution, suggesting cortical feedback recipient layers may be more susceptible to micro-occlusion damage. This new precision model of microvascular ischemia revealed, for the first time, laminar distinctions in micro-infarct response, and raised the possibility that relatively greater impact on on micro-capillary function contribute to cognitive decline in neurodegenerative disease.

Audience Takeaway

This work provides novel and quantitative understanding of microvascular function in neurodegenerative disease:

- Novel precise micro-occlusion method: For the first time, we established single microvessel occlusion for depths up to 815µm in cortex and a method to study layer-specific microvascular flow.
- Single neurons are maintained by a few (1-3) local capillaries: Using this method, we show in vivo that occlusion of a few (2-3) neighboring capillaries leads to severe neurodegeneration.
- Laminar-specific flow effects: Vascular flow in laminae 2/3 (feedback recipient layers) appears more susceptible to micro-occlusion, suggesting a novel microvascular basis for feedback-related cognitive decline in neurovascular disease.

Biography

Dr. Xi Wang studied Biotechnology at Nanjing University as BS in 2001. He then joined the research group of Prof. Aike Guo at the institute of Neuroscience in CAS. He received his PhD degree in 2008. After postdoctoral fellowship supervised by Prof. ZhangHong and Duan Shumin at Zhejiang University. He obtained the position of an associate Professor at ZIINT. He published more than 20 research papers in many high impact journals.

Pengxu Wei

National Research Center for Rehabilitation Technical Aids, China

Ultra-early screening of slight cognitive decline

Adults with the risk of dementia may present reduced cognitive performance several decades before the typical onset of Alzheimer's Disease (AD). It is necessary to detect slight cognitive decline in an early stage for timely initiating active interventions. The slight cognitive decline, called the Transitional Cognitive Decline (TCD) defined in the Alzheimer's continuum in 2018 NIA-AA Research Framework, may be a better time window than Mild Cognitive Impairment (MCI) for early intervention of subjects with Alzheimer's Disease (AD) risks. The slight cognitive decline may involve memory and other cognitive domains which Subjective Cognitive Decline (SCD) tests cannot assess. Additionally, SCD tests are better to be corroborated by objective cognitive testing. Here, a rapid performance-based screening instrument (the Quick-Cog test) for detecting TCD is introduced.

The Quick-Cog test assesses different cognitive domains, including language, orientation in time and space, memory, calculations, attention, conceptual thinking, visuospatial ability, executive function, and processing speed. The Quick-Cog test is designed to avoid celling effects when assessing individuals presenting "normal" performance with an objective cognitive test such as the Montreal Cognitive Assessment (MoCA). The Quick-Cog test includes several simple questions to avoid floor effects. If serial tests were performed, the performance of each time may be compared to show changes in cognitive function of the subject. When examining healthy college students and community-dwelling elderly people living independently with normal instrumental activities of daily living scores, the Quick-Cog, SCD-9, and MoCA tests were compared the elderly presented greater decline in Quick-Cog scores than those of MoCA and SCD-9 in comparison with the young students. This result indicates that the Quick-Cog test may be used as a sensitive instrument for ultra-early screening of slight cognitive decline.

Audience Takeaway

An object cognitive test for the stage Transitional Cognitive Decline is lacking:

- The presentation introduces a practical instrument for ultra-early screening of slight cognitive decline.
- The introduced instrument appears to be more sensitive than existed, commonly-used tools.

Biography

Pengxu Wei, M.D., Ph.D., Director of Integrative rehabilitation department, National Research Centre for Rehabilitation Technical Aids; General secretary and executive director of Alzheimer's Disease & Cognitive Rehabilitation Committee, China Association Rehabilitation Medicine; Principal investigator of a National Key Research and Development Program of China; Leader of a National CME project; Leading talent of Beijing E-town.

Ming Zhang

North Sichuan Medical College, China

Intestinal flora composition determines microglia activation and improves epileptic

In response to environmental stimuli, immune memory mediates the plasticity of myeloid cells. Immune training and immune tolerance are two aspects of plasticity. Microglia that are immunologically trained or immunologically tolerant are endowed with a tendency to differentiate into alternative dominant phenotypes (M1/M2). Male C57BL/6 mice (immune-training group, immune-tolerant group, and control group) were used to establish the kainic acid epilepsy model. The seizure grade, duration, latency, hippocampal potential, and energy density were used to evaluate seizures, and the changes in the polarization of microglia were detected by western blot. 16S rDNA sequencing showed that the abundance of Ruminococcus in the immune-tolerant group was the dominant flora. Our research connections Intestinal microorganisms, brain immune status, and epilepsy behavior together. Pro-inflammatory M1 phenotype and anti-inflammatory M2 phenotype mediate and enhance and suppress subsequent inflammation, respectively. We conclude that intestinal microorganisms influence the occurrence and development of epilepsy by regulating the polarization of microglia.

Audience Takeaway

- Immune training and immune tolerance models have been experimented on cerebral ischemia and Alzheimer's disease models. However, a KA epilepsy mouse model have not been established. Furthermore, this is the first study to investigate the role of the intestinal flora in immune tolerance.

- In this study, 16S rDNA sequence analysis was used to identify the key flora of immune tolerance, we investigated whether intestinal microflora can regulate microglial phenotype activation and affect epileptic seizures after immune state change.

- This study provides novel insights on intestinal flora regulation through microglial cell phenotype transition and contributes to the development of new clinical interventions in epilepsy.

Biography

Ming Zhang studied in Southwest Medical University, 3 years of Standardized resident training in West China Hospital, Sichuan University, now a graduate student at North Sichuan Medical College, under the guidance of her mentor Dr. Guohui Jiang, mainly engaged in epilepsy and cognition.
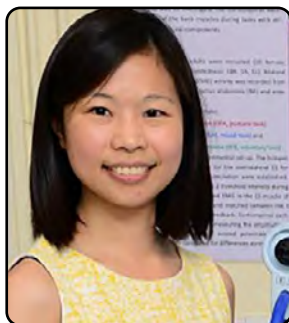
Emma Alexander[1], Eduardo Martinez-Valdes[1], Joy Roy Chowdhury[2], Joeri van Helden[1], Shin-Yi Chloe Chiou[1,3]*

[1]School of Sport, Exercise and Rehabilitation Sciences, University of Birmingham, United Kingdom

[2]The Robert Jones and Agnes Hunt Orthopaedic Hospital NHS Foundation Trust, United Kingdom

[3]The Centre for Trauma Sciences Research, University of Birmingham, United Kingdom

The effects of arm exercise on trunk function in individuals with spinal cord injury

Objectives: Many people living with Spinal Cord Injury (SCI) exhibit reduced trunk control which can severely compromise the use of upper and lower extremities, hindering functional recovery. Current rehabilitation for improving trunk function remains complex and time-consuming, highlighting a need to develop new rehabilitation interventions that are simple and low-cost. It is well accepted that increasing the excitability of neural pathways can improve function of that muscle in patients with neurological disorders. Numerous studies have reported a voluntary contraction of muscles with one arm increasing corticospinal excitability of the contralateral resting arm, a phenomenon known as crossed facilitation. We previously showed that voluntary contractions of upper-limb muscles facilitated corticospinal excitability of trunk muscles in healthy adults and in adults with SCI. However, it remains unclear the therapeutic effects of arm exercise on trunk function in individuals with SCI. Hence, the study aimed to investigate the effects of a six-week, home-based, arm crank exercise programme on motor control of the trunk in individuals with chronic SCI.

Methods: Participants with chronic, incomplete cervical or high thoracic SCI patients (mean age±SD: 53±15 years; cervical SCI; 9) undertook a home-based arm crank exercise programme. The programme consisted of 30 minutes of exercise for five consecutive days, for six weeks. Corticospinal excitability of Erector Spinae (ES) muscles were assessed prior to and following the exercise programme using Transcranial Magnetic Stimulation (TMS). Motor Evoked Potentials (MEPs) of the ES induced by TMS were recorded using Electromyography (EMG). Neuromuscular control of the ES muscles was evaluated using high-density surface EMG (HDsEMG) during multidirectional reaching tasks. Amplitudes of the activity and entropy were analysed. Reaching distance, trunk displacement, and centre of pressure were recorded using a 3-D motion capture system and a force plate.

Results: We found amplitudes of ES MEPs increased 50%, 25% and 35% with respect to the baseline after 10, 20, and 30 minutes post exercise. After 6 weeks of the intervention, activity of the bilateral ES muscles during the reaching tasks also increased, suggesting an improvement in volitional control of the ES muscles. In addition, there was an increase in entropy in the bilateral ES muscles compared to baseline during reaching, suggesting improved efficiency and spatial homogeneity of muscle recruitment. There was also improvement in reaching distance, indicating improved dynamic sitting balance after the intervention.

Conclusions: We demonstrated that a six-week, home-based arm crank exercise programme can improve trunk function in people with chronic, incomplete SCI. Our findings provide a simple and affordable rehabilitation method for restoring trunk function after SCI.

Audience Takeaway

- The concept of crossed facilitation or crossed education and its clinical application in rehabilitation.
- Methodology of neurophysiology in humans in providing objective measurements for quantifying the effects of rehabilitation.
- The intervention discussed in this presentation is simple and affordable, increasing the accessibility of equipment to patients.
- The intervention discussed in this presentation is patient-initiated exercise and does not increase workload of healthcare professionals, providing patients an option that may help them recover.

Biography

Dr. Chiou qualified as a physiotherapist in 2007 and received her PhD in Physiotherapy from National Yang-Ming University, Taiwan, in 2013. She attended Imperial College London as a post-doctoral research associate at the MSk laboratory until April 2018. She became a lecturer in the School of Sport, Exercise, and Rehabilitation Sciences at University of Birmingham in May 2018. Her research utilizes neurophysiological measures to investigate the control of movement and how it is affected or adapted following injuries. Her research interests also include improving the effectiveness of therapeutic exercises using neuromodulatory strategies.

M.ª João Cabral Sacadura*, Helena Neves Almeida

Faculty of Psychology and Education Sciences, University of Coimbra, Portugal

Catholic University of Portugal and University of Coimbra, Portugal

Psychosocial dimensions on adherence to treatment regime in hemodialysis : A crossover between social work and neurology

The aim of the present communication is to share some results of the data analysis of a PhD Social Work investigation in progress in Portugal, that focus the determinant factors of adherence to treatment regime in the context of end-stage renal disease. In the field of chronic and epidemic diseases, where chronic kidney disease is included, is possible to identify many psychosocial dimensions associated with these disease and neurological complications that can interfere with daily lives of the patients.

Scientific research points that there are links between the impairments of the kidney and cognitive decline and cerebrovascular diseases. The brain and the kidney share common and traditional risk factors, like hypertension and diabetes, which can lead to vascular injury. Social factors in a chronic disease, can determinate the progression of a illness and influence adherence to treatment. The social health determinants and the social conditions of disadvantage can compromise and cause health impacts in the lifespan of people and maxime psychosocial problems. The non-adherence to a treatment regime, is considered a public health problem and an important issue in chronic kidney disease but also in other diseases with long therapies, because of the impacts in the health system and in the suboptimal results of the treatments in the patients.

Social Work is a practice-based profession and an academic discipline that promotes social change and development (ISFW, 2014) where the interdisciplinary knowledge contributions play a role in the development of a kaleidoscopic vision of the social reality. Social workers are the professional that in the health field contributes to the multidisciplinary team objectives, with the social assessment for the biopsychosocial approach, evaluate the person-in-context and promotes the adherence to treatment regime. For social work the knowledge of cerebral and nervous systems foundations contributes for a better understanding of social problems that social workers deal in every day, and with chronic diseases linked with brain disorders. In this paper we entend to analyse psychosocial dimensions on these field.

Audience Takeaway

- To give an applied perspective of psychosocial determinants in the social phenomena of adherence to treatment in the context of end-stage renal disease.

- To reflect on how these contributions may or may not inform the practice of social intervention with people on a hemodialysis program.

- The analysis will entail a correlation between the Social Work and neurology in end-stage renal disease in adherence to treatment regime.

Biography

Gratuate and PhD student in Social Work. Worked at Fresenius Medical Care Portugal (FMC), both as a Director and Social Worker and had been a Member of the Ethics Committee of FMC Portugal - NephroCare. 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.

Sabine J. Bischoff*, Astrid Enkelmann

Animal Welfare Unit, Jena University Hospital, Germany

CIRS-LAS - outstandingly improve quality of preclinical studies and animal welfare

Preclinical studies are an important step on the way to new knowledge in the fields of neurology. Animal experiments are often used to validate newly developed drugs or to gain more knowledge about neurologic disorders. The use of laboratory animals is indispensable in this field of complex neurological research models and at the same time represents a great risk for the animals. In addition, the repetition of animal experiments is very time-consuming and cost-intensive. In preclinical trials, there are often a large number of animals that die unexpectedly or show an unexpected clinical course. These results are also an important step on the way to new insights. Transparent handling of these unforeseen and critical events is enormously important.

The CIRS-LAS portal offers the possibility for every person working with laboratory animals to deal with these events transparently and openly, without fear of consequences. On CIRS-LAS.de, critical events and incidents can be entered anonymously if desired. A database offers the possibility to search for further cases concerning one's own work. The database search as well as the exchange about the cases is reserved for registered users. This ensures a safe environment to discuss errors or serious critical events that may occur in preclinical trials, especially with large animals.

Your contribution to CIRS-LAS leads to an exchange of experiences - so everyone can learn from each other and e.g. failed trials are not repeated. At the same time, the exchange and open discussion can contribute to safe and successful preclinical studies. By actively contributing to CIRS-LAS.de, everyone involved in animal experimentation can make a contribution to more animal welfare, fewer laboratory animals and a transparent approach to animal experimentation!

Audience Takeaway

- Everybody who is working with laboratory animals can help to increase animal welfare and transparency in laboratory animal science by participation at CIRS-LAS.
- We all learn from negative results or critical incidents if we speak about it!
- Reporting an incident takes only a few minutes but helps to think about critical events and is an important way to achieve a good error culture.
- Research in the CIRS-LAS database can save money and time for further experiments.
- Use of the CIRS-LAS database supports the progress of science.

Biography

Dr. Sabine J. Bischoff studied veterinary medicine at Leipzig University, Germany and graduated as veterinarian in 2003. She then worked with Dr. D. Barnewitz at the Biotechnology Research Center (fzmb) and has since been involved with several animal models of intensive care and neurological research. She received her Dr. Degree in 2008 and graduated as expert in laboratory animal science in 2013. Since 2017 she obtained the position of head of Animal Welfare Unit at University Hospital Jena. She and her research group have published several research articles in neurological preclinical studies about cerebral circulation.

Rocío Fernández-Serra[1,2,3,8]*, **Emma Martínez-Alonso**[4], **Alberto Alcázar**[4], **Mourad Chioua**[5], **José Marco-Contelles**[5], **Ricardo Martínez-Murillo**[6], **Milagros Ramos**[1,2,7], **Gustavo V. Guinea**[1,3,7,8,9] and **Daniel González-Nieto**[1,2,7,8]

[1]Center for Biomedical Technology, Universidad Politécnica de Madrid, Spain
[2]Departamento de Tecnología Fotónica y Bioingeniería, Universidad Politécnica de Madrid, Spain
[3]Departamento de Ciencia de Materiales, Universidad Politécnica de Madrid, Spain
[4]Department of Research, Hospital Universitario Ramón y Cajal, Spain
[5]Laboratory of Medicinal Chemistry, Institute of General Organic Chemistry (CSIC), Spain
[6]Department of Translational Neuroscience, Instituto Cajal (CSIC), Spain
[7]Biomedical Research Networking Center in Bioengineering Biomaterials and Nanomedicine (CIBER-BBN), Spain
[8]Silk Biomed SL, Spain
[9]Biomaterials and Regenerative Medicine Group, Spain

2-aminoethoxydiphenyl borate neuroprotects the stroke brain through shortening of peri - infarct depolarizations and enhanced perfusion in the ischemic penumbra

Ischemic stroke is the cause of high number of deaths and disability-adjusted life years among older adults, with an increasing prevalence due to demographic changes. Secondary mechanisms of injury associated to stroke contribute to exacerbate the damage, thus enhancing the neurological deficits. The lack of clinical translatability of many neuroprotective strategies against secondary damage that were successful during preclinical validation, predisposes to the identification of new molecular targets and molecules. In this study, we examined the neuroprotective properties of 2-aminoethoxydiphenyl borate (2-APB), an antagonizer of several channels and receptors involved in calcium dynamics. This molecule was assessed in non-consanguineous stroke mice that might better model the diversity of human population. After permanent cerebral ischemia induced by irreversible ligation of the middle cerebral artery, 2-APB reduced the size of the damage and preserved the functionality of the somatosensory cortex evaluated by somatosensory evoked potentials, which translated in better behavioral performance. While in this permanent ischemia model the neuroprotective effect exerted by the antioxidant scavenger cholesteronitrone F2 was associated with a reduction of Reactive Oxygen Species (ROS) and better neuronal survival in the penumbra, 2-APB did not modify the inflammatory response or decrease the content of ROS, and its neuroprotective effect was mostly related with a shortening of peri-infarct depolarizations and higher residual cerebral blood flow in the penumbra. Our study points out the potential of 2-APB to target spreading depolarization events and the associated inverse hemodynamic response and contributes for the searching of alternative directions to drive neuroprotection after stroke.

Audience Takeaway

- The relevance of the election of the mouse strain used for research on stroke. Non-consanguineous specimens develop further variability compared to inbred animals, but might mimic better the diversity of human population.

- The importance of the selection of the appropriate behavioral tests to prove the effects of a neuroprotective drug, highlighting the differential time line among the different behavioral tests analyzed.

- The capability of 2-APB to modulate the duration of cortical spreading depolarizations despite the variability observed in the results as a consequence of the use of a non-consanguineous mouse strain.

- The importance of how the modulation of peri-infarct depolarization translates into specific hemodynamic changes in the penumbra. 2-APB-treated mice showed higher residual cerebral blood flow after permanent ischemia.

- The efficacy of the antioxidant scavenger cholesteronitrone F2 in reducing reactive oxygen species content in a model of permanent ischemia; until now this molecule has only been tested in ischemia- reperfusion models.

- The work is timely and will be of interest to a broad readership of biologists and physicians, essentially scientists who are interested in searching alternative avenues to drive neuroprotection after stroke.

- The introduction of electrophysiological measurements such as somatosensory evoked potentials in the experimental design as a quantitive way to infer post-stroke cortical functionality in response to any potential treatment.

- The detection of reactive oxygen species in vivo, alternatively to ex vivo detection, might display a more accurate perspective of stroke derived oxidative stress that can be transferred to study other neuropathologies linked with oxidative stress.

Biography

Rocío Fernandez-Serra studied B.S. in Biochemistry (2016, Universidad Complutense de Madrid (UCM)) and Master in Neuroscience (2017, UCM). She then joined to Daniel Gonzalez-Nieto´s Lab at the Center for Biomedical Technology of the Universidad Politécnica de Madrid (UPM). She is doing her PhD in Biomedical Engineering on the development of silk fibroin-based drug delivery systems for stroke neuroprotection. She has participated in the teaching programs of Biomedical Engineering, Biotechnology and Material Science degrees at UPM. She has supervised several projects from Biomedical Engineering and Biotechnology students. She has published 2 reviews and other 2 are now under revision.

J.L. Gómez-Ariza[1,2]* and G. Montaña-Moreno[1]

[1]Department of Chemistry, University of Huelva, Spain
[2]Research Center for Natural Resources, University of Huelva, Spain

Neurology 2022

Bioanalytical methodologies for the study of phospholipids in alzheimer disease

Phospholipids play an important role in living organisms' lipid bilayers, acting as a structural barrier for cellular and subcellular protection, being a fundamental component for the correct function of membrane proteins, receptors and ion channels. In addition, phospholipids also act as storage depot of a complex meshwork of lipid mediators, such as eicosanoids, lysophospholipids, platelet activating factors or diacylglycerides, which exert a diverse array of effects on cellular functional activities, including neural cell homeostasis, immune responsiveness, oxidative stress, and neuroinflammation. Therefore, defects in phospholipids metabolism are related to numerous diseases, such as Alzheimer Disease (AD)[1]. Thereby, although major hallmarks of AD are the formation of senile plaques and neurofibrillary tangles, the changes in phospholipids could be used as biomarkers in AD, using non- invasive samples such as human serum.

In this study, shotgun metabolomics of serum samples was performed by Direct Infusion Mass Spectrometry (DIMS) for the screening of phospholipids involved in neurodegenerative processes associated with AD. Furthermore, a targeted analytical approach focused on phospholipids was optimized, using Reversed Phase Ultra-High Performance Liquid Chromatography (RP-UPLC) and detection by molecular mass spectrometry. However, the high complexity of samples requires selective detection methods based on phosphorous-tagging by the coupling of LC with ICP-MS [2], enabling phospholipids quantification without the use of structurally matched standards. This combination of different mass spectrometry-based metabolomics strategies allows to deep insight into the dyshomeostasis of phospholipids in AD.

This abnormal metabolism results in important biochemical changes in brain, which are reflected in peripheral serum, who's most important results are the following:

1) Lysophospholipids. Decreased levels of different species of lysophosphatidylcholine (LPC), and similar trend for lysophosphatidylethanolamine (LPE) and lysoplasmenylcholine (LPPC), which reflects the alteration of metabolism of choline-containing compounds, along with other families such as ethanolamine and plasmenylcholine, in serum.

2) Phosphatidylcholine (PC). It has been observed the decreases of Polyunsaturated Fatty Acid (PUFA) in the molecular moiety of PCs and the correlative increase of Saturated Fatty Acids (SFA). This change in fatty acid composition of membrane lipids affects their biophysical properties (fluidity, permeability and charge) contributing to membrane damage in AD pathogenesis.

3) Phosphatidylethanolamine (PE). PEs are an important family of brain phospholipids with a content of PUFAs higher that PC, having been observed a decrease of these polyunsaturated fatty acids in serum of AD patients, possibly as a consequence of oxidative stress.

4) Plasmalogens. which are major constituents of neural membrane forming part of myelin sheath, being also involved in other metabolic functions. It has been observed deficiency of plasmenylethanolamine (PPE), which constitutes up to 70% of total plasmalogens, in serum that has been traditionally associated with AD development. González-Domínguez R, García-Barrera T, Gómez-Ariza JL. Combination of metabolomic and phospholipid- profiling approaches for the study of Alzheimer's disease. J. Proteomics 2014; 104: 37-47. Kovačevič M, Leber R, Kohlwein S D, Goessler W. Application of inductively coupled plasma mass spectrometry to phospholipid analysis. J Anal At Spectrum 2004; 19:80–4.

Audience Takeaway

- The study demonstrates the great possibilities of the combined use of molecular and atomic mass spectrometry in metabolomics.

- The application of these methodologies to serum samples reflects the systemic nature of Alzheimer's disease and the interest in using early warning biomarkers based on non-invasive samples.

- Diagnostic tests can be designed based on several families of metabolites, such as lysophospholipids, phosphatidylcholines, phosphatidylethanolamines and plasmalogens.

Biography

Jose Luis Gómez-Ariza is full professor of Analytical Chemistry and member of the Research Center for Natural Resources Health and the Environment of the University of Huelva (Spain). Member of the Spanish Society for Analytical Chemistry (SEQA) and the Academy of Sciences, Arts and Letters of Huelva. In 2003 received the Andalusia Research Award and the Huelva Industry Excellence Award. In 2022 has received the Gold Medal of the University of Huelva. He is the author or co-author of over 300 scientific publications (h-index 46), several books and book chapters, as well as over 600 contributions to scientific conferences, he also holds several patents. Over 35 students have received doctorates under his direction.
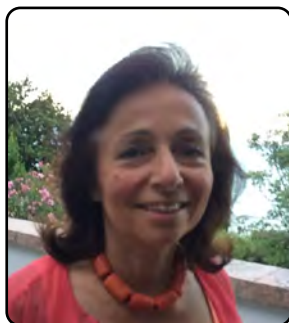
[1]Manuela Marcoli*, [1]Chiara Cervetto, [2]Diego Guidolin, [3,4]Luigi F Agnati, [1]Guido Maura

[1]Department of Pharmacy, University of Genova, Italy
[2]Department of Neuroscience, University of Padova, Italy
[3]Department of Biomedical Sciences, University of Modena and Reggio Emilia, Istaly
[4]Department of Neuroscience, Karolinska Institutet, Sweden

New insights in neuron-astrocyte communication : Astrocytic G protein coupled receptor heteromers and release of glutamate

It is increasingly recognized that neurons and astrocytes are co-actors in the communication in Central Nervous System (CNS). Central to the view of astrocytes as active contributors in brain signaling are the astrocytic receptors that respond to neurotransmitters and the neuronal receptors acted upon by gliotransmitters at tripartite synapses. Recognition and decoding of signals via G Protein-Coupled Receptor (GPCR)-receptor interaction and heterodimer formation at neuronal plasma membrane, hypothesized since the '80s by Agnati and Fuxe, at Karolinska, is now generally accepted. GPCR heteromers exhibit a unique pharmacology, with changes in receptor agonist recognition, signaling and trafficking, providing targets for innovative pharmacological approaches to CNS disorders. Despite the relevance of neuron-astrocyte networking in the CNS function, the possibility that the integrative information handling via receptor-receptor interactions may also function at astrocyte membrane level has barely been investigated.

Here, the presence and function of A2A-D2 heteromers on striatal astrocytes was investigated on slices and isolated purified astrocyte processes obtained *ex vivo* from adult rat striatum. We measured the release of the glutamate analogue 3H-D-Aspartate from superfused processes; receptor colocalization in slices and in the processes was investigated by immunofluorescence and Proximity Ligation Assay (PLA), co-immunoprecipitation by Western analysis. Our major findings: purified processes prepared ex vivo from astrocytes matured *in situ* in striatum neuron-astrocyte network were capable of vesicular glutamate release, that could be controlled by D2 receptor activation; A2A receptors appear co-localized with D2 receptors on the processes, and able to functionally interact with the receptor. Biochemical and biophysical evidence indicating heteromerization of native A2A-D2 on the striatal astrocyte plasma membrane were obtained by co-immunoprecipitation and PLA analysis.

It is concluded that A2A-D2 heteromers at striatal astrocyte plasma membrane are a new research area to understand the striatal astrocyte-neuron intercellular communication and the astrocyte involvement in glutamate transmission dysfunction in neurodegenerative/neuropsychiatric conditions. Also, striatal astrocytic A2A-D2 heteromers can be exploited as druggable targets. Defective function of striatal astrocytic D2 (and consequent glutamatergic dysregulation and vulnerability to neuron injury) may be improved by targeting the allosteric inhibition of D2 in A2A-D2 heteromer, by acting upon A2A (A2A antagonists or bivalent compounds) or by controlling homocysteine production (COMT inhibitors), for a potential therapeutic for PD and L-DOPA dyskinesias in PD treatment.

Our findings, which suggest that reduced D2-mediated control at striatal perisynaptic astrocyte processes might result in an increase in synaptic glutamate level, could help to understand how astrocytes (and remodeling of astrocyte processes) contribute to the pathophysiology of Parkinson's disease. Indeed, expansion of perisynaptic astrocyte processes, and altered neuron-astrocyte interactions at striatal glutamatergic synapses, have been reported in Parkinson's disease. We propose that we cannot longer think about striatal D2 signaling, adenosine transmission, and glutamate transmission (and L-DOPA dyskinesias) without considering striatal astrocytes, and astrocyte-neuron communication.

Audience Takeaway

- G Protein-Coupled receptor heteromers at astrocyte plasma membrane are a new research area to understand striatal astrocyte-neuron intercellular communication.
- Astrocytes can be involved in glutamate transmission dysfunction in neurodegenerative/neuropsychiatric conditions.

- Striatal astrocytic A2A-D2 heteromers can be exploited as druggable targets.

- We cannot longer think about striatal D2 signaling, adenosine transmission, and glutamate transmission (and L-DOPA dyskinesias) without considering striatal astrocytes, and astrocyte-neuron communication.

Biography

Prof. Manuela Marcoli studied and graduated as M.D. at the Pavia University, Italy in 1980; she received her PhD degree in Clinical Pharmacology in 1984 at the same institution. She joined the research group of pharmacology at the University of Pavia, and then the group of neuropharmacology at the University of Genova, Italy. She is Researcher and Professor of Pharmacology, Bachelor Course in Biology and Master Course in Molecular and Health Biology, and Ph.D program in Experimental Medicine, Curriculum: Pharmacology and Toxicology, University of Genova, Italy. She has published more than 100 research articles in peer-reviewed scientific journals.

Surina Natalia M*., Fedotova Irina B., Poletaeva Inga I

Department of Biology, Lomonosov Moscow State University, Moscow, Russia

Audiogenic epilepsy model–krushinsky-molodkina strain rats : Neurophysiology, Genetics and behavior

The trait – audiogenic seizure proneness (Audiogenic Epilepsy, AE) in KM strain rats - is characterized by high expressivity and penetrance – the short latency (1-2 s) seizure fit in the 100 % of cases with maximal intensity of clonic and then tonic seizures of trunk and extremities musculature. The long lasting postical catalepsy follows the audiogenic epilepsy fit. The postictal "audiogenic" catalepsy is also regarded as the perspective laboratory model of another type of brain pathology. The KM rat strain is maintained in the Laboratory of Physiology   and Genetics of Behavior (Biology Department, Lomonosov Moscow State University) during 60 years and starting from 1989 – as the inbred strain). It served as the valuable laboratory model for testing the big range of anticonvulsant drugs (both - already used in clinic and the new ones). Valproate sodium, difeninum, carbamazepin, lamotrigine, levetiracetam and several other anticonvulsants proved to decrease both the intensity and increase the latency of seizure fit in response to sound.

Audiogenic epilepsy seizure fit could be regarded as the model for pre-clinical investigation of anticonvulsant drugs. Audiogenic seizure fit is initiated in brain stem and the neural network which is responsible for initiation and sustaining of seizures in response to sound is now identified. It means that this pathological trait in audiogenic epilepsy prone rats could provide the important knowledge for the study of epileptogenesis. In order to create the more reliable group of control animals which would share the larger part of genotype of AE susceptible KM rats the new selection experiment was initiated in 2001. The aim of this selection was to breed the strain of rats which are related to KM by at least part of genotype but which would be non-prone to audiogenic epilepsy. The new strain was labeled as "0" strain. The proportion of rats with "0" reaction to loud sound exposure in "0" strain was about 50% at 21-th generation. The differences between KM and "0" strains were also found in the intensity of postictal catalepsy. We would like to point out that KM strain rats can be fruitfully used not only as an audiogenic convulsive seizure model, but also as a model of several other disorders, such as myoclonic hyperkinesis, acute disorders of cerebral circulation, cataleptic states of various genesis: spontaneous, post-convulsive, pharmacological, pinching, and comorbid neuropsychiatric disorders: anxiety, depressive-like states, cognitive deficits.

Audience Takeaway

- I would like to present our recent research results on audiogenic epilepsy model – Krushinsky-Molodkina audiogenic prone rats. It is including neurochemistry, genetics, psysiology and behaviour investigation.

- It might be interesting for the audience as a new approach to epilepsy modelling – working with non-pharmacological, non-invasive genetic model of the audiogenic epilepsy.

- Main advantages of our genetic model are following: clearness and reproducibility of the phenomenon, the ability to quantify indices, noninvasiveness of provocation, absence of difficulties in interpreting the results associated with side effects of provoking pharmacological drugs, genetic conditioning.

Biography

Dr. Surina studied neurophysiology at the Lomonosov Moscow State University, Russia and graduated as MS in 2007. She then joined the research group of Prof. Poletaeva, Laboratory for Physiology and Genetics of Behavior, Chair of Higher Nervous Activity (Neurobiology). She received her PhD degree in 2011 at the same institution and became senior researcher. Providing RFBR and RGNF grants (ongoing works) and UMNIK grants (winner of the program "Participant of the youth scientific and innovative competition-2009, 2013".) Winner of the conjoint program of MSU-DAAD "Vladimir Vernadsky". She has published more than 38 research articles in SCI(E) journals and 60 abstracts.
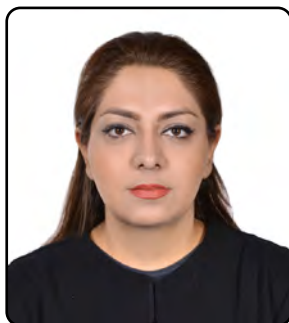
Roshanak Mehdipour, MD[1]*, Fereshteh Ashtari, MD[2]

[1]Isfahan University of Medical Science, Iran
[2]Neurology Department, Isfahan University of Medical Science, Iran

The clinical and psychological effect of COVID19 pandemic on neuromyelitis optica spectrum disorder patients

Coronavirus 2019 was a new coronavirus created a pandemic with high mortality. People with underlying disease and immune system suppression are potentially prone to infection. The nature of NMOSD disease as well as its treatment by immunosuppressants predisposes patients to infection. The coronavirus epidemic in a few months lead to significant changes in people's lifestyles and attitudes. Public health measures such as quarantine to prevent the spread of the disease have had a negative psychological impact. People with chronic illnesses, including Neuromyelitis Optica (NMOSD) are more prone to neuropsychiatric disorders, which can lead to fear of continuing treatment. The aim of study was to evaluate the effect of COVID19 pandemic on the clinical course of NMOSD and the characteristics of COVID19 infection in NMOSD patients after a year. Also to investigate the level of anxiety and its changes in NMOSD patients during COVID19 pandemic. The another issue was determining patients' adherence to precautionary measures and health protocols.

Study included 120 patients (41 seropositive, 96 female). Mean age was 36.37±9.69 and mean duration of disease was 8.49±5.35 years. We considered relapses during the year of epidemic and the year before and the presentation of COVID19 infection in the patients of NMOSD Clinic of Isfahan Kashani hospital. Patients were asked also about changes in maintenance therapy. We asked patients if they were anxious or afraid of the pandemic subjectively. To investigate the objective level of anxiety, we benefited Hospital Anxiety and Depression Scale (HADS-A) questionnaire. We asked them about respecting general health cautions to prevent infection too.

Results showed that in spite of suppression of the immune system, neither incidence nor the number of the serious complications of COVID19 infection was high. Therefore, regarding the disabling nature of NMOSD as well as prolonged epidemic period, it may be reasonable to continue the routine treatment of these patients along with training patients to stick to health protection instructions. Along with the pandemic prolongation, the level of anxiety decreased gradually while the level of alertness was almost high. The COVID19 vaccination had a dramatic effect on decreasing fear and anxiety and improving mental health

Audience takeaway

- In spite of immunosuppressive therapy in NMOSD patients, the cases infected by COVID-19 didn't experience severe complications or atypical presentation.
- Immunosuppressant therapy can be continued routinely in NMOSD patients during COVID19 epidemic, but any expansion of dosing interval should be according to clinical and paraclinic findings of patients under recommendation of neurologist.
- Patients with NMOSD are at a great risk of psychiatric effects of COVID-19 epidemic such as anxiety and depression.
- The anxiety level decreased gradually during epidemic period specially after vaccination program but they stick health protection measures more.
- The COVID19 vaccination have positive effect on improving mental health.

Biography

Dr. Mehdipour studied Medicine at the Isfahan University of Medical Sciences, Iran and graduated as MD in 2013. She then joined the Neurology residency program in the same university and received Neurologist degree in 2017. She is member of Isfahan Neuroscience Research Center since 2015 and her specific field of research is Demyelinating disease. She did as postdoctoral fellowship since 2017 to 2019 in Kashani hospital MS center. She has published several articles especially in NMOSD category.

Maria Eulina Quiliao

Instituto da Criança of Hospital das Clinicas de São Paulo/USP, Brazil

Neurological development, Epilepsy, and the pharmacotherapy approach in children with congenital zika syndrome : Results from a two-year follow-up study

Introduction: Background: During the Zika virus epidemic in Brazil, from January 2015 to December 2018, there were 278,790 people infected, and of these approximately 22,000 women who were pregnant. Consequently, there was a considerable increase in the number of newborns (17,041) with microcephaly and other malformations - Congenital Zika Syndrome (CZS). In our state, in this same period, 427 cases of the disease were confirmed, 185 of which occurred in pregnant women, and there were 16 cases of Congenital Zika Syndrome (CZS).Objective: Clinical folllow up of neuropsychomotor development, and clinical staging and electroencephalographic evolution of the epileptic condition.

Methods: Follow up and clinical evaluation of eleven children suffering from CZS, in which the following were evaluated: head circumference growth, neuropsychomotor development, existence or lack of spasticity, presence or absence of epilepsy and its pharmacological treatment.

Results: The average age of the children was 24 months at the time of the first evaluation and 36 months at the time of the second evaluation. Cerebral palsy has been identified in all children (GMFCS level 5 in 9 cases), microcephaly and pseudobulbar syndrome in eight, and hydrocephalus in three cases. Malnutrition was identified in seven children. Four of the children did not present epilepsy, three evolved to West Syndrome, one had generalized epileptic seizures and three had focal seizures. Four cases of refractory epilepsy are undergoing polytherapy and one is in monotherapy, and two others progressed without seizures and without anticonvulsants.

Conclusion: In the vast majority of cases, CZS evolves to severe Cerebral Palsy and refractory epilepsy.

Lori A Gray

Holistic Health Programs, Western Michigan University, USA

Introducing a Mindfulness-Based Protocol for Recovery from Stroke (MBRfS)

Decades of research suggest that Mindfulness-Based Stress Reduction (MBSR) training supports a greater capacity to live with chronic medical conditions and contributes to lowering stress levels. This paper introduces a model for a Mindfulness-Based Recovery from Stroke (MBRfS) for promoting stroke recovery, informed by the lived experience of the author (a stroke survivor and certified MBSR instructor), the research literature regarding MBSR training, and the specific challenges of stroke recovery. A literature search was conducted to explore possible research and publications adapting M.B.S.R. to medical populations, including stroke survivors. There is very limited research in this area. A summary will be presented of similar protocols, such as applying M.B.S.R. in programs for cancer survivors and chronic pain patients. An autoethnography was developed as a methodology to support to the model.

A theoretical case is made for the value and need for the development of a mindfulness-based program for those who have experienced and are recovering from a stroke. The proposed mindfulness-based stroke recovery intervention (MBRfS) is outlined in detail, informed by similar evidence-based programs. Case vignettes are offered in the autoethnography that emerged, drawing on the authors experiences as a stroke survivor and MBSR certified teacher. A four-component MBRfS model is offered, which consists of an integration amongst a modified MBSR framework, emergent attitudinal themes, and insights from the autoethnographic vignettes. The MBRfS model offers a path for providing participants with a supportive experience within stroke recovery. Recommendations and suggestions for future studies are offered to support the development of MBRfS for stroke survivors and their caregivers, as well as contributing to healthcare providers.

Audience Takeaway

- The audience will have a deepened understanding of mindfulness and how it can support stroke recovery.
- The audience can apply this information to benefit their patients in stroke recovery and caregivers.
- This paper will provide potential research and teaching collaborations amongst the audience.
- The audience will be able to take the information shared and improve stroke recovery models and treatment protocols.

Biography

Dr. Lori Gray is an Assistant Professor at Western Michigan University. Since 2000, she has served WMU as an instructor in the Integrative Holistic Health and Wellness program and is the current program coordinator. In 2014 she earned a post-doctoral certificate from the University of Massachusetts Medical School to teach Mindfulness-Based Stress Reduction; she is also a National Board-Certified Health and Wellness Coach (NBC-HWC). Dr. Gray's research interests include the efficacy of mindfulness-based interventions and stroke recovery, and the role of contemplative pedagogy in higher education.

Armaan Saith*, Shikhar Khurana

Temple University, United States

Information extraction from EHR and EEG signals in search of a potential biomarker for ADHD diagnosis

ADHD is the most common neurobehavioral disorder of childhood characterised by inattention, impulsivity and motor hyperactivity. The estimated number of children ever diagnosed with ADHD, according to a national 2016 parent survey is 6.1 million (9.4%). An estimated 8.7 million adults live with ADHD in the United States, resulting in a total societal excess cost attributable to ADHD of $122.8 billion ($14,092 per adult). Electroencephalography (EEG) is a common tool for prognostic and diagnostic purposes. Previous studies have shown that the most robust EEG feature associated with ADHD is elevated power of slow waves (4–7Hz "theta") and/or decreased power of fast waves (14–30Hz "beta"), quantified by the Theta/Beta Ratio (TBR). The transient neural dynamics captured by ERPs have also been explored for distinguishing features of ADHD. Significant non-linear association between resting gamma power and age in the lower frequency gamma-1 (30-39 Hz), stronger relative right frontal alpha power and a highly significant rightward EEG high-beta (16-21 Hz) asymmetry in inferior parietal brain regions are other findings seen in previous studies. But neither of the mentioned findings have been observed in datasets large enough to draw conclusions in terms of finding a specific biomarker for ADHD on EEG.

The TUH-EEG Corpus is an ongoing data collection effort that has recently released 14 years of clinical EEG data collected at Temple University Hospital. The records have been curated, organized, and paired with textual clinician reports that describe the patients and scans. The completed corpus comprises 16,986 sessions from 10,874 unique subjects. Number of sessions per year varies from approximately 1,000-2,500. Our team used AI (Natural language processing and Signal analysis techniques) to extract patterns from clinical information and EEG waves. Electronic records and EEG data of patients who were diagnosed with ADHD and had ever been placed on any kind of stimulants for treatment of ADHD were collected from the TUH-EEG (Temple University Hospital) dataset. The health records of all the patients were analyzed using Natural Language Processing algorithms to extract the textual info and identify and select the abnormal from the normal EEGs. The abnormal EEG were further analysed using Signal analysis and Machine learning techniques. Using patient EHR and EEG waveforms from the TUH-EEG corpus, we seek to find a potential biomarker to help in diagnosis and assessing the prognosis of ADHD patients.

Biography

Dr. Armaan Saith studied medical science at Maulana Azad Medical College, Delhi University, New Delhi, India and graduated as MBBS in 2022. He is currently a part of the Temple University team doing research under Dr Vikas Khurana and Prof. Subodha Kumar on the TUH-EEG corpus, one of the world's largest EEG datasets. He also has a publication in a pubmed indexed journal.

KEYNOTE FORUM

DAY 02

5TH EDITION OF
INTERNATIONAL CONFERENCE ON

NEUROLOGY AND NEUROLOGICAL DISORDERS

15–16 JUNE

Sergey Suchkov[1-3]*, Noel Rose[4], Aleks Gabibov[5], Harry Schroeder[6]

[1]MINO MGUPP, Russia

[2]A.I. Evdokimov Moscow State Medical & Dental University, Russia

[3]EPMA (European Association for Prediction, Prevention and Personalized Medicine), EU

[4]Johns Hopkins Medical Institutions, USA

[5]Institute for Bioorganic Chemistry, Russian Academy of Sciences, Russia

[6]Division of Immunology & Rheumatology, USA

Antibodies with functionality as a new generation of translational tools designed to monitor Multiple Sclerosis (MS) at clinical and subclinical stages

Catalytic Abs (catAbs) are multivalent immunoglobulins (Igs) with a capacity to hydrolyze the antigenic (Ag) substrate. In this sense, proteolytic Abs (*Ab-proteases*) represent Abs to provide proteolytic effects. Abs against myelin basic protein/MBP with proteolytic activity exhibiting sequence-specific cleavage of MBP are of great value to monitor demyelination whilst in MS. The activity of Ab-proteases was first registered at the *subclinical* stages 1-2 years prior to the clinical illness. And the activity of the Ab-proteases revealed significant correlation with scales of demyelination and the disability of the patients as well. So, the activity of Ab-proteases and its dynamics tested would confirm a high *subclinical* and *predictive* value of the tools as applicable for monitoring protocols. Of tremendous value are Ab-proteases directly affecting remodeling of tissues with multilevel architectonics (for instance, myelin). By changing sequence specificity one may reach reduction of a density of the negative proteolytic effects within the myelin sheath and thus minimizing scales of demyelination. Ab-proteases can be programmed and re-programmed to suit the needs of the body metabolism or could be designed for the development of new catalysts with no natural counterparts. Further studies are needed to secure artificial or edited Ab-proteases as fantastic tools of the newest generation to diagnose, to monitor, to control and to treat and rehabilitate MS patients at clinical stages and to prevent the disorder at subclinical stages in persons-at-risks to secure the efficacy of regenerative manipulations.

Audience Takeaway

- To learn more about Ab-mediated catalysis (proteolysis) as the biological phenomenon of the next-step generation and its impact in daily practice of practitioner!

- The presentation would get the audiences armed with new approaches to monitor both clinical and subclinical stages of MS in precise scenarios!

- In this translational research faculty could use the latter for setting up translational pipelines on one hand and to teach the students and postgraduate physicians on the other one!

- The findings will solve a problem of predictive diagnostics, preventive and rehabilitative measures to be applied for monitoring the MS patients, the persons-at-risks and to prevent transformation of subclinical stages of MS into clinical ones!

Biography

Sergey Suchkov graduated from Astrakhan State Medical University and awarded with MD, then in 1985 maintained his PhD at the I.M. Sechenov Moscow Medical Academy and in 2001, maintained his Doctorship Degree at the Nat Inst of Immunology, Russia. From 1987 through 1989, he was a Sen Researcher, Koltzov Inst of Developmental Biology. From 1989 through 1995, he was a Head of the Lab of Clin Immunology, Helmholtz Eye Res Institute in Moscow. From 1995 through 2004, a Chair of the Dept for Clin Immunology, MONIKI Dr. Suchkov has been trained at: NIH; Wills Eye Hospital, PA, USA; Univ of Florida in Gainesville; UCSF, S-F, CA, USA; Johns Hopkins University, Baltimore, MD, USA. He was an Exe Secretary-in-Chief of the Edit Board, *Biomedical Science*, an int journal published jointly by the USSR Academy of Sciences and the Royal Society of Chemistry, UK. At present, Dr Sergey Suchkov is a Chair, Dept for Personalized Medicine, Precision Nutriciology and Biodesign at MINO MGUPP, Russia. He is a member of the: New York Academy of Sciences, American Chemical Society (ACS), American Heart Association (AHA), EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU; ARVO (American Association for Research in Vision and Ophthalmology); ISER (International Society for Eye Research); PMC (Personalized Medicine Coalition), Washington, USA.

Torbjörn Bäckström

Department of Clinical Sciences, Umeå University, Sweden

GABA-a receptor modulating steroids impair learning and memory but can be antagonized with GABa-a receptor modulating steroid antagonists

Gamma-Amino Butyric Acid (GABA) is the main inhibitory neurotransmitter in the brain and GABA-ergic transmission is shown to be of importance for regulation of mood, memory, and food intake. The progesterone metabolite allopregnanolone (Allo) is a positive GABAA receptor modulating steroid with potent effects. In humans, disorders like Premenstrual Dysphoric Disorder (PMDD), hepatic encephalopathy and polycystic ovarian syndrome are associated with elevated Allo levels and increased negative mood, disturbed memory, and increased food intake in some individuals. This is surprising as Allo shares many properties with benzodiazepines and is mainly considered to be anxiolytic and anti-depressant.

However, it is well established that in certain individuals GABAA receptor active compounds could have paradoxical effects and thus be anxiogenic in low physiological plasma concentrations while anxiolytic at high levels. In Alzheimer transgenic mice continuous allo in low stress concentrations deteriorated the dementia progress. We have demonstrated that isoallopregnanolone (Isoallo), the 3β-OH sibling of Allo, functions as a GABAA receptor Modulating Steroid Antagonist (GAMSA), but without any effects by its own on the GABAA receptors. The antagonistic effect is noted in most GABAA subtypes investigated so far. *In vivo*, Isoallo inhibits Allo-induced anesthesia in rats, as well as sedation or saccadic eye velocity in humans. Isoallo has been studied in women with PMDD. In two phase II studies, Isoallo (Sepranolone) injections significantly ameliorated negative mood in women with PMDD compared with placebo. One GAMSA, UC1011, could inhibit Allo induced memory disturbances in rats. A GAMSA for oral administration have also been developed, GR3027, has been shown to restore learning and motor coordination in rats with hepatic encephalopathy. In human's, vigilance, cognition, and pathological EEG was improved in patients with hepatic encephalopathy when treated with GR3027.

Biography

Torbjörn Bäckström is a Chief Executive Officer, co-founder and CEO of Umecrine AB, is professor at the Department of Clinical Science, Obstetrics and Gynecology, Umeå University. He is also head of the Umeå Neurosteroid Research Center. Professor Bäckström's main focus of research since 1972 is the effect of sex and stress hormones on the brain and conditions induced by these hormones, he has more than 400 scientific publications within this area.

Thomas J. Webster

Interstellar Therapeutics, Boston, MA, USA

Nanomedicine for treating neurological disorders : Small materials for big problems

Nanomedicine has already revolutionized medicine. This is because nanomaterials can mimic the size of natural components of tissue to increase tissue growth, inhibit infection, and limit inflammation. The surface energy of nanomaterials can also be easily controlled to in turn mediate initial protein adsorption that can promote neuron function. This presentation will summarize how nanomaterials can be designed, fabricated, and used to stimulate neuronal cell function to treat numerous neurological disorders. Specifically, as just one nanomaterial example, carbon nanotubes were implanted with stem cells into stroke-induced rat brains and demonstrated statistically faster return of motor function compared to stem cells alone. Impressively, carbon nanotube implantation alone into stroke-induced rats returned motor function faster than stem cells alone after as early as 7 days. This presentation will cover other nanomaterials that can improve neuronal cell function and describe critical studies needed for the field of neurological nanomedicine to grow and reach the clinic.

Audience Takeaway

- How nanomaterials can increase tissue growth, decrease infection, and inhibit inflammation.

- How nanomaterials can be used to treat neurological problems of stroke, Alzheimer's, Parkinson's, and others.

- How to create nanomaterials for improving disease prevention, detection, and therapy.

Biography

Thomas J. Webster's (H index: 108; Google Scholar) degrees are in chemical engineering from the University of Pittsburgh (B.S., 1995; USA) and in biomedical engineering from RPI (Ph.D., 2000; USA). He has served as a professor at Purdue (2000-2005), Brown (2005-2012), and Northeastern (2012-2021; serving as Chemical Engineering Department Chair from 2012 - 2019) Universities and has formed over a dozen companies who have numerous FDA approved medical products currently improving human health. Dr. Webster has numerous awards including: 2020, World Top 2% Scientist by Citations (PLOS); 2020, SCOPUS Highly Cited Research (Top 1% Materials Science and Mixed Fields); 2021, Clarivate Top 0.1% Most Influential Researchers (Pharmacology and Toxicology); and is a fellow of over 8 societies.

Day
02

Neurology 2022

Juliana Fort*, Michael Kenny

Louisiana State University Health Shreveport, United States

Using laughter and the expressive arts to promote creativity and learning for neurological and psychiatric scientists and their clients, An experiential example using animal as metaphor

Using metaphor with themselves or patients can give neurological and psychiatric scientists and clinicians a safe way to help understand how we cope with stress and how that can affect our experiences of neurological, physical and psychiatric illnesses. The expressive /creative arts are well suited to digital wellness and overall wellness. The use of the body reduces stress and helps to be present in the body in the here and now and enhances creativity and learning. "Scientists and students of science need "skillful know-how in situated and embodied action"(Engel et al., 2013 p. 202). Scientists will learn how to be embodied. Participants will become familiar with expressive/ creative arts modalities such as drama/role play, improvisation, storytelling, movement and embodiment, music, art, poetry and creative writing they can use as tools for patients to have outlets to manage stress as well as tools for scientists to manage their own stress and wellbeing. Laughter can promote learning and wellbeing; participants will have some tools to incorporate laughter into their practice with themselves and patients. This workshop will demonstrate examples of a workshops with experiential activities, using a few examples of expressive arts such as: movement, imagination, and metaphor experientially to help clinicians come up with ideas to incorporate the expressive arts and creativity into their clinical practice.

Biography

Juliana Fort, MD, MPH, MBA, medical student clerkship director and Clinical Associate Professor in Psychiatry at LSUHS in Shreveport is board certified in child and adolescent, geriatric, forensic, and addictions psychiatry. With a MA in mental health counseling with a specialization in drama therapy from Lesley University, a MA in creative writing, she is a play therapist / supervisor and enjoys co-facilitating workshops with students and colleagues, and psychiatry/neurology residents. Using expressive arts: movement, improvisation, music, art, creative writing, and poetry enriches experiential teaching, wellness, medicine and the arts/humanities, psychotherapy, and personal growth. To collaborate with virtual workshop: juliana.fort@lsuhs.edu

37

Kenneth B Storey

Carleton University, Canada

The brain in winter

The human brain operates in a protected environment of constant temperature and uninterrupted resources of oxygen and nutrients. However, many other vertebrates show amazing plasticity of their brain function. Their brains function with greater flexibility and provide important lessons for us about "How Brains Work" under stressful environmental conditions. My lab studies vertebrate species that freeze solid in winter (including their brains), changing brain biochemistry and gene regulation to endure freeze/thaw with no damage. The brains other vertebrates can survive with no oxygen; we would be dead but for them it's no problem, no damage. Many non-human mammals hibernate in winter – turning down their brain metabolism to near zero using epigenetic controls to shut down gene expression and posttranslational controls to suppress protein/enzyme function, all reversible with no damage. Some hibernators can chill their bodies to near 0°C, suppressing brain function for weeks/months. Others hibernate at high body temperature with complex controls to coordinate the suppression of their metabolism. Studies of all these animals teach us new paradigms of metabolic control and demonstrate the enormous plasticity of animal metabolism. Future of Brain Health: can we find ways to utilize lessons from Nature to unlock more flexibility of brain function than we have found to date and apply these lessons to protecting the human brain from disease and damage.

Biography

Dr. Kenneth B. Storey, Ph.D., F.R.S.C., is a Professor of Biochemistry at Carleton University in Ottawa and holds the Canada Research Chair in Molecular Physiology. He received his B.Sc. from the University of Calgary and his Ph.D. from the University of British Columbia. Ken is a world leader in the field of biochemical adaptation. He uses tools of enzymology, protein chemistry and molecular biology to identify the adaptations of gene regulation and enzyme structure/function that support amazing animal phenomena including hibernation, freezing survival, estivation and anoxia tolerance with a particular focus on the mechanisms metabolic rate depression that support these phenomena. Ken is a prolific author and speaker – he has over 1000 publications to his name and has given hundreds of talks around the world. Ken won the 2010 Flavelle medal in Biological Sciences from the Royal Society of Canada, "CryoFellow" of the CryoBiology Society in 2012 and the 2011 Fry medal from the Canadian Society of Zoologists.

SPEAKERS
DAY 02

5TH EDITION OF
INTERNATIONAL CONFERENCE ON

NEUROLOGY AND
NEUROLOGICAL DISORDERS

15-16 JUNE

Rakesh Sahu[1], Sidharth Mehan[1], Sumit Kumar[1]*, Aradhana Prajapati[1], Abdulrahman Alshammari[2], Metab Alharbi[2], Mohammed A. Assiri[2], Acharan S. Narula[3]

[1]Neuropharmacology Division, Department of Pharmacology, India

[2]Department of Pharmacology and Toxicology, College of Pharmacy, King Saud University, Saudi Arabia

[3]Narula Research, USA

Effect of alpha-mangostin in the prevention of behavioral and neurochemical defects in methylmercury-induced neurotoxicity in experimental rats

Methylmercury (MeHg+) is a known neurotoxin that causes progressive motor neuron degeneration in the central nervous system. Axonal degeneration, oligodendrocyte degeneration, and Myelin Basic Protein (MBP) deficits are among the neuropathological abnormalities caused by MeHg+ in Amyotrophic Lateral Sclerosis (ALS). This results in demyelination and motor neuron death in both humans and animals. Previous experimental studies have confirmed that overexpression of the extracellular signalling regulated kinase (ERK1/2) signalling contributes to glutamate excitotoxicity, inflammatory response of microglial cells, and oligodendrocyte (OL) dysfunction that promotes myelin loss. Alpha-mangostin (AMG), an active ingredient obtained from the tree "Garcinia mangostana Linn," has been used in experimental animals to treat a variety of brain disorders, including Parkinson's and Huntington's disease memory impairment, Alzheimer's disease, and schizophrenia, including Parkinson's disease and Huntington's disease memory impairment, Alzheimer's disease, and schizophrenia.

AMG has traditionally been used as an antioxidant, anti-inflammatory, and neuroprotective agent. Accordingly, we investigated the therapeutic potential of AMG (100 and 200 mg/kg) in experimental rats with methylmercury (MeHg+)-induced neurotoxicity. The neuroprotective effect of AMG on behavioural, cellular, molecular, and other gross pathological changes, such as histopathological alterations in MeHg+ -treated rat brains, is presented. The neurological behaviour of experimental rats was evaluated using a Morris Water Maze (MWM), Open Field Test (OFT), Grip Strength Test (GST), And Force Swim Test (FST). In addition, we investigate AMG's neuroprotective effect by restoring MBP levels in cerebral spinal fluid and whole rat brain homogenate. The apoptotic, pro-inflammatory, and oxidative stress markers were measured in rat blood plasma samples and brain homogenate. According to the findings of this study, AMG decreases ERK-1/2 levels and modulates neurochemical alterations in rat brains, minimising MeHg+ -induced neurotoxicity.

Audience Takeaway

- Amyotrophic lateral sclerosis (ALS) is a motor neuron demyelinating disorder that causes spinal nerve degeneration. It is a subset of disease which primarily affects the brain stem, motor cortex, spinal cord, and corticospinal tract.

- The increase in the number of patients with ALS has led to increasing demand for effective therapy and a particular diagnosis, which can help this motor neuron dysfunction.

- Currently, there are several diagnostic tests available to diagnose ALS, including blood and urine tests that are laboratory tests which analyse blood and urine samples that include spinal tap lumbar puncture, electromyogram, nerve conduction study, MRI etc. The major limitations of all these diagnostic tests are quite laborious, intrusive, and costly. As a result, people avoid or delay getting diagnosed, causing their symptoms to worsen. Furthermore, no single test can provide a definitive diagnosis of ALS, which might lead to the disorders progression and worsening. This could be a prominent of mortality rate.

- This research will serve as a foundation for future research on these signaling cascades in other neurological disorders such as MS, OCD, and Bipolar Disorder. Using the target modulators outlined in this review to influence these signaling cascades could be a potential therapy for various neurological disorders, including MS, OCD, and Bipolar Disorder.

Biography

Mr. Sumit Kumar has completed his bachelor in pharmacy at the age of 21 years from ISF College of Pharmacy, Moga, INDIA. Currently, he is a budding research scholar pursuing his Masters in Pharmacology at the age of 23 years from INDIA. He presented posters in international conferences. His area of interest is translational neuroscience and at present he is working on motor degenerative disorder- ALS (Amyotrophic lateral sclerosis). He is investigating the neuroprotecting potential of various phytoconstituents.

Saranya Jayapalan

Department of Bioinformatics, Pondicherry University, India

Comparative cognitive profile to decode the candidate genes and eQTLs associated with schizophrenia and other psychiatric disorders

Cognitive functions possess important roles in mental and physical well-being of human. Healthier cognition functions is directly associated with lower risk of certain psychiatric disorders and physical illness in the later life course with reduced mortality risk. Cognitive decline is a central manifestation which is commonly seen in patients suffering with Schizophrenia and other psychiatric disorders causing focal impairment to brain structures mediating mild/moderate/ severe cognition dysfunction which leads to affect memory, personality, behavior and decision making abilities etc. Over the years, numerous studies reported on psychiatric disorders, despite the incidence of cognitive impact in Schizophrenia and other psychiatric disorders, the candidate miRNAs and SNPs respect to cognition is reported less. In this study, an integrated pipeline was developed with text mining, GWAS and functional annotation studies were employed to identify gene/miRNA biomarkers and functional SNPs in the GWS loci and eQTL to decipher the genetic mechanism behind the cognitive function in Schizophrenia and other three major psychiatric disorders. Gene sets enrichment analysis was performed to discover the functional significance of predicted targets with cognitive effect in psychiatric disorders. Gene based association analysis and gene expression heatmap of different tissue types for the target genes have been identified. Disease associated eQTLs analysis demonstrated the tissue specific expression levels to the corresponding disorders. The study will give insights into the interrelationships between significant pathways and genes to explore the biological mechanisms and related functions from the relevant cortical region with respect to Schizophrenia and other three major psychiatric disorders. An enhanced comprehend of mechanisms facilitating the cognitive dysfunction in psychiatric disorders may aid in the characterization of disease scenario and in the development of new cognitive treatments, since the cognitive treatment and management still remains a lack in therapeutic need. These results will provide insight into putative mechanisms related to improvement of cognitive ability in patients with Schizophrenia and other psychiatric disorders, in order to find new potential drug targets.

Keywords: Cognition, Schizophrenia, Psychiatric disoders, GWAS, eQTLs, SNPs

Audience Takeaway

- Understanding the role of cognitive impairment in Schizophrenia and other psychiatric disorders, predicting the candidate genes and their functions and pathways.

- Identification of the role of genetic biomarkers in the progression of Schizophrenia and other psychiatric disorders.

- Comparative analysis on major four neuro -psychiatric disorders to unravel the association of cognitive impairment.

Biography

Dr. Saranya Jayapalan, received PhD degree in Bioinformatics from Bharathiar University in 2016. Now she is working as Post- Doctoral Research Associate at Pondicherry University. She has published several research papers, two book chapters in reputed scientific journals and developed two protein kinase databases. Her area of interests includes computational biology, NGS analysis, systems biology and structural biology. Currently she is working on Genome Wide Association Studies based on neuropsychiatric disorders in association with cognition.

Rafaela Vasiliadou

The Open University, United Kingdom

Microfluidic electrochemistry/mass spectrometry : Potential applications in neurological disorders

Electrochemistry/Mass Spectrometry is a pure instrumental approach that can mimic the metabolic process in humans. Metabolic reactions are mainly oxidations and reactions, thereby electrochemistry is an ideal alternative to the current *in vitro* and *in vivo* methods. Hyphenating electrochemical methods with Mass Spectrometry enables the synthesis and detection of metabolites[1]. Microfluidics is a highly interdisciplinary field combining science with engineering. Microfluidic devices also known as chips are tiny devices operating at extremely low volumes (µL to pL), in which fluids are transported, mixed and separated. The combination of Electrochemistry with Microfluidics offers portability, faster reaction times, low volumes of reagents and wastes, reproducibility and affordability. The oxidation of dopamine leads to the formation of dopamine o-quinone and aminochrome. Toxic metabolites that are probably the causal agents of Parkinson's disease.

According to literature, dopamine o-quinone and aminochrome induce 1) Protein degradation dysfunction 2) Mitochondria dysfunction 3) Alpha synuclein aggregation and 4) Oxidative stress[2]. Herein, the successful mimicry of dopamine oxidation into dopamine o-quinone and aminochrome is described by Microfluidic Electrochemistry/Mass spectrometry, as an instrumental approach to monitor the reactive metabolites in less than ten minutes. Traditional *in vitro* and *in vivo* methods require several months of tedious procedures. The cost of chips is affordable and the unethical use of animal models can be significantly reduced in medicinal and pharmaceutical research[1]. The technique has potential applications as an affordable and fast screening method for the identification of reactive metabolites in neurological disorders.

1.	Vasiliadou, R.; Nasr Esfahani, M.M.; Brown, N.J.; Welham, K.J. A Disposable Microfluidic Device with a Screen-Printed Electrode for Mimicking Phase II Metabolism. *Sensors* 2016, 16, 1418.

2.	Monoz, P.; Melendez, C.; Paris, I.; Sequra-Aquilar, J. Molecular and neurochemical mechanism dopamine oxidation to 0-quinone in Parkinson disease pathogenesis. *Toxicity and Autophage in Neurodegenerative Disorders*; Springer: Basel, Switzerland, 2015, 205–223.

Audience Takeaway

- Familiarize with the emerging fields of Microfluidics and Electrochemistry/Mass spectrometry towards the mimicry of endogenous and exogenous metabolism in humans. An affordable and fast instrumental approach that identifies reactive metabolites in just a few minutes. The microfluidic devices can be used as screening methods to identify reactive metabolites, which might be the causal agents of neurological disorders or diseases. Researchers, academics and doctoral students can benefit from the approach since it can be improve their study design, acquire data relatively fast, reduce the consumption of expensive reagents.

- Introduction to the main categories of reactive metabolites. Researchers, academics and doctoral students will understand how the reactive metabolites can cause Parkinson disease. Why is important to understand their formation and mechanisms of action. Important parameters for diagnosis, treatment drug design and development.

- Interdisciplinary in Neurology. The combination of Chemistry, Biology, Medicine and Engineering can answer important neurological questions/hypothesis.

Biography

Dr. Vasiliadou holds a Bachelor degree in Human Biology and a Master degree in Analytical and Forensic Chemistry from the University of Hull (UK). She received her Doctorate degree in Analytical Chemistry at the same institute in 2016. She spent her post-doctorate years in the group of Professor Nick Lane, at University College London (UK). In 2020, Dr Vasiliadou joined the Open University as a Lecturer in Biological Chemistry. Her research focuses on Microfluidics and Electrochemistry for applications in Biochemistry, Medicine and Pharmaceutical Sciences.

Wing Kiu Chou*, Abhinav Balasubramanian

Norwich Medical School, United Kingdom

Polymorphisms of the interleukin 6 gene and susceptibility in multiple sclerosis

Background: Multiple Sclerosis (MS) is a chronic, degenerative inflammatory disease of the central nervous system characterised by demyelination. Genetic factors have been implicated in its pathogenesis, however their association with MS remains unclear. Genetic variants in the inflammatory mediator, interleukin 6, may determine why some patients are more susceptible to developing the disease while others are not. We undertook a systematic review of Single Nucleotide Polymorphisms (SNPs) in the IL-6 gene to determine their relationship with disease susceptibility.

Methods: We systematically searched Embase, MEDLINE, Cochrane and Google Scholar databases. Studies which investigated SNPs in interleukin-6 and their associations with MS were included. A meta-analysis was conducted in selected variants reported by at least two studies.

Results: Twelve studies which reported on three genetic variants of interleukin 6 gene were studied. One study reported increased risk of MS in patients who carry the G allele for IL-6 572 G/C to statistical significance. Whereas, IL-6 597 G/A was reported to not have any association with disease susceptibility reported by one paper. However, both variants have not been replicated by other studies. IL-6 174 G/C was reported by seven studies respectively and selected for meta-analysis. In our meta-analysis, 1062 cases of MS and 795 healthy controls were included from seven studies. We estimated a pooled odds ratio of 1.14 (95% CI 1.01-1.29), $p<0.01$ for the IL-6 174 G/C (C vs G) polymorphism and susceptibility of MS

Conclusion: Our meta-analysis suggests the polymorphism of IL-6 174 G/C are associated with increased susceptibility of MS.

Biography

Mr Wing Kiu Chou MRSPH AFHEA is an Associate Tutor for Norwich Medical School. He is an intercalating (MBBS) medical student undertaking a Masters of Research (MRes). His degree is supported by the prestigious Wolfson Scholarship awarded jointly by the Wolfson Foundation Charity and the Royal College of Physicians (London). He has been awarded £28,793 in academic scholarships, prizes and funding to date.
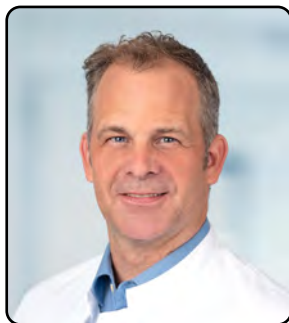
Andreas Till*, Johannes Jungverdorben, Beatrice Weykopf, Vesselina Semkova, Lea Flitsch, Wiebke K. Fenske and Oliver Brüstle

University Hospital of the University of Bonn, Germany

In vitro modelling of brain development and neurodegenerative diseases

Given the complex etiopathology of neurodegenerative diseases and the limited access to vital human brain cells as suitable research platform, there is an increasing demand of innovative human-specific model systems that help elucidating the causative mechanisms of these disorder and open the avenues for novel prevention and/or treatment strategies. The advent of cell reprogramming has enabled the generation of induced Pluripotent Stem Cells (iPSCs) from patient fibroblasts or blood cells and their subsequent differentiation into tissue-specific cells, including neurons and glia. This approach in combination with in vitro genome-editing technology is suitable to recapitulate disease-specific phenotypes but also neurodevelopmental aspects in classical cell culture paradigms and thus represents an invaluable asset for developmental research, disease modelling and drug validation in the framework of personalized medicine. Here we demonstrate how in vitro modelling of neurodevelopment and neurodegeneration in classical two-dimensional and 3D model systems may unveil underlying causative mechanisms and target pathways.

First, we demonstrate that the cytoprotective NRF2 signaling pathway is upregulated and activated during neuronal differentiation from neural stem cells to maturing neural populations. We continue to show how this pathway is regulated and able to protect neural populations from environmental insults, e.g. oxidative stress, during development. Next, using iPSC-derived dopaminergic neurons from a Parkinson's Disease (PD) patient carrying the causative LRRK2 G2019S mutation, we demonstrate that mutant carrier cells display defects in homeostatic turnover of mitochondria ('mitophagy') as early as at day 8 of differentiation. These data pinpoint one important causative mechanism in the pathology of PD, i.e. quality control of mitochondria, and emphasize the vital role of mitophagy regulation during early steps of neuronal development. Moreover, we asked which subcellular pathways contribute to formation of Neuronal Intranuclear Inclusions (NIIs) of mutant Ataxin-3 protein in an iPSC model of polyglutamine disease (spinocerebellar ataxia type 3/ SCA3). Inhibition of autophagy (an essential lysosomal degradation system) resulted in a significant increase of NIIs in the susceptible cell population, and the build-up of NIIs could be abated by pharmacological upregulation of autophagic flux. As outlook, we present our approach on how to dissect the connection between diet, the gut microbiome composition, brain circuitry and manifestation of metabolic diseases such as obesity and its comorbidities (including dementia) using a translational approach in combination with in vitro model systems. In summary, our work emphasizes the usability of innovative in vitro models for assessment of development, disease pathways and even organ crosstalk.

Audience Takeaway

- Which in vitro model systems are nowadays available to study disease mechanisms in the dish.
- Which histological and molecular features could be used in the clinic to interpret pathological findings.
- Which shared cellular and molecular events underly the pathogenesis of neurodegenerative diseases.
- How preclinical drug screening can be performed on authentic human cells.

Biography

Andreas Till is a dedicated cell biologist whose research focuses on cellular stress pathways associated with human diseases. He received his PhD at the University of Kiel, Germany, where he studied immunesignaling and pathogen detection. After receiving a research fellowship, he worked at the University of California San Diego where he focused on selective autophagy. Next, he joined the University of Bonn as lecturer for Molecular Biomedicine, and was later appointed lab head in the Section for Metabolic Diseases. Over the last years, he has specialized on in vitro cell models for stress pathways such as antioxidative responses, autophagy and metabolic reprogramming. Recent projects aim to elucidate the interconnection between the gut microbiome and neural circuits regulating energy homeostasis in obesity. Dr. Till has published more than 60 papers and presented his research at several international conferences in Europe, USA, Japan and China.

Hamza Wadi[a]* , Rabab Shannaq[b], Tareq Abu-Libdeh[a], Rama Hailat[b], Sajeda Albustanji[c], Radi Tofaha Alhusseini[a], Amer Rawajfeh[d], Zidan Alsaleh[e], Ghina Zaidan[g], Anas Hassouneh[a], Salsabeel Abughanmi[a], Lina Al-Twal[h], Ala' Dandis[i], Shorooq Almaaitah[e]

[a]AlBashir Hospital, Medicine, Jordan

[b]Princess Basma Teaching Hospital, Jordan

[c]Assalt Governmental Hospital, Jordan

[d]Mutah University, Psychiatry, Jordan

[e]Mutah University, Faculty of Medicine, Amman, Jordan

[f]Mutah University, Faculty of Medicine, Alkarak, Jordan

[g]Prince Rashid Ben Al-Hasan Military Hospital, Jordan

[h]Alnadeem hospital, Jordan

[i]Applied science private university, Jordan

The psychological impact of sleep quality on health care workers during COVID-19 in Jordan

Background and aims: Sleep quality is a key aspect of the overall psychological health. Sleep quality can affect mental health in many aspects, causing major psychological disorders, such as: depression and anxiety.

Objectives: to assess the overall psychological impact of sleep quality on health care providers in Jordan during COVID-19 pandemic.

Methods: This is a descriptive cross-sectional multicenter hospital-based study conducted in Jordan from the period of 2/7/2021 to 12/7/2021. 418 participants were included in the study. Data collection was done online via Google form. A questionnaire was used containing 4 validated scales, the insomnia severity index (ISI), Epworth sleepiness scale (EPS), generalized anxiety disorder-7 (GAD-7), patient health questionnaire-9 (PHQ-9).

Results: (56.7%) were males, (43.3%) were females. (28.8%) were residents, followed by (20.3%) were nurses, (19.7%) were general practitioners, and (16.8%) were specialists. About (44.8%) of the participants were diagnosed with COVID-19 and (75.4%) managed patients with COVID19. (32%) suffered from moderate difficulty falling asleep, and (30%) have moderate difficulty staying asleep. About (21.2%) said that there sleep problems much interfere with their daily functioning. (34.4%) feel nervous and anxious more than half of the days, while (26%) had the same problem nearly every day. (34.5%) feel tired more than half of the days, while (26%) suffered from the same issue nearly every day.

Conclusions: The results of this study support the view that poor sleep quality can affect the overall mental health dramatically. Poor sleep quality can cause a psychological problems, such as: depression, and anxiety.

Biography

Dr. Hamza Wadi studied Medicine and surgery at the Mutah University, AlKarak, Jordan and graduated as MD in 2020. He then completed his internship training at Albasheer hospital, Amman, Jordan. Dr. Hamza worked as lecturer for basic clinical sciences for 2 years. His Abstract was presented at the world congress of neurology, Rome, Italy 2021. The abstract was published in the official journal of the world federation of neurological sciences (The journal of neurological sciences).

Juliana Fort*, Justin Hardin, Caroline Sagrera, Michael Kenny

Louisiana State University Health Shreveport, United States

Innovative student and faculty created and facilitated experiential wellness workshops for medical students, and neurology residents, A model that can be adapted for the neurological sciences

- Burnout is higher among both physicians and basic scientists due to work demands and loss of some social connectivity during COVID and only adding obligations of wellness surveys or training can add to the workload burden and so care needs to be put into the format of how to do so in a way that promotes learning, relaxation, social connectiveness, and mental, physical, spiritual, and digital wellbeing.

- Experiential innovative learning facilitates creative, divergent problem solving, retention of learning and is the most effective way to teach but is hard to adopt and implement at institutions unless it is regarded as the norm rather than innovative.

- Involving students or target audience members in the creation of wellness programs and in the facilitation of those programs is more effective than simply delivering the programs to them as more passive participants.

- This workshop will demonstrate examples of student workshops with experiential activities, using a few examples of expressive arts such as: art, movement and role play to collaborate to help reduce burnout, enhancing creativity, wellbeing, learning and productivity in neurological scientists/students.

Biography

Juliana Fort, MD, MPH, MBA, medical student clerkship director and Clinical Associate Professor in Psychiatry at LSUHS in Shreveport is board certified in child and adolescent, geriatric, forensic, and addictions psychiatry. With a MA in mental health counseling with a specialization in drama therapy from Lesley University, a MA in creative writing, she is a play therapist / supervisor and enjoys co-facilitating workshops with students and colleagues, and psychiatry/neurology residents. Using expressive arts: movement, improvisation, music, art, creative writing, and poetry enriches experiential teaching, wellness, medicine and the arts/humanities, psychotherapy, and personal growth. To collaborate with virtual workshop: juliana.fort@lsuhs.edu

Nicolas Casadei [1,2]*, Jakob Admard [1,2], Michaela Pogoda [1,2], Tobias Haack [1,2], Christopher Schroeder [1,2], Stephan Ossowski[1,2], Olaf Riess[1,2]*

[1]Institute of Medical Genetics and Applied Genomics, Eberhard Karls University Tübingen, Germany

[2]NGS Competence Centre Tübingen (NCCT), Eberhard Karls University Tübingen, Germany

Implementing RNA analysis in human genetics : From gene expression profiling to integration into genome analysis improvement

First applications of RNA analysis in human genetics aimed mainly to detect splicing defects or loss of gene expression. More recent developments of RNA-analyses are focusing on the analysis of gene expression changes in disease as biomarkers, in response to treatments or to predict targeted medication as in cancer therapy. By using blood RNA-seq analysis, we developed a strategy enabling both the comprehensive analysis of disease and the validation of genomic events in genetic or sporadic disorders. We are using high-throughput and standardized protocols to complementary monitor gene expression in blood and to systematically investigate the impact of mutations detected using whole-genome sequencing in splicing and in regulatory elements in rare diseases. RNA-seq was optimized for tumour material in clinical oncology to prioritize therapeutic relevance of somatic mutation detected by NGS-based DNA panel sequencing by investigating loss of heterozygosity as well as the identification of gene fusion at the RNA level. We will present how ongoing developments using unique molecular identifiers, single-cell sequencing or long-read sequencing will lead to novel application of RNA diagnostics such as the identification of infection or immune response related to therapy.

Audience Takeaway

- In this oral presentation, I will introduce how RNA-sequencing can be used in diagnostics to validate genomic variants and to prioritize actionable genes.

- The presentation aims to briefly introduce the audience to more classic modern genetic diagnostic, highlighting current difficulties and proposing alternative solutions.

- Description of the solutions using latest technological developments will be integrated by concrete applications in human genetics research.

- All the tools and solutions proposed in this presentation are available either as commercial products or research services. They can be implemented in modern genomic facilities or genetic research laboratories.

Biography

Dr. Nicolas Casadei studied Organic Chemistry at Besancon University, France. He graduated as an MS in Pharmacology in 2008. He joined the research group of Prof. Riess at the Institute of Human Genetics of the University Hospital of Tübingen, Germany where he received his PhD degree in neuroscience in 2015. After a one-year postdoctoral fellowship, Dr. Casadei is leading the genomics core facility at the University Hospital Tübingen and is coordinating the NGS Competence Center. He received an MBA in 2020 at the University Bicocca, Italy. The core facility performed over 100 studies in 2021 and Dr. Casadei published 28 research articles.

Baojin Ding

Louisiana State University Health Sciences Center in Shreveport, USA

Modeling neurological diseases using patient-derived neurons

The limited access to human neurons greatly impedes the progress of research in neurological diseases. Although animal models provide insights into disease mechanisms, significant species-dependent differences exist, and animal models only mirror the limited aspects of the pathophysiology of human diseases. It is believed that these species-dependent differences caused the high failure rate in clinical trials that have been derived from successful results in animal models. Excitingly, reprogramming of human neurons from adult fibroblasts overcomes this limitation and provides an unprecedented approach in deciphering the molecular pathogenesis underlying neurological diseases. Recently, we have reported two techniques for the generation of patient-specific neurons. One is the direct conversion from patient fibroblasts using lentiviral delivery of transcription factors, and the second method is induced Pluripotent Stem Cells (iPSCs)-based induction and differentiation. Using different combinations of reprogramming factors, different neuronal subtypes could be generated, such as Generic Neurons (iGNs), Motor Neurons (iMNs), And Cholinergic Interneurons (iChINs). We modeled the movement disorder DYT1 dystonia using patient-specific neurons and recapitulated the disease-dependent cellular deficits, including deformed nucleus, impaired neurodevelopment, disrupted nucleocytoplasmic transport, and the unexpected finding of nuclear Lamin B1 mislocalization. This study provided the novel insights into the pathogenesis of dystonia and revealed the high value of patient-derived neurons in modeling neurological diseases.

Audience Takeaway

- Two novel techniques for the generation of patient-specific neurons.

- The generation of different neural subtypes via different reprogramming factors.

- The differences and similarities of directly converted neurons and iPSC-derived neurons in modeling neurological diseases.

- The novel insights into the pathogenesis of DYT1 dystonia in patient-specific neurons.

Biography

Dr. Baojin Ding received his bachelor's degree in Medicine (MD equivalent) and master's degree in Clinical Laboratory in China. After he received PhD in Biochemistry and Molecular Biology from Louisiana State University (LSU), he did postdoctoral training in Neuroscience at the UMass Medical School and worked as a research faculty in the UT Southwestern Medical Center. In 2018, Dr. Ding obtained an Assistant Professor (tenure-track) at the UL Lafayette and then relocated to LSU Health Shreveport. The research in Dr. Ding's laboratory is focusing on Molecular and Cellular Neuroscience and Neurological Diseases, and currently funded by NIH and DoD.

POSTERS

DAY 02

5TH EDITION OF
INTERNATIONAL CONFERENCE ON

NEUROLOGY AND
NEUROLOGICAL DISORDERS

15–16 JUNE

Yuya Aotsuka*, Kazumoto Shibuya, Sonoko Misawa, Tomoki Suichi, Keigo Nakamura, Hiroki Kano, Marie Morooka, Ryo Otani, Satoshi Kuwabara

Department of Neurology, Chiba University, Japan

long term prognosis of chronic inflammatory demyelinating polyradiculoneuropathy : Five years follow up

Objective: Recently, there are few reports on the long-term prognosis of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). We aimed to clarify the long-term prognosis and prognostic factors of CIDP.

Methods: Fifty-one consecutive patients with CIDP who had attended our department since 2000 and for whom the overall neuropathy limitation scale was available at the time of initial treatment and 5 years after the start of treatment were included in this study. Anti-Neurofascin155 antibody-positive and anti-Contactin1 antibody-positive patients were excluded. Patients were divided into two groups by median ONLS after 5 years of treatment; good prognosis group (5 years ONLS 0 or 1) and poor prognosis group (5 years ONLS over 2), and factors predicting the prognosis were retrospectively examined.

Results: The fifty-one CIDP patients consisted of twenty-seven males with a mean age at onset of 40.7 years. The clinical phenotypes of CIDP were typical CIDP (34 patients) and multifocal CIDP (17 patients). Male, shorter disease duration, good responsiveness to primary treatment, absence of diabetes, and prolonged ulnar nerve distal latency/F wave latency tended to be associated with a better prognosis (p value < 0.1). Subsequently, multivariate analysis including these factors showed that shorter disease duration, good responsiveness to primary treatment, and absence of diabetes were significantly associated with better prognosis (adjusted p value < 0.05).

Conclusions: Shorter disease duration, responsiveness to primary treatment, and absence of diabetes may be prognostic factors for predicting a favorable outcome.

Audience Takeaway

- To be able to learn about the prognostic factors of CIDP.
- Understand how to predict the prognosis of CIDP.
- As Nodopathy is now defined as a disease independent of CIDP, this research may provide a clue to reclassify CIDP, a heterogeneous group of diseases, and to clarify a new pathogenesis.

Biography

Dr. Aotsuka studied medicine at Chiba University in Japan and graduated in 2012. After that, he became a neurologist in 2014 after 2 years of training at Chiba City Aoba Hospital, and after 6 years of clinical experience, he entered the doctoral program at Chiba University Graduate School of Medicine and Pharmaceutical Sciences in 2020. He is mainly engaged in clinical research and epidemiological investigation of immunological peripheral nerve diseases.

T. Karthikeyan

National Institute of Mental Health and Neurosciences, India

Therapeutic effectiveness of sensory cues versus without sensory cues on gait dysfunction of parkinson's disease

Parkinson's disease is a neurological movement disorder caused by the progressive degeneration of dopamine producing cells in the substantianigra. Post PD patient develop gait instability and gait characteristics changes noted mainly increased cadence, reduced stride length, develop freezing gait and reduced walking speed, velocity.

Objective: To find out the effectiveness of sensory cues on the gait impairment in patients with Parkinson's disease.

Methodology: Total 30 PD subjects considered for this study, Each Group dividend equally into 15 participants in each groups. Simple random sampling technique used in this study. The sampling procedure convenient sampling methods used in this study. Research outcome: Time Up and Go Test (TUG)

Results: The pre and post test values were assessed by BBS, TUG in group A and group B. The mean difference value of TUG is 42 and 37($P<0.5$).

Conclusion: The author concluded that the Sensory cueing techniques significantly better form of treatment than non sensory cues in terms of improving gait in patients with parkinson's disease.

Audience Takeaway

- This specific treatment approach integral component of functional rehabilitation/physiotherapy techniques. The investigator applied sensory cues treatment technique (Gait/Walking pattern corrected) for the management of gait dysfunction PD. This treatment strategy which helps to improve walking pattern of PD.

- Physiotherapy in clinical practice & research in physiotherapy, this specific treatment protocols could be utilize to manage gait dysfunction of PD.

- This specific research protocols need to be reevaluate with larger sample with RCT study.

- This specific treatment protocols approach help to improve walking pattern, mobility, balance of PD.

- Presently I have used simple random sampling technique utilize in this study.

- Overall this research help to improve gait charterstic changes (Cadence, stride length, step length,), Overall gait speed, accuracy, balance, well- being, livelihood of the PD.

Biography

Dr. T. Karthikeyan working as physiotherapist with 22 years rich experience at NIMHANS, Bangalore, Karnataka, India. He completed my terminal degree of D.LITT in 2018 from Tumkur University, Tumkur. His doctoral degree completed 2012 at RKMVU, Coimbatore. His scholar activities /track record publication include: publication(Books-60, Journal-100). He received 25 medals professional recognition.

Begüm Bulgurluoğlu*, Özlem Bozkurt

Hisar Schools, Turkey

Shopping and working with AI- a psychological perspective

Artificial Intelligence (AI), often known as the fourth industrial revolution 4.0, will alter not only the way we do things and interact with people, but also our understanding of ourselves. Without directly affecting human connections AI, sparked a massive social shift. Modern AI has a huge impact on how we do things and how we interact with one another. The future of humans and artificial intelligence (AI) and how they affect each other is a growing concern and source of fear. While experts predict that artificial intelligence will improve the lives of most people over the next decade, many people are concerned about how AI will impact what it means to be human, productive, and free; This also raises the question "What happens to our free will ? The ain thing about being a human is having a free will, but does AI limit our free will?"

Today, in the new 2022, we have Artificial Intelligence that has now replaced many types of workers/laborers with its increasing automation levels in diverse sectors (from the automotive industry to agriculture). Moreover, during and post Covid19 pandemic, artificial intelligence has shaped the ways we shop online and helped us make our decisions. These developments make us question why we really need AI. Do we really need artificial intelligence for everything? Professor Benoit Monin, from Stanford University Psychology Department, has an ongoing project that compliments the same university's research in understanding and developing better the ways humans and artificial intelligence can benefit from each other now and in the future. His upcoming research, with co-author Ph.D. student Erik Santoro, titled "The Impact of Artificial Intelligence on Perceptions of Humanhood" opens up the discussion that this essay wants to address: In what ways does and will AI impact human psychology? This work specifically aims to investigate the ways AI influences human psychology in the areas of consumption (shopping) and employment (labor).

Keywords: Artificial Intelligence, Psychology, Labor, Shopping, Free Will, Human, Industrial Revolution 4.0

Audience Takeaway

- How humans and artificial intelligence affect each other.
- How AI affects human psychology.
- The effect of artificial intelligence on shopping.
- The effect of artificial intelligence on jobs.
- AI from human perspective.

Biography

Begüm Bulgurluoğlu was born in 2005 in Istanbul, Turkey. She graduated from Hisar High School in 2023.

Georgios Matis

University of Cologne, Germany

Chronic pain, Intrathecal therapy and ziconotide : An overview

Ziconotide is a synthetic, water-soluble cone snail venom-derived peptide with a molecular weight of 2,639 Daltons. It is a nonopioid analgesic that selectively binds to N-type voltage-sensitive calcium channels on primary nociceptive afferent nerves in the dorsal horn of the spinal cord. This mechanism releases analgesic neurotransmitters into the synaptic gap and subsequently blocks pain signal transmission. Ziconotide does not easily cross the blood-brain barrier, instead revealing its highly potent antinociceptive effect only after intrathecal administration. Because it has a narrow therapeutic window, careful dose titration, and a lag time to allow for onset (and offset) of analgesia and adverse effects are required. The presentation will focus on a recently published consensus proposal and highlight the potential of this drug as well as the areas where additional experience is needed.

Audience Takeaway

- Expand the knowledge on possible neuromodulation therapies.
- Learn how intrathecal therapy can help patients with chronic pain.
- Learn how a non-opioid drug (Ziconotide) could be a viable treatment option.
- Learn about the advantages and disadvantages of Ziconotide.
- Help physicians provide one more therapy to their chronic pain patients.

Biography

Dr. Georgios Matis is a senior consultant for neurosurgery. He leads the chronic pain / spasticity sector of the Department of Stereotactic & Functional Neurosurgery in the University Hospital of Cologne. He has been trained in Greece (General University Hospital of Alexandroupolis, G. Papanikolaou General Hospital of Thessaloniki & 417 Army Equity Fund Hospital of Athens), USA (Department of Neurosurgery, Weill Cornell Medical College, New York, NY), Switzerland (Department of Neuroradiology, University Hospital of Zurich, Zurich) and Germany (Department of Stereotactic & Functional Neurosurgery, University Hospital Cologne, Cologne). Dr. Matis is a member of two medical associations (Thessaloniki, Greece & North Rhine, Germany) and also a member of the German Neuromodulation Society (DGNM) and the International Neuromodulation Society (INS).

He serves as reviewer for many international journals and is Editorial Board member for Neuromodulation: Technology at the Neural Interface and Interventional Pain Medicine and Neuromodulation. He holds the position of Editor-in-Chief of the Internet Journal of Neurosurgery. Dr. Matis has published many articles in Greek and international Pubmed-indexed journals and hold many lectures as invited speaker in numerous international congresses and webinars. At the same time, he is Public Education Committee member of the International Neuromodulation Society. Dr. Matis is involved in many international clinical studies and has been active as instructor for many colleagues in Germany and abroad. He is also an active member of the medical advisory board of the German CRPS Support Group and member of several online consultation platforms. He is actively involved in social media trying to raise awareness about spinal cord stimulation and neuromodulation.

Participants List

Andreas Till University Of Bonn, Germany	**45**
Armaan Saith Temple University, United States	**31**
Baojin Ding Louisiana State University Health Sciences Center-Shreveport, United States	**49**
Begum Bulgurluoglu Hisar Schools, Turkey	**53**
Chonghe Jiang The Sixth Affiliated Hospital of Guangzhou Medical University, China	**12**
Georgios Matis University of Cologne, Germany	**54**
Hamza wadi Mutah University, Jordan	**46**
Jose-Luis Gomez-Ariza University of Huelva, Spain	**23**
Juliana Melody Fort LSUHSC- Shreveport, United States	**37**
Juliana Melody Fort LSUHSC-Shreveport, United States	**47**
Karthikeyan T National Institute of Mental Health and Neurosciences, India	**52**
Ken Ware, NeuroPhysics Therapy Institute, Australia	**7**
Kenneth Storey Carleton University, Canada	**38**
Lori Gray Western Michigan University, United States	**30**

Manuela Marcoli University of Genoa, Italy	**25**
Maria Eulina Quiliao Instituto da Criança of Hospital das Clinicas de São Paulo/USP, Brazil	**29**
Maria Joao Cabral Sacadura Catholic University of Portugal and University of Coimbra, Portugal	**19**
Ming Zhang Institute of neurological diseases, China	**16**
Natalia Surina Lomonosov Moscow State University, Russian Federation	**27**
Nicolas Casadei Eberhard Karls University Tubingen, Germany	**48**
Pengxu Wei National Research Centre for Rehabilitation Technical Aids, China	**15**
Rafaela Vasiliadou The Open University, United Kingdom	**43**
Rocío Fernández-Serra Universidad Politécnica de Madrid, Spain	**21**
Roshanak Mehdipour Isfahan University of Medical Sciences, Iran (Islamic Republic of)	**28**
Sabine J Bischoff University Hospital Jena, Germany	**20**
Saranya Jayapalan Pondicherry University, India	**42**
Sergey Suchkov Moscow State University of Food Industry (MGUPP), Russian Federation	**33**
Shinyi Chloe Chiou University of Birmingham, United Kingdom	**17**
Stephen Grossberg Boston University, United States	**10**

Sumit Kumar ISF College of Pharmacy, India	**41**
Thomas J Webster Interstellar Therapeutics, United States	**36**
Torbjorn Backstrom Umea University, Sweden	**35**
W S El Masri Keele University, United Kingdom	**8**
Wing Kiu Chou University of East Anglia, United Kingdom	**44**
Xi Wang Zhejiang University, China	**14**
Yuya Aotsuka Chiba University, Japan	**51**

UPCOMING CONFERENCES

6th Edition of International Conference on
Neurology and Brain Disorders
October 24-26, 2022 | Orlando, Florida, USA |
Hybrid Event
https://neurologycongress.com/

7th Edition of International Conference on
Neurology and Neurological Disorders
June 22-24, 2023 | Rome, Italy | Hybrid Event
https://neurology.magnusconferences.com

Questions? Contact

+1 (702) 988-2320 or

Inquires: neuro@magnusconference.com

For Registration:

https://neurology.magnusconferences.com/register