magnus Group
Share, Receive & Grow !

NEUROLOGY

12TH EDITION OF INTERNATIONAL CONFERENCE ON

NEUROLOGY AND NEUROLOGICAL DISORDERS

June 22-24, 2026
Barcelona, Spain

12TH EDITION OF

International Conference on

NEUROLOGY AND NEUROLOGICAL DISORDERS

HYBRID EVENT

22-24 JUNE 2026

NEUROLOGY

JUNE 22-24 | 2026

BOOK OF ABSTRACTS

Index

Keynote Speakers

Keynote Speakers

Alan C Jackson
University of Calgary, Canada

Bernd Blobel
University of Regensburg, Germany

Carlos Alberto Mangone
Santojanni General Hospital, Argentina

Edie Raether
NeuroShifts and Wings for Wishes Academy, United States

Jaqueline Tuppen
COGS Club, United Kingdom

Jonathan Eskenazi
Cedars Sinai / UCLA, United States

Keynote Speakers

Ken Ware

NeuroPhysics Therapy, Australia

Khue Vu Nguyen

University of California, United States

Luiz Moutinho

University of Suffolk, United Kingdom

Robert B Slocum

University of Kentucky HealthCare, United States

Roger H Coletti

Interventional Health, PA, United States

Salvador Ventura

Autonomous University of Barcelona, Spain

Sergey Victorovich Suchkov

N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia

Sid O Bryant

Texas College of Osteopathic Medicine and University of North Texas Health Science Center, Fort Worth, United States

W S El Masri

Keele University, United Kingdom

Zhenhuan Liu

Guangzhou University of Chinese Medicine, China

Welcome Message

Professor Alan C. Jackson

Clinical Neurosciences University of Calgary, Alberta, Canada

Dear Conference Attendees

It is an honor and great pleasure to welcome clinicians and researchers to the 12th International Conference on Neurology and Neurological Disorders in Barcelona. A variety of neurologic topics will be covered from bench to bedside and the conference will allow an important exchange of knowledge and motivate insightful discussions across a variety of disciplines and borders. The development of novel therapeutic strategies to address many diseases requires experimental models to understand basic mechanisms. This applies to my research area in order to develop effective therapy for human rabies. I wish all attendees an inspiring and productive conference that will be highly rewarding.

Welcome Message

Prof. Dr. Habil. Bernd Blobel

Former Head of the German National eHealth Competence Center and the International Interdisciplinary PhD and Post Doc College at the University of Regensburg, Medical Faculty, Regensburg, Germany

Dear Colleagues and Friends

It is an honor, to welcome you on behalf of the Scientific Committee to the 12th Edition of International Conference on Neurology and Neurological Disorders, (Neurology-2026), which will be held on June 22-24, 2026, in Barcelona, Spain, and Virtually. The Neurology-2026 Conference will address the pressing Challenges and Innovations in Neurology and Patient Care. The healthcare paradigm advanced from empiric and phenomenological through evidence-based and person-centered medicine to personalized, preventive, predictive, participative precision medicine (P5M). Thereby, we must consider and understand the individual health status, conditions and genetic and genomic dispositions in personal social, occupational, environmental and behavioral context at any level from elementary particles up to society and universe. This requires the inclusion of actors from multiple domains as well as the subject of care with their own methodologies, languages, ontologies, education and skills. I have developed the model and framework for managing integration and interoperability in such highly complex, dynamic, context-aware, multi-disciplinary transformed healthcare ecosystem, meanwhile standardized in ISO 23903. Because of its formal and foundational nature, the methodology is not restricted to health and social care, but has been successfully deployed already in many other domains. Welcoming you in Barcelona, I'm sure that this conference will provide an excellent platform to discuss trends, innovations, and best practices in clinical care, healthcare technologies, research and education. So enjoy a successful event.

Welcome Message

Jaqueline Tuppen

Admiral Nurse and Specialist Practitioner,
United Kingdom

Dear Conference Visitors and Delegates

Welcome to the "12th Edition of International Conference on Neurology and Neurological Disorders" (Neurology 2026), scheduled for June 22-24, 2026, in Barcelona, Spain and Virtually. Centered around the theme "Connecting Minds: Innovations in Neurology and Patient Care" I am a specialist Dementia Nurse caring for people living with dementia and their families so my emphasis is not so much scientific as practical. This is what is so impressive about conferences organised by the Magnus Group-there is a diversity of topics presented which balances scientific depth with broader perspectives. This makes for a dynamic and inclusive conference experience that is informative and interesting. This year, I hope to share some insights into the impact poor diagnosis rates are having, worldwide, on families and people living with dementia.

I look forward to seeing you in Barcelona.

Welcome Message

Ken Ware

Founder of Neurotricional Sciences Pty Ltd & Neuro Physics Therapy, Gold Coast, Queensland, Australia

It is an honour to once again be part of this distinguished gathering of clinicians, researchers, and thought leaders at Neurology 2026.

This event represents an important opportunity to come together across disciplines, borders, and perspectives to share not only knowledge, but a commitment to innovation and human betterment. The diversity of expertise and inquiry represented here is a powerful reminder of the richness and responsibility we hold in shaping the future of neurological science and care.

As we embark on this year's program, I encourage a spirit of openness, bold inquiry, and collaboration. It is through the synthesis of our unique contributions that new paradigms emerge, and lives are changed.

Wishing all attendees an inspiring and deeply productive conference.

Welcome Message

Professor Luiz Moutinho, BA, MA, Ph.D., MAE, FCIM

Artificial Intelligence, Biometrics and Neuroscience Theory, Futures Research, United Kingdom
University of Suffolk, England. Member of the Academia Europaea, United Kingdom

Dear Conference Attendees,

The "12th Edition of International Conference on Neurology and Neurological Disorders" will be held in Barcelona, Spain, from June 22-24, 2026. It will be a hybrid event, allowing both in-person and virtual participation. The conference theme is "Connecting Minds: Innovations in Neurology and Patient Care".

Neurology 2026 gathers a multidisciplinary audience including academic neurologists, neuroscientific researchers, clinical practitioners, pharmaceutical innovators, and policy advocates united by a shared commitment to advancing neurological care. The conference is an open forum for thought leaders and rising voices to collaborate, contribute, and challenge conventions. Exploring a wide array of scientific and clinical topics, the agenda underscores the integration of advanced diagnostics, therapeutic approaches, neurotechnological innovations, and holistic patient-centred strategies. It embraces both theoretical exploration and evidence-based application. The event also highlights emerging trends such as artificial intelligence, precision medicine, and neurorehabilitation, reflecting the dynamic evolution of the neurological sciences.

There will be key mind-stretching sessions on, for example, Molecular Neuroscience, Neuroscience Research, Artificial Intelligence in Neurology and Neurosurgery, Cognitive Neuroscience and Psychology, Neurobiology, Advances in Neuroimaging Techniques, among many other areas.

The comments and testimonials from past participants and keynote speakers of last conferences in this Series are incredible and amazing.

This Conference would be a great opportunity for past and new participants, including young and senior researchers, scientists, clinicians and academics to gain knowledge with the up-to-date research in neurology.

Welcome Message

Robert B. Slocum Ph.D

Narrative Medicine Program Coordinator, Integrative Medicine, University of Kentucky HealthCare, USA
Assistant Professor (Voluntary Faculty Series), University of Kentucky College of Medicine, Department of Internal Medicine, USA

Dear Conference Participants and Visitors,

I am honored to offer these brief notes of welcome for you concerning my presentation.

Functional Seizures (FS) [also known as Psychogenic Non-Epileptic Seizures (PNES)] are involuntary paroxysmal episodes that are frequently misdiagnosed and mistreated as epileptic seizures. Many patients with FS have a history of sexual, physical, or emotional abuse or other traumatic experiences. Cultural influences, a family code of silence, or a personal sense of shame may inhibit patient communication about their overwhelming experiences.

FS is a communication disorder in which distress is expressed somatically in a pathological way instead of an adaptive and verbal manner. A seizure-like event may provide distraction from an overwhelming situation or experience but at a terrible cost to the patient. Narrative Medicine (NM) is a communication therapy that engages and integrates the patient's life story and overwhelming experiences through interactive conversations and writing exercises. NM helps patients work through the biographical disruption of their condition that threatens their coherent sense of self. NM helps patients communicate more effectively about unspeakable distress and discover a narrative antidote for their condition. NM is a communication therapy for the communication disorder of FS.

I look forward to your participation in this conference and my presentation.

Welcome Message

Dr Sergey Suchkov, MD, PhD

N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia

Dear Colleagues, Partners, Scientists, Clinicians, and Friends,

On behalf of the Organizing Committee, we would extend a warm welcome to all of the distinguished speakers and participants to attend the 12th Edition of International Conference on Neurology and Neurological Disorders, a leading global event dedicated to advancing neurological science and practice, scheduled to be hold during June 22-24, 2026, in Barcelona, being a Tapestry of Historical Architectural Brilliance, Urban Innovation and Grand Beauty in the Globe.

This Grand Event offers an exceptional opportunity to stay at the forefront of advancements in upgraded neurology, stimulated by advances in systems biology, integrative medicine and design-driven clinical research, transforming the healthcare landscape into Personalized & Precision Neurology (PPN). Being a cornerstone of modern OMICS-guided diagnostics, targeted treatment and IT support, PPN as a model of clinical precise neuropathology of the next-step generation generates global avenues to secure the health and wellness via illustrating the phenomenal impact of Personalized and Precision Medicine (PPM)-related philosophy and armamentarium in the practice of practitioners.

Advances in biomarker-driven targeting would gift a new hope opening up gates in the field of regenerative medicine and in the treatment and management of neurodegenerative diseases. Dive deep into the world of clinical neurology, precision neuropathology, design-driven neurotechnology and neurobiointerfaces-its advancements and challenges and imagine multitargeted neuromodulators, molecular neuroimaging and neurorobotics-and move ahead with evolving trends and future research projects.

In this context, the next-generation neurologists are starting up to play a unique role in developing and implementing molecular profiling tests in practice and communicate the results and their relevance with partners and students. Those neurologists are becoming he final certifier of a complex diagnostic process and the core experts in implementing OMICS-driven tests in neurology practice.

The Conference will feature a highly interactive, stimulating and multidisciplinary Program to raise the top contributions in the upgraded neuropathology-related diagnostic training, design-driven translational research & applications, and education of the next step generation. The topics will include IT-driven diagnostics, personalized treatment strategies, and advances in neuroplasticity and brain molecular imaging, all aimed at improving outcomes for neurological disorder patients and prevention of subclinical stages at pre-illness personas-at-risk.

This Conference is not only a celebration of the past, but also a reaffirmation of our commitment to advancing neurological knowledge through open dialogue and critical thinking. Our goal is to open a forum to facilitate the exchange of knowledge and experience and to invigorate the field with young clinicians, scientists, biodesigners and biotechnologists. Making progress in the field of PPN is thus one of the most significant global challenges of our time.

Personally, I am convinced that the international partnership and collaboration would play a crucial promoting role for the jointly set projects from any points of view. We do hope that your interaction with your colleagues from many different countries will stimulate a creative exchange of ideas and will be personally rewarding.

We look forward to welcoming you to the summit to taste a smell of the deeply rooted neuroscience, practice and culture, and to enjoy the interaction with your colleagues from different countries whilst stimulating a creative exchange of ideas! Warmest and productive wishes and hope to meet and to see you soon in brilliant Barcelona!

Welcome Message

Sid O'Bryant

Dr. Joe and Peggy Schooler Endowed Chair, Executive Director, Institute for Translational Research, Professor, Department of Family Medicine, TCOM, University of North Texas Health, Fort Worth, United States

Hello and thank you for joining me in a very important discussion at Neurology 2026 on Understanding Alzheimer's Disease Biomarkers Across Diverse Populations: Opportunities and Insights for Novel Precision Medicine Approaches. I am truly excited to share cutting-edge information with you and engage with this forward-thinking community.

In the absence of representation of everyone in our science and trials, there will be precision medicine for no one! It is critical that we build and sustain systems and infrastructures for everyone in each of your communities to have access to state-of-the-art science, diagnostics and therapies. By understanding how Alzheimer's disease biomarkers function across all populations, we will build more accurate and person-centered diagnoses, trials and, eventually, personalized care.

Let's think collaboratively and inclusively as we approach the new era of Alzheimer's disease science so we can, as a scientific and medical community and family, achieve A World of Optimal Brain Health for ALL.

Thank you and I look forward to seeing you in Barcelona.

Welcome Message

Prof W S El Masri FRCS Ed, FRCP, PHF

Keele University, United Kingdom

It is a great pleasure to welcome you to this significant scientific gathering, “Connecting Minds: Innovations in Neurology and Patient Care”. Neurological injuries and disorders remain a major challenge worldwide. Ongoing research is making remarkable strides in improving diagnosis, treatment, outcomes and prevention strategies. Early detection through comprehensive neurological assessments can help identify conditions before they manifest severely, allowing for timely interventions and better outcomes.

Exploring the natural course of recovery and the role of biomarkers, genetic factors, and environmental influences in high-risk populations is helping pave the way for more personalized and effective therapies. Additionally, emerging technologies such as artificial intelligence and advanced neuroimaging are transforming how we understand and manage neurological diseases, offering new possibilities for assessing risks and tailoring care.

We stand at an exciting crossroads in neuroscience, where expanding knowledge deepens our understanding of brain function and its impact on overall health. Enhancing neurological care not only alleviates immediate symptoms but also promotes long-term cognitive health and improves quality of life across all ages.

I warmly welcome you all and look forward to a conference filled with inspiring presentations, potentially helpful collaborations, and breakthrough findings that are likely to shape the future of neurology.

Table of Contents

Table of Contents

Table of Contents

Table of Contents

Table of Contents

Table of Contents

About
Magnus Group

About

Magnus Group, a distinguished scientific event organizer, has been at the forefront of fostering knowledge exchange and collaboration since its inception in 2015. With a steadfast commitment to the ethos of Share, receive, grow, Magnus Group has successfully organized over 200 conferences spanning diverse fields, including Healthcare, Medical, Pharmaceutics, Chemistry, Nursing, Agriculture, and Plant Sciences.

The core philosophy of Magnus Group revolves around creating dynamic platforms that facilitate the exchange of cutting-edge research, insights, and innovations within the global scientific community. By bringing together experts, scholars, and professionals from various disciplines, Magnus Group cultivates an environment conducive to intellectual discourse, networking, and interdisciplinary collaboration.

Magnus Group's unwavering dedication to organizing impactful scientific events has positioned it as a key player in the global scientific community. By adhering to the motto of Share, receive, grow, Magnus Group continues to contribute significantly to the advancement of knowledge and the development of innovative solutions in various scientific domains.

About Neurology 2026

About

The 12th Edition of International Conference on Neurology and Neurological Disorders (Neurology 2026), scheduled for **June 22–24, 2026,** in **Barcelona, Spain** and **virtually,** brings together global experts, researchers, clinicians, and healthcare innovators dedicated to advancing neurological science and patient care. Under the theme "*Connecting Minds: Innovations in Neurology and Patient Care*," the conference serves as a dynamic platform for scientific exchange, interdisciplinary collaboration, and the presentation of emerging discoveries in neurology and neuroscience. Through keynote lectures, scientific sessions, case discussions, and networking opportunities, **Neurology 2026** aims to inspire innovation, foster collaboration, and contribute to improved neurological health outcomes worldwide.

About
CPD Accreditation

About

Neurology 2026 is officially accredited for Continuing Professional Development (CPD) by **The CPD Group (UK),** a globally recognized provider of continuing education certification. CPD accreditation ensures that professionals continue to enhance their knowledge, skills, and competencies through structured learning activities.

At Neurology 2026, participants can earn **1 CPD credit for every hour of attendance** in conference sessions. These credits formally recognize the time spent in professional learning and support ongoing professional development across clinical, academic, and research fields.

Earning CPD credits demonstrates a commitment to maintaining high professional standards and staying updated with the latest developments in Neurology. CPD participation can also support **career advancement, license renewal requirements, and academic or professional portfolios,** depending on the policies of individual institutions or regulatory bodies.

In addition to educational value, the conference provides opportunities to engage with experts, researchers, and peers from around the world, encouraging knowledge exchange and professional networking.

Attending **Neurology 2026** not only offers a high-quality scientific learning experience but also provides internationally recognized CPD credits that reflect participants' dedication to continuous professional growth.

12TH EDITION OF

International Conference on

NEUROLOGY AND NEUROLOGICAL DISORDERS

HYBRID EVENT

22-24 JUNE 2026

NEUROLOGY

JUNE 22-24 | 2026

KEYNOTE PRESENTATIONS

Dr. Alan C. Jackson

Department of Clinical Neurosciences (Neurology), Cumming School of Medicine, University of Calgary, Calgary, Alberta, Canada

Biography: Dr. Jackson is an Adjunct Clinical Professor of Clinical Neurosciences at University of Calgary. Previously Jackson was Professor of Medicine and Section Head of Neurology at University of Manitoba and was at Queen's University (Canada). Dr. Jackson continues working as a neurologist. Dr. Jackson graduated from medical school at Queen's. Dr. Jackson completed an internship at University of Southern California, residencies in internal medicine at Queen's and neurology at Western, and a neurovirology fellowship at Johns Hopkins University. Jackson was active in research for 30 years and has numerous peer-reviewed publications, book chapters, and seven edited books on rabies/infections of the nervous system.

Rabies: Challenges in taming the beast

Rabies has occurred in humans and animals since antiquity and remains an important public health problem with about 60,000 human deaths per year, particularly in Asia and Africa. The disease is virtually always fatal, although at least 34 survivors have been documented; unfortunately, many have severe neurological sequelae. Louis Pasteur developed a rabies vaccine and successfully immunized Joseph Meister in 1885, which was an important development for post-exposure prophylaxis of rabies. Rabies can be effectively prevented after recognized exposures with wound cleansing and administration of rabies immune globulin and rabies vaccine. Endemic dog rabies is the main threat to humans, although transmission also occurs to humans and companion animals from wildlife. Bat exposures from small bats may not be recognized and transmission from bats is the main cause of human rabies in the USA and Canada. Our understanding of rabies pathogenesis remains incomplete. Rabies virus spreads from neuron-to-neuron via axonal transport to and within the central nervous system and causes an encephalomyelitis with mild inflammatory changes and without prominent degenerative neuronal changes. Studies performed in models of experimental rabies have recently shown degenerative changes involving axons and dendrites due to oxidative stress, which is caused by mitochondrial dysfunction. One rabies virus protein, the rabies virus Phosphoprotein (P), interacts with mitochondrial Complex I, resulting in increased Complex I activity and the production of Reactive Oxygen Species (ROS), which preferentially damage neuronal processes. Mutational analysis

suggested the importance of the 157-169 region of the P and serine residues at 162 and 166 are important. Two rabies virus recombinants with serine to alanine mutations at positions 162 and 166 did not increase Complex I activity and result in ROS generation. The treatment of human rabies has not yet demonstrated any efficacious therapies beyond critical care. The Milwaukee protocol, which includes therapeutic coma as the key component, has been aggressively promoted. Numerous repeated failures over the past 20 years, a lack of published successes, and the lack of a sound scientific rationale have led this protocol to be considered ineffective therapy and its use is not recommended. For aggressive care, a critical care unit is essential. Combination therapy is reasonable and effective in other diseases. Antiviral therapy is important, but new antiviral drugs are needed because currently available drugs have not shown efficacy. Immunotherapies, including new rabies vaccines and neutralizing rabies virus antibodies, require careful consideration. Antibodies have shown some efficacy in a mouse model. There are important delivery issues for drugs and antibodies as a result of blood-brain and blood-spinal cord barriers. Neuroprotective therapies that antagonize injurious biochemical and molecular events are lacking for common acute neurological diseases. Efficacy has been shown for hypothermia for brain injury after cardiac arrest. Selective cerebral hypothermia could be considered a potential therapy for rabies because it is associated with many beneficial effects and has less systemic adverse effects than whole body cooling. The development of novel therapies for rabies in the future will likely depend on an improved understanding of rabies pathogenesis.

Prof. Dr. Habil Bernd Blobel FACMI, FACHI, FHL7, FEFMI, FIAHSI

University of Regensburg, Medical Faculty, Regensburg, Germany
Charles University Prague, First Medical Faculty, Prague, Czech Republic
Faculty European Campus Rottal-Inn, Deggendorf Institute of Technology, Deggendorf, Germany
University of Genoa, DIBRIS, Genoa, Italy

Biography: Dr. Bernd Blobel studied Mathematics, Technical Cybernetics and Electronics, Bio-Cybernetics, Physics, Medicine and Informatics at the University of Magdeburg and other universities in the former GDR. Bernd received his PhD in Physics with a neurophysiological study. Furthermore, Bernd performed the Habilitation (qualification as university professor) in Medicine and Informatics. Bernd was Head of the Institute for Biometrics and Medical Informatics at the University of Magdeburg, and thereafter Head of the Health Telematics Project Group at the Fraunhofer IIS in Erlangen. Thereafter, Bernd acted until his retirement as Head of the German National eHealth Competence Center at the University of Regensburg as well as Head of the globally unique International Interdisciplinary PhD and PostDoc College. Bernd was and is still leadingly involved in many countries health digitalization as well as electronic health record strategy. Bernd published more than 600 papers, published/edited many books and supervised a big number of PhD students from all around the world. Bernd was German Representative to many SDOs such as HL7, ISO, CEN, OMG, IEEE, ASTM, SNOMED, etc., also chairing the national mirror groups. Furthermore, Bernd still engaged in international higher education. Bernd is Fellow of several international academies.

Designing and managing intelligent and ethical transformed health and social care ecosystems

Health and social care systems around the globe currently undergo a transformation towards Personalized, Preventive, Predictive, Participative Precision Medicine (5PM), supported by technology. It considers individual health status, conditions, genetic and genomic dispositions in personal social, occupational, environmental and behavioural context, understanding the pathology of diseases and turning health and social care from reactive to proactive. The aforementioned transformation is strongly supported by technologies such as micro-and nanotechnologies, advanced computing, artificial intelligence, autonomous systems and robotics, knowledge representation and management, etc. Beside their opportunities, those advanced technologies also bear risks to be managed, requiring the detailed consideration from a humanistic, moral and ethical perspective. For enabling communication and cooperation between all actors from different disciplines involved, using different methodologies, perspectives, intentions, languages, we shall understand and formally and consistently represent the multidisciplinary, highly complex and dynamic 5PM ecosystem. The outcome is a system-theoretical, architecture-centric, ontology-

based, policy-driven approach for designing and managing intelligent and ethical 5PM ecosystems. The necessary model and framework has been developed by the author and meanwhile standardized as ISO 23903 Interoperability and Integration Reference Architecture. The formal representation of any ecosystem and its development process including examples of practical deployment of the approach are presented in detail. This includes correct systems and standards integration and interoperability solutions.

Dr. Carlos Alberto Mangone*, De Pascale Ana, Genovese O, Roxana Grillo (in memoriam)

Neurology Unit, Santojanni General, Argentina

Biography: Carlos Mangone, MD, PhD, is a Neurologist and Adjunct Prof of Neurology, School of Medicine, Bs AS University. Chair of Neurology Unit Santojanni Hospital, Bs As, Argentina.

Standardization of the cognitive reaction test: Verification of its reliability and utility in the diagnosis of mild cognitive impairment

Introduction: Early diagnosis of patients with Mild Cognitive Impairment (MCI) is essential to initiate early treatments that may slow their progression towards dementia. Because accurate biological and imaging markers are not accessible to all healthcare systems, neuropsychological clinical assessment assumes a leading role as the first approach to the target subject. We present the Cognitive Reaction Test (CRT), a computerized personal development test, which evaluates learning ability, executive control, attention, and speed of information processing, dimensions initially affected in subjects with DCM.

Objectives: To standardize and establish the power of CRT for differentiation of MCI from normal controls.

Patients and Methods: CRT was administered to 100 healthy subjects and to 100 patients with MCI with age between 50 and 80 years. They were evaluated with the combination of scales, batteries, and standardized tests (Mini-Mental State Examination, ADAS Memory Sub-test, Trail Making Test A and B, and Hughes' CDR Score).

Results: The results of the Mann-Whitney U test and the Kruskal-Wallis test supported the power of CRT in differentiating healthy controls and MCI patients. The normative values of the CRT are presented.

Conclusions: We report a new standardized test designed to differentiate MCI patients from normal subjects. The tasks that structure the CRT give reaction times and errors, and correspond to a reliable test since they can differentiate MCI from normal controls.

Edie Raether

NeuroShifts and Wings for Wishes Academy, United States

Biography: Edie Raether, MS, CSP, is a behavioral neuroscientist, psychotherapist, and global authority on rapid cognitive change and identity level transformation. With a career spanning five continents, Edie Raether has worked with Fortune 100 companies, healthcare systems, and educational institutions to translate cutting edge brain science into practical, scalable behavior change strategies. As the creator of NeuroShift™ and the three second NeuroShift™, Edie integrates decision neuroscience, dopaminergic signaling research, and micro intervention models to demonstrate how micro moments can interrupt maladaptive patterns and initiate rapid neuroplastic change. Edie Raether work draws on evidence from sub second dopamine fluctuation studies, real time decision reversal research, and autonomic regulation science to create accessible tools for emotional regulation, habit change, and cognitive reframing. A seven time author and TEDx speaker, Edie is recognized for bridging rigorous neuroscience with practical, real world application. Edie Raether programs have been implemented in corporate wellness, leadership development, addiction recovery, and youth mental health initiatives worldwide.

The three second NeuroShift™: How micro moments rewire the mind

Background: Emerging neuroscience demonstrates that the brain is capable of rapid, sub second shifts in cognition, affect, and behavioral intention. Research across decision neuroscience, dopaminergic signaling, and neuroplasticity shows that micro moment interventions can interrupt automatic neural patterns and initiate adaptive rewiring. The three second NeuroShift™ is a structured, micro intervention model designed to leverage these rapid neural dynamics to produce measurable changes in behavior, emotional regulation, and cognitive control.

Objective: To present a neuroscience based framework demonstrating how a three second cognitive emotional interruption can modulate prefrontal, limbic, and dopaminergic systems, enabling rapid pattern disruption and the initiation of new neural pathways. The goal is to integrate evidence from neurophysiology with applied behavioral techniques to support a scalable, real time intervention for health, wellness, and performance.

Methods: The three second NeuroShift™ integrates three components:

1. **Interrupt:** A brief, intentional pause that disrupts automatic limbic driven responses.
2. **Reframe:** A rapid cognitive reassessment that engages prefrontal cortical networks responsible for meaning making and inhibitory control.
3. **Decide:** A deliberate behavioral choice that leverages dopaminergic prediction error signaling to reinforce new neural pathways.

This Model Draws on:

- Studies showing that humans can reverse or modify decisions within 100-200ms, indicating ongoing competition between neural action plans.
- Evidence that dopamine fluctuations occur on sub second timescales, influencing motivation, reward prediction, and action selection in real time.
- Research demonstrating that micro interventions and micro habits can produce measurable neuroplastic changes through repeated, brief activation of prefrontal striatal circuits.
- Fast scan cyclic voltammetry findings showing moment to moment dopamine signaling during decision making and emotional evaluation.
- Neuroimaging data supporting the role of cognitive reframing in down regulating amygdala activity and enhancing prefrontal control.

Intervention: Participants are trained in a series of 3 minute NeuroShift™-brief, repeatable cognitive somatic techniques incorporating:

- Pattern interruption
- Breath induced autonomic regulation
- NLP based anchoring
- Sub modality shifts
- Identity level visualization in an alpha relaxed brain state
- Dopamine aligned micro reward strategies

These techniques are designed to be used in real time during stress, cravings, emotional reactivity, or habitual behavioral loops.

Results (Pilot Data & Observational Outcomes):

Preliminary implementation in corporate and clinical settings suggests improvements in:

- Emotional regulation
- Stress recovery
- Cognitive flexibility
- Reduction in maladaptive habits
- Increased adherence to health promoting behaviors
- Enhanced workplace performance and decision making

Participants report rapid subjective shifts in state, increased sense of agency, and improved ability to interrupt automatic negative patterns.

Conclusion: The three second NeuroShift™ provides a neuroscience aligned, micro intervention framework capable of leveraging the brain's natural capacity for rapid state change and real time neuroplasticity. By combining cognitive reframing, autonomic regulation, and dopaminergic reinforcement within a three second window, this model offers a scalable, evidence informed approach to improving health, behavior, and performance. The findings support the growing recognition that micro moments-not long interventions-are the leverage point for meaningful neural and behavioral change.

Keywords: Micro Interventions, Neuroplasticity, Dopamine, Decision Neuroscience, Cognitive Reframing, Autonomic Regulation, Micro Habits, Identity Based Change, NeuroShift™, Rapid Neural Modulation.

Jaqueline Tuppen RN, BSc

Hons & Specialist Practitioner, Mental Health and Admiral Nurse Community and Dementia UK, United Kingdom

Biography: Mrs Jacqueline Tuppen graduated in 1997 with a BSc Hons and Specialist Practitioner from the Universtiy of Greenwich, London. Jacqueline Tuppen worked as a CPN and then as the Acting Community Services Manager before becoming an Admiral Nurse in 2008. Retiring from the NHS in 2011, as an independent specialist nurse practitioner Jacqueline Tuppen started-COGS Club for people in the early stages of Dementia. Jacqueline Tuppen continues to work for Dementia UK as a sessional Admiral Nurse on their Dementia Helpline. Jacqueline Tuppen has published articles, and presented at a variety of events in the UK, Eire and Italy and France.

When dementia strikes early: The implications for family and services

Addressing the social determinants of health, including poverty, education, and access to healthcare, is critical in reducing the impact of public health crises and ensuring better health outcomes for all.

This presentation will share the worldwide impact of people and families living with Young Onset Dementia (YOD). It will include statistics highlighting the difference between diagnosis rates for older people compared to diagnosis rates for younger people. This presentation will consider the statistical & epidemiological, socio-economic, personal & psychological impact and the risk factors & future outlook.

The presentation will then consider the impact of young onset dementia on CARERS worldwide, looking at who the carers are and how YOD affects different people at different stages of their lives, when YOD becomes a feature of the family life. This will include the impact on people living alone with YOD worldwide.

The impact will be similar but due to the different ages of the carer, the impact highlights different needs and risk: Emotional & psychological, social & developmental, educational, financial & practical and coping & support needs.

Finally, this presentation will consider what kinds of support are available, from international to local support organisations.

Jonathan Eskenazi MD

Cedars Sinai / UCLA, USA

Biography: Dr. Jonathan Eskenazi is a well-rounded, Double Board-Certified Neurologist with in-depth training in Traumatic Brain Injury, Vascular, and Neurocognitive disorders. He has received training in clinical neurophysiology, neuro-stimulation for Parkinson's disease, and epilepsy. Dr. Eskenazi holds the position of Clinical Assistant Professor at UCLA School of Medicine and serves as a Clinical Neurology attending at Cedars Sinai. He played a key role in establishing Acute Neurology programs at Hollywood Presbyterian Medical Center and California Medical Center in Downtown Los Angeles. With many years of training followed by several years of clinical practice experience, Dr. Eskenazi covers all aspects of neurology, including trauma, stroke, epilepsy, multiple sclerosis, brain tumors, sleep disorders, and botulinum toxin (Botox) injections for migraine, dystonia, and spasticity in hemifacial spasm patients. Additionally, he specializes in Neurostimulation for Parkinson's disease, electroencephalogram studies, and Vestibular Disorders.

Traumatic brain injury updates

Definition and Types: TBI is an injury to the brain caused by external force, resulting in a range of effects. It can be classified as mild (concussions), moderate, or severe based on symptoms and impact.

Causes: Common causes include falls, vehicle accidents, sports injuries, and violence.

Symptoms: Symptoms can vary widely and may include headaches, confusion, memory issues, mood changes, and loss of consciousness.

Diagnosis: Diagnosis often involves physical exams, imaging tests like CT or MRI scans, and neuropsychological assessments to evaluate cognitive function.

Treatment: Treatment approaches may involve rest, rehabilitation therapies (physical, occupational, speech), medication for symptoms, and in severe cases, surgery.

Long-term Effects: Some individuals may experience lasting cognitive, physical, and emotional challenges, requiring ongoing support and management.

Prevention: Strategies for preventing TBI include wearing helmets, using seatbelts, and fall-proofing homes, especially for vulnerable populations like the elderly.

The lecture will emphasize the importance of early intervention and a multidisciplinary approach to care.

Traumatic brain injury (TBI) remains a leading global cause of disability, and new data highlight a shift toward precision neurotrauma. In 2024–2025, rapid blood biomarker tests (GFAP, UCH-L1) gained FDA clearance in whole-blood and automated formats, enabling point-of-care triage and safe CT reduction for mild TBI. In the ICU, hypertonic saline is increasingly favored over mannitol for intracranial pressure crises, while the NIH BOOST-3 trial is testing whether combining ICP with brain oxygen monitoring improves outcomes. Single-cell brain atlases and large consortia studies (CENTER-TBI, TRACK-TBI) are mapping the biological and psychosocial heterogeneity of injury, laying the groundwork for personalized treatment. Together, these updates mark a turning point: from generic protocols to tailored, biomarker-driven and multimodal strategies that aim to improve survival and long-term recovery.

For mild traumatic brain injury (mTBI), 2024–2025 updates emphasize earlier, more precise diagnosis and tailored follow-up. Blood biomarkers GFAP and UCH-L1 have now been validated and FDA-cleared in whole-blood, point-of-care formats and automated lab platforms, allowing emergency teams to safely rule out CT-detectable injury within minutes and reduce unnecessary imaging. Studies show GFAP rises within 30 minutes of injury, making ultra-early triage feasible. Large cohort data (CENTER-TBI, TRACK-TBI) highlight how outcomes vary by biological and psychosocial factors, including genetic vulnerability to persistent post-concussion symptoms and mental health challenges, supporting a move toward risk-stratified follow-up. The field is moving from a “rest and discharge” model toward precision mTBI care, where biomarker-guided triage, individualized monitoring, and targeted rehab interventions aim to prevent chronic symptoms and improve recovery trajectories.

Ken Ware

Founder of Neurotricional Sciences Pty Ltd & NeuroPhysics Therapy, Gold Coast, Queensland, Australia

Biography: Ken Ware was founder of Neurotricional Sciences Pty Ltd and NeuroPhysics Therapy and Research and Ken had been in private practice for almost 30 years, while doing independent and collaborative research. Ken Ware also presented unique research at 10 major International Science Conferences including neuroscience, Physics, Psychology and Life Sciences, which covers a very broad scientific audience. Ken is Former Mr. Universe 1994, National powerlifting and Bodybuilding champion and record holder. Ken had published relative publications in 'Frontiers in Clinical Physiology'-'World Journal of Neuroscience'-'World Journal of Cardiovascular diseases'. Ken 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.

Perception and individuality in patient cases identifying the ongoing evolution of Myalgic Encephalomyelitis/ Chronic Fatigue Syndrome (ME/CFS)

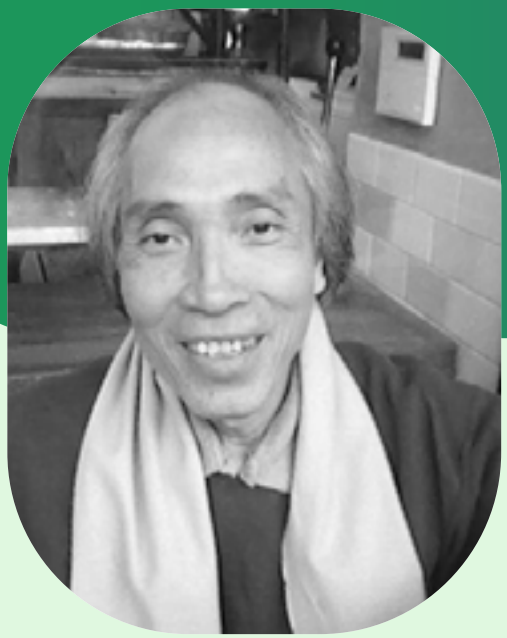
The way individuals perceive and interpret their internal and external environments directly influences how they function and adapt within them. In a stable and well-regulated human system, perception tends to align closely with reality, allowing for accurate motor responses and functional regulation. However, when subjective sensory perception becomes distorted-through trauma, illness, or prolonged stress-errors can emerge within the sensory-motor feedback loop. These errors accumulate over time and contribute to the emergence and persistence of psychophysical dysfunction.

ME/CFS (Myalgic Encephalomyelitis/Chronic Fatigue Syndrome) presents a highly relevant and increasingly complex example of this phenomenon. It is a multisystem neuroimmune condition affecting millions worldwide, with hallmark symptoms such as profound fatigue, post-exertional malaise, cognitive dysfunction, and increased sensory sensitivity. While diagnoses may be shared across patients, the lived experiences and system-level responses remain uniquely individual.

This presentation explores how patient individuality-shaped by divergent perceptual errors and adaptive histories-must become central to any meaningful intervention. Drawing on case studies from Neuro Physics Therapy, this presentation demonstrates how real-time correction of sensory perception errors within controlled yet dynamic environments can

lead to profound neurological recovery. By moving beyond disease-label-driven protocols and into the domain of psychophysical individuality, NPT provides a powerful model for uncovering and reversing the underlying drivers of ME/CFS and related neuroimmune disorders.

Keywords: ME/CFS, Perception, Individuality NeuroPhysics Therapy, Sensory Perception Errors, Neurological Diseases and Disorders, Psychophysical.



Khue Vu Nguyen

School of Medical Imaging, Jiangsu Medical College, Yancheng, 224005, China

Medical Imaging Institute of Jiangsu Medical College, Yancheng, 224005, China

Center for Molecular Biophysics, CNRS Orleans, 45071 Orleans, France

Former Institution Attended: School of Medicine, Departments of Medicine and Pediatrics, Biochemical Genetics and Metabolism, The Mitochondrial and Metabolic Disease Center, University of California, San Diego, California, 92103 USA

Biography: Dr. Khue Vu Nguyen (born at Hà Nội, Việt Nam) is currently collaborating for research work with the Center for Molecular Biophysics from Centre National de la Recherche Scientifique, CNRS, at Orleans, 45071 Orleans, France. Nguyen was Visiting Professor in the School of Medical Imaging, Jiangsu Medical College, Yancheng, 224005, China-Medical Imaging Institute of Jiangsu Medical College, Yancheng, 224005, China. Nguyen was also Full Project Scientist in the Departments of Medicine and Pediatrics, University of California, San Diego, School of Medicine, San Diego, California 92103, U.S.A. Nguyen has worked as Scientist in different French and U.S. Research Institutions and Companies. Nguyen has studied at University Louis Pasteur, Strasbourg, France: B.S. in Biochemistry; M.S. in Molecular Biology; Ph.D. in Macromolecular Physical Chemistry; Ph.D. in Physical Sciences (Doctor of Science, D.Sc., in Physics: Doctorat d'Etat ès Sciences Physiques). Nguyen is author and co-author of numerous publications and holder of many patents. Nguyen was a recipient of the foreign expert award for program No. G2021014081L under the Ministry of Science and Technology of the Peoples's Republic of China, 2022, and listed in Who's Who in the World; Who's Who in America; Who's Who in Science and Engineering; etc. from Marquis Who's Who, New Providence, N.J., U.S.A., and named a 2024 Top Scholar by ScholarGPS® (within the top 0.5% of all scholars worldwide). Nguyen is member of different academic organizations such as American Society for Microbiology; American Chemical Society. Dr. Khue Vu Nguyen is Editor-in-Chief of different scientific journals such as Current Pediatric Research; Enliven: Journal of Genetics, Molecular and Cellular Biology; Journal of Brain Research; etc. Nguyen is Editorial Board Member of different scientific journals such as Metabolic Brain Disease; Journal of Pediatric Genetics; Expert Opinion on Orphan Drugs; etc. Nguyen is also Invited Reviewer of different scientific journals such as Molecular Genetics and Metabolism; AGE: Journal of the American Aging Association; Journal of Neural Transmission; etc. Nguyen is Scientist Reviewer for grant proposals of the Metabolic Disease Two (MB-2) of the Peer Review Medical Research Program (PRMRP) for the U.S. Department of Defense Congressionally Directed Medical Research Programs (CDMRP). Current research includes genetic diseases, neurodevelopmental and neurodegenerative disorders, cardiovascular diseases, cancer, and environmental science.

Epigenetic modulation of human neurological disorders: Lesch-Nyhan disease as a model disorder

Epigenetics is the study of how cells control gene activity without changing the DNA sequence. Epigenetic changes affect how genes are turned on and off, or expressed, and thus help regulate how cells in different parts of the body use the same genetic code. Errors in epigenetic process can not only lead to abnormal gene activity or inactivity but also influence Alternative Splicing (AS) and could cause human diseases. Otherwise, there is also epistasis (gene-gene interactions) in genetics in which the effect of a gene mutation is dependent on the presence or absence of mutations in one or more other genes, respectively termed modifier genes. So, the effects of a given gene on a biological trait are masked or enhanced by one or more other genes. This abstract concerns the Lesch-Nyhan Disease, LND, a rare X-linked recessive neurogenetic disorder, MIM: 300322). Despite having been characterized since more than 60 years ago (from the first report of Lesch M, Nyhan WL in 1964), however, up to now, there is no satisfactory explanation of how the loss of the Hypoxanthine-Guanine Phosphoribosyltransferase (HGprt) enzyme function affects the brain to cause the intellectual impairment, and self-mutilating behaviors in LND. This has made difficult for the development of an effective treatment for LND. Some findings reported that expression of the β-Amyloid Precursor Protein (APP) gene from human skin fibroblasts from normal subject (control) as well as LND patients was (a) Under epigenetic regulation of alternative APP pre-mRNA splicing; and (b) Via epistasis between the Hypoxanthine Phosphoribosyltransferase 1 (HPRT1, encoded for HGprt) and APP genes affecting alternative APP pre-mRNA splicing leading to the production of alternative APP fragments that might be responsible for the differing severity in LND patients. Understanding of how epigenetic defects and epistasis affecting human health, especially for neurological disorders, could suggest targets for therapeutic interventions. For such a purpose, LND has been selected as a valuable model for studying genetic-epigenetic interplay as well as for exploring epistasis between HPRT1 and APP genes. And so, the construction of expression vectors for HGprt enzyme and APP via the glycosyl-phosphatidylinositol, GPI, anchor is performed (Figure 1). Information obtained from such expression vectors would be useful for future directions to design therapies. The present abstract is aimed at enhancing, for the first time, the importance of an epigenetic modulation via epistasis between APP and HGprt in LND based on findings found from divers publications suggesting that the pathogenesis of this monogenic LND results from combinatorial and multigenic defects and could be considered as a model disorder for the research on other genetic diseases, especially human neurological disorders.

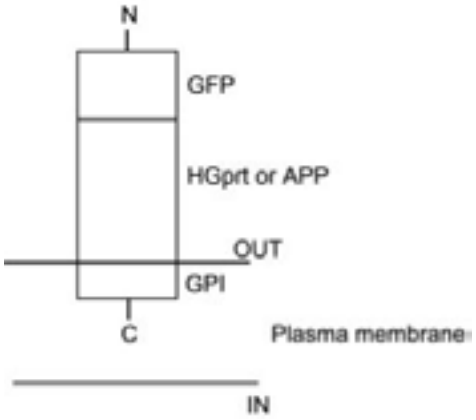


Figure 1: Schematic representation of the membrane topology of the expression vectors for HGprt and APP.

The mammalian expression vector pcDNATM 3.1 (+) is used as backbone in which all genes of interest are inserted in the right frame into the pcDNATM 3.1 (+) vector. The construct comprising the sequence encoding the C-terminal of the glycosyl-phosphatidylinositol, GPI, anchor derived from the human Folate Receptor 1 (FOLR1) protein; the entire Coding Sequence (CDS) of Hypoxanthine Phospho Ribosyl Transferase 1 (HPRT1) gene encoding HGprt enzyme or the CDS of APP gene encoding APP coupled with the CDS of the Green Fluorescent Protein (GFP) gene.

Luiz Moutinho

University of Suffolk, United Kingdom

Biography: Luiz Moutinho was elected as the member of The Academia Europaea in 2020. In 2017 Moutinho received a degree of Prof. Honoris Causa from the Univ. of Tourism and Management Skopje, North Macedonia. In 2024 was rated among the 100 best scientists in Business and Management by Research. com. During 2015 - 2017 Moutinho was professor of Bio Marketing and Futures Research at the DCU Business School, Dublin City University, Ireland. This was the first Chair in the world on both domains-Bio Marketing and Futures Research. Previously, and for 20 years, Moutinho had been appointed as the Foundation Chair of Marketing at the Adam Smith Business School, University of Glasgow, Scotland. Moutinho completed his PhD at the University of Sheffield in 1982. Moutinho has been a Full Professor for 35 years and held posts at Cardiff Business School, University of Wales College of Cardiff, Cleveland State University, Ohio, USA, Northern Arizona University, USA and California State University, USA. Moutinho has held Visiting Professorship positions at numerous universities in China, Lithuania, Austria, New Zealand, Denmark, Slovenia, Portugal, Hungary, Taiwan, Brazil, Colombia, Fiji and Cyprus. Between 1987 and 1989 Moutinho was the director of the Doctoral Programmes at the Confederation of Scottish Business Schools and at the Cardiff Business School between 1993 and 1996. Moutinho was director of the Doctoral Programme in Management at the University of Glasgow between 1996 and 2004. Professor Moutinho is the Founding Editor-in-Chief of the Journal of Modelling in Management (JM2) and Co-editor-in-Chief of the Innovative Marketing Journal. Moutinho has another 4 associate editorships as well as being in the editorial boards of another 47 international academic journals. Moutinho areas of research interest encompass marketing and management futurecast, artificial intelligence, biometrics and neuroscience in marketing, futures research algorithmic self, EmoWear - a wearable tech device that detects human emotions, evolutionary algorithms, human-computer interaction, the use of artificial neural networks in marketing, modelling processes of consumer behaviour and tourism futurecast. Moutinho has developed a number of conceptual models over the years in areas such as tourism destination decision processes, automated banking, supermarket patronage, among other areas. The testing of these research models has been based on the application of many different statistical, computer and mathematical modelling techniques ranging from multidimensional scaling, multinomial logit Generalised Linear Models (GLMs) and linear structural relations to neural networks, ordered probit, simulated annealing, tabu search, genetic algorithms, memetic algorithms and fuzzy logic. Prof. Moutinho has given keynote speeches, lectures, seminars, talks, etc. in 49 countries worldwide. Prof. Moutinho has 39 books published, over 161 articles published in refereed academic journals. Moutinho has 171419 academic citations, the h-index of 59 and the i10-index of 166 (Google Scholar, April 27th, 2024).

Neuro sensorium

The presentation starts with coverage of advanced neuroimaging and neuro technologies. Next, there will be an introduction to behavioural neuroscience, gut-brain-axis, microbiome, computational neuroscience and neurology. The discussion will the move on to dissecting concepts like, connectomics and network neuroscience, as well as brain-inspired computing and digital twins of the brain. Finally, the talk ends with the analysis of important issues like, cognitive neuroscience, open science, neuroinformatics, social neuroscience and collective intelligence.

Robert B. Slocum Ph.D

Narrative Medicine Program Coordinator, University of Kentucky HealthCare, Lexington, Kentucky, USA

Biography: Robert B. Slocum is the Narrative Medicine Program Coordinator at University of Kentucky HealthCare. Robert B. Slocum holds doctorates in law (Vanderbilt), ministry (University of the South), and theology (Marquette). Robert B. Slocum has experience in pastoral ministry as well as academic teaching and administration. Robert B. Slocum has taught undergraduate courses in religious studies and ethics. Robert B. Slocum is an Assistant Professor (voluntary faculty, Internal Medicine) at the University of Kentucky College of Medicine (COM). Robert B. Slocum teaches a fourth-year COM elective on the narrative basis for patient care and resilient practice. Robert B. Slocum is a member of the Hospital Ethics Committee. Robert B. Slocum is the author, editor, or co-editor of 14 books, including a journal of reflections. Robert B. Slocum 36 articles have appeared in theological or medical journals and as book chapters, and Robert B. Slocum has made presentations at more than two dozen theological and medical conferences. Robert B. Slocum has also published short fiction and poetry. Robert B. Slocum is interested in the clinical application of narrative and the significance of narrative for identity formation. Robert B. Slocum sees Narrative Medicine as a bridge between medical humanities and clinical practice.

Narrative medicine: A communication therapy for the communication disorder of Functional Seizures (FS) [also known as Psychogenic Non-Epileptic Seizures (PNES)]

Patients with Functional Seizures (FS) (also known as Psychogenic Non-Epileptic Seizures (PNES), have involuntary paroxysmal episodes that resemble epileptic seizures but without organic etiology. Many patients with FS have a history of sexual, physical, or emotional abuse, or other traumatic or overwhelming experiences. FS is a communication disorder in which distress is expressed somatically in a pathological way instead of a healthy verbal manner. The patient's body may seem to enact a communication of its own as the patient cannot or will not communicate directly about an overwhelming and unspeakable subject.

Patients with FS are frequently misdiagnosed and mistreated for epileptic seizures. Accurate diagnosis may be delayed for many years. FS may cause severe disruption of the patient's quality of life in terms of employment or schooling as well as relationships, and activities of daily living. Some patients with FS have been accused of faking symptoms or malingering, and stigmatized by health care providers, coworkers, family members, and others in society. Patients with FS may have family histories of poor interpersonal communication and conflict resolution, with inherited codes of silence and shame

concerning sensitive or traumatic subjects. Patients with FS may have Post-Traumatic Stress Disorder (PTSD) as a comorbidity. They may have significant dissociation and "emotional blindness" with inadequate emotional awareness and expression.

Narrative Medicine (NM) is a communication therapy that draws out the patient's narrative of illness or injury and overwhelming experiences through interactive conversations and writing exercises. NM provides space for the patient to explore thoughts and feelings in a guided conversation with a collaborator who listens attentively. NM is patient-centered and open-ended with focus on exploring topics the patient needs to discuss. NM seeks to help patients identify meaning and identity in the context of their lives and challenges.

Unlike Cognitive Behavioral Therapy (CBT), there is no script or checklist for NM sessions. NM sessions are not confrontational. The "teller" and "listener" share a "dyadic" professional relationship that encourages trust and respect. This interactive process is dynamic and may take unexpected turns. Both teller and listener can be changed by an NM session. Patients can reflect on their difficult stories relative to their sense of identity, sources of strength, new insights, and hope for the future. Unstated or previously silenced concerns may be voiced by the patient. An unhurried context of trust where the patient is heard can encourage the patient to communicate about disturbing history and situations. NM helps patients work through the biographical disruption of their condition that may threaten their sense of an integrated and coherent self. Narrative writing exercises have also proven helpful for patients facing a variety of traumas and major stresses.

A patient with FS who constructs a story (written or oral) about personal trauma or overwhelming stress can discover a narrative antidote to the communication disorder and inhibition of FS. The patient with FS can become the teller of the story who discovers hope by putting the unspeakable into words. Old taboos and codes of silence can be let go as the patient collaborates with an attentive NM provider. Finding words for difficult experiences and sharing the story can help patients process their thoughts and feelings to reintegrate traumas and other experiences relative to their sense of meaning, self-identity, beliefs, and goals. Difficult personal history cannot be changed, but the patient may come to see their challenges in a new light. Patients can begin to reclaim their lives from the communication disorder of FS and other functional disorders.

Roger H. Coletti, MD, FACC, FASNC, FSCAI

Interventional Health, PA, Lewes, DE, USA

Biography: Dr. Coletti did a fellowship in interventional cardiology in New York and had a career in interventional cardiology in New Jersy and Delaware, USA. Coletti was board certified in internal medicine, cardiovascular disease, interventional cardiology, and nuclear cardiology. Coletti had an interest in chronic muscle spasm and found that chronic muscle spasm had an ischemic etiology and developed a technique using EMG guidance to reverse the ischemia and resolve the chronic muscle spasm. Coletti publication in this area is 12 abstracts, a book and 2 recent articles. Coletti is currently retired from clinical practice and no longer has institutional affiliations.

Treatment of chronic muscle spasm and pain with the CMECD® procedure

It has been noted by multiple researchers that there is Spontaneous Electrical Activity (SEA) at painful trigger points. This author has studied chronic muscle spasm and found that SEA is always present and appears to be the cause for the chronic nature of muscle spasm and resulting chronic pain. Chronic muscle spasm and resulting chronic pain can last for years and cases where the spasm lasted for decades were not only found but successfully treated with the CMECD® procedure. This procedure consists of EMG guidance searching for the SEA and using a combination of phenoxybenzamine, lidocaine and dexamethasone to extinguish the SEA. Large areas of muscle often need to be treated. Thanks to lidocaine acting as an antiarrhythmic, the SEA is extinguished within seconds and the phenoxybenzamine then takes over after about one hour. With the resolution of the SEA, the muscle can immediately relax. The phenoxybenzamine forms a covalent bond on the alpha motoneuron receptor and the result is a duration of action of 2-3 months. This is enough time for the muscle to recover the prolonged effect of ischemia resulting from the prolonged spasm. Muscles treated in this fashion need only a single injection. Recurrences are rare and only occur if there is a repeat overuse or traumatic injury. The CMECD® procedure is available for use by any medical caregiver that is licensed to give injections. The ability to permanently relieve chronic pain without the use of opioid drugs should prompt interest in this procedure.

Salvador Ventura

Institut de Biotecnologia i Biomedicina and Departament de Bioquímica i Biologia Molecular, Universitat Autònoma de Barcelona, Bellaterra, Barcelona, 08193, Spain

Biography: Salvador Ventura is Professor of Biochemistry and Molecular Biology at the Autonomous University of Barcelona (UAB) and co-founder of Eureka Nanobioengineering. Salvador has received numerous prestigious awards, including the Bruker 'Manuel Rico' Prize, four ICREA Academia awards, the Narcís Monturiol Medal, and the Serra Hunter Knowledge Transference Award. Salvador is a member of the Academia Europaea. As a Group Leader at the Institute of Biotechnology and Biomedicine (IBB) of UAB, where Salvador previously served as Director, Salvador has authored over 320 scientific publications and holds 19 patents. Salvador research focuses on the relationship between protein misfolding and neurodegenerative diseases, with the aim of developing novel therapeutic molecules to fight these debilitating disorders.

A structure-based strategy to target pathogenic α-synuclein in Parkinson's disease

A-Synuclein (aSyn) aggregation is a key factor in neurodegeneration in Parkinson's Disease (PD). We have used the different structural properties of toxic oligomers and amyloid fibrils to identify a family of peptides that bind to these a-synuclein species with low nanomolar affinity without interfering with the monomeric, functional protein. This activity results in high anti-aggregation potency and the ability to prevent oligomer-induced neuronal damage. With a structure-function relationship established, we identified human candidates expressed in the brain with similar binding, anti-aggregation, and detoxifying properties. Administration of the leading candidate in a PD animal model preserved the nigrostriatal pathway and completely prevented motor dysfunction.

Using a combination of structural methods, including cryo-EM, ssNMR, SAXS, XL-MS, and HXD-MS, we have delineated the interaction region between these peptides and aSyn in the context of oligomers. Deletion or mutation of this region abolishes both aSyn aggregation and neurotoxicity, validating it as a disease-relevant structural hotspot. This provided the foundation for generating conformation-specific nanobodies and human IgGs that selectively recognize toxic oligomers and suppress aSyn amyloid formation. Collectively, our findings define a new structure-based therapeutic paradigm for PD, providing both disease-modifying agents and conformation-specific tools with strong potential for diagnosis and intervention in synucleinopathies.

Sergey Suchkov[1-14*], Eric J. Sorenson[15], Shawn Murphy[16,17], Alan Wu[18,19]

[1]N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia
[2]China Hong Kong Innovation International Business Association, Hong Kong
[3]InMedStar, Russia
[4]New York Academy of Sciences, USA
[5]EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU, Belgium
[6]ISPM (International Society for Personalized Medicine), Tokyo, Japan
[7]PMC (Personalized Medicine Coalition), Washington, DC, USA
[8]AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland
[9]ACS (American Chemical Society), Washington, DC, USA
[10]AHA (American Heart Association), Dallas, TX, USA
[11]ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA
[12]ISER (International Society for Eye Research), Anchorage, AK, USA
[13]Secretary General, United Cultural Convention (UCC), Cambridge, UK
[14]The Russian Academy of Natural Sciences, Moscow, Russia
[15]Dept for Neurology, Mayo Clinic, Rochester, MN, USA
[16]Center for Bioinformatics of the Harvard Medical School, Boston, MA, USA
[17]MGH, Boston, MA, USA
[18]Clinical Chemistry Lab, SFGH, San Francisco, CA, USA
[19]Clinical Pharmacogenomics Lab, UCSF, San Francisco, CA, USA

Biography: Sergey Suchkov was born in the City of Astrakhan, Russia, in a family of dynasty medical doctors. In 1980, graduated from Astrakhan State Medical University and was awarded with MD. In 1985, Suchkov maintained his PhD as a PhD student of the I.M. Sechenov Moscow Medical Academy and Institute of Medical Enzymology. In 2001, Suchkov maintained his Doctor Degree at the National Institute of Immunology, Russia. From 1989 through 1995, Dr Suchkov was being a Head of the Lab of Clinical Immunology, Helmholtz Eye Research Institute in Moscow. From 1995 through 2004-a Chair of the Dept for Clinical Immunology, Moscow Clinical Research Institute (MONIKI). In 1993-1996, Dr Suchkov was a Secretary-in-Chief of the Editorial Board, Biomedical Science, an international journal published jointly by the USSR Academy of Sciences and the Royal Society of Chemistry, UK.

Personalized and Precision Medicine (PPM), as a unique healthcare model through biodesign-driven biotech and biopharma, translational applications, and neurology-related biomarketing to secure human healthcare and biosafety

Over the course of history, healthcare and thus healthcare philosophy have been focused predominantly on efforts to probe the already diseased individual by focusing down on a type of disorder (nosology) rather than on health or so-called pre-illness conditions. Much less effort has been placed on keeping individuals from developing disorders in the first place. PPM is expected to transform this situation giving healthcare professionals of tomorrow much more reliable control over morbidity, mortality and disabling rates, and significantly optimize the cost and efficacy of treatment for those who have fallen ill and already diseased, or are still persons-at-risk. PPM is a name for the grand new paradigm in healthcare management being based first on prevention, pre-clinical detection of the illness, and delivery of drugs to target tissues with exceptional levels of precision.

Policy formation in the field of individual health promotion and protection is one of the priority tasks of national healthcare systems. Canonical health care is becoming increasingly unaffordable in most of the countries, yet it remains ineffective in preventing or effectively treating chronic diseases. The medicine of the XXI century is Personalized & Precision Medicine (PPM), by protecting and preserving human health throughout the life. To achieve the goals of value-based healthcare and the implementation of the PPM concept, it is necessary to combine the assets of the newest advances in basic science, OMICS technologies and IT resources with clinical medicine, followed by the introduction and promotion of new generation's translational applications.

The goal of PPM is to deliver optimally targeted and timed interventions tailored to an individual's molecular drivers of disease. In this context, neurological diseases are promisingly suited models for PPM because of the rapidly expanding genetic knowledge base, phenotypic classification, the development of biomarkers and the potential modifying treatments. Neurological diseases have high degrees of genetic and pathophysiological heterogeneity, irrespective of clinical manifestations. Traditional medical paradigms have focused on late-stage syndromic aspects of these diseases, with little consideration of the underlying biology. Advances in disease modelling and methodological design have paved the way for the development of personalized neurology. PPM-guided neurology is the application of principles of PPM, ie, the prescription of specific therapeutics best suited for an individual taking into consideration both genetic and environmental factors that influence response to therapy. The aim is to improve the efficacy and reduce the adverse effects of various therapies. Biomarkers, biomarker-driven targeting and integration of diagnostics with therapeutics are important for the selection and monitoring of treatments of neurologic disorders, covering: Molecular profiling, clinical evaluation, personalized diagnosis, targeted treatment selection, monitoring and adjustment.

The future of PPM-guided neurology lies in multimodal digital data, enabling the principles of PPM to be applied in neurological disease diagnostics, treatment, and monitoring at scale, expanding the benefits to everyone. This approach offers a highly accessible, cost-efficient, and non-invasive approach for diagnosing neurological diseases at their clinical and subclinical stages, placing an individual precisely along a disease continuum, and providing the most effective possible canonical and preventive treatment pathways.

For instance, Multiple Sclerosis (MS), Parkinson's disease and Amyotrophic Lateral Sclerosis (ALS), being chronic, autoimmune, demyelinating disease of the central nervous system, are now main targets for implementation of PPM-related resources and search for specific biomarkers of the disease subtypes. PPM in those disorders include the development of targeted therapies that aim to modulate specific immune pathways involved in the Pathogenesis.

PPM-guided neurology stands at the threshold of a revolutionary transformation with the advent of PPM. The intricate tapestry of neurological disorders, long characterized by heterogeneity and complexity, is now being unraveled at the molecular level. By delving into the genetic underpinnings of neurological conditions, we uncover the potential for tailored interventions that promise not only to improve treatment outcomes but also to reshape our understanding of neurological diseases. And a journey from genomics and related OMICS-driven technologies to personalized therapies is not only transforming clinical neurology-related practice but also offering hope to individuals and families affected by neurological disorders. It heralds a new era of neurology where treatments are tailored to the individual, leading to improved outcomes, reduced side effects, and a deeper understanding of disease mechanisms.

By understanding the unique characteristics of a patient's neurological condition, such as genetic predispositions, biomarkers, and disease mechanisms, PPM aims to optimize treatment outcomes and improve patient care. Overall, PPM in neurology holds the promise of advancing our understanding of neurological diseases and transforming healthcare by tailoring interventions to the unique needs of each patient. So, to fully harvest the unique potential of PPM-guided neurology, new generations of new precision diagnostic, predictive, prognostic, preventive, prophylactic, therapeutic, rehabilitative and digital products will need to be matched with new thinking and new practice on the part of all the participants in the clinical neurology-related practice.

Dr. Sid O'Bryant

Dr. Joe and Peggy Schooler Endowed Chair, Executive Director, Institute for Translational Research, Professor, Department of Family Medicine, Texas College of Osteopathic Medicine, University of North Texas Health Science Center, Fort Worth, TX, USA

Biography: Dr. Sid O'Bryant is the principal investigator of the Health & Aging Brain Study-Health Disparities (HABS-HD), which is the most comprehensive study of Alzheimer's disease among the three largest racial/ethnic groups in the U.S. ever conducted-African Americans, Hispanics, non-Hispanic whites. The goal of the HABS-HD program is to understand the life course factors, including biological, sociocultural, environmental, and behavioral, that impact risk for Alzheimer's disease in late life. This work will ultimately lead to population-specific precision medicine approaches to treating and preventing Alzheimer's disease (i.e., "treating your Alzheimer's disease"). In addition to being a global leader in health disparities in cognitive aging, Dr. O'Bryant is a global expert in the use of blood-based biomarkers for the generation of a precision medicine approach to novel diagnostic and therapeutic strategies for Alzheimer's disease, Parkinson's disease, Dementia with Lewy Bodies and Alzheimer's disease among adults with Down Syndrome.

Understanding Alzheimer's disease biomarkers across divers populations-opportunities and insights for novel prevision medicine approaches

The recent explosion of biomarker science in Alzheimer's disease and brain aging has led to novel therapies finally reaching patients. However, the utility of these advancements to diverse populations remains largely unknown due to lack of inclusion of these populations in the science that drove these advancements. Dr. O'Bryant is a global expert in Alzheimer's disease and brain aging among diverse communities, a renowned leader in AD biomarker science, and the principal investigator of the Health & Aging Brain Study–Health Disparities (HABS-HD). HABS-HD is the most comprehensive study of advanced brain biomarkers across diverse populations ever conducted all within a sociocultural context. In this talk, Dr. O'Bryant will present recent HABS-HD findings examining imaging (MRI and PET), plasma and genetic biomarker across diverse populations and discuss how these population differences offer novel opportunities for precision medicine approaches for treating and preventing Alzheimer's disease. Lastly, he will provide actionable items for the audience to Own Your Brain Health.

Professor W S El Masri (Y) MB, BCH, FRCS, FRCP, PHF

Clinical Professor of Spinal Injuries, Keele University, Emeritus Consultant Surgeon in Spinal Injuries, RJ & AH Orthopaedic Hospital-Oswestry Shropshire, UK

Biography: Prof W S El Masri will discuss the change of both the methods of management and model of service delivery to patients with tSCI following the development of CT and MRI. El Masri will also discuss Rationale, Outcomes and Evidence of benefit or loss of the various models of service delivery and each of the various methods of management of the injured spine with damage of the spinal cord and cauda equina. WEM specifically trained in the speciality of traumatic spinal injuries (tSCI) and its allied specialities at Stoke Mandeville, Oxford, Guys Hospitals in the UK & the USA between 1971 and 1983. El Masri training was accredited by the Joint Committee of Higher Surgical Training representing the Royal Colleges of Surgeons, the Association of Professors of Surgery and the Specialists Surgical Associations in Great Britain and Ireland. To date WEM personally treated 10,000 patients with traumatic Spinal & Spinal Cord Injuries. WEM developed, and led the Midland Centre for Spinal Injuries (MCSI) between 1983 & 2014 and raised £6 million from Charity to: Rebuild and furnish the Centre, help with the development of laboratory research and the development of two purpose built bungalows for Transitional Housing of patients in the Community. El Masri took responsibility for the management of the injured spine and its consequent multi-organ malfunction as well as the range of non-medical and physical effects of cord injury in the acute, subacute, rehabilitation phases as well as in the long term. WEM lectures worldwide in developed and developing countries. El Masri contributed to the literature with 155 publications and published his observations on pin prick sensory sparing as a prognostic indicators of spontaneous neurological recovery following Traumatic Spinal Cord Injuries. El Masri published a long term population based incidence and characteristics of Bladder Cancer as well as the long term population based incidence and clinical presentation of Post Traumatic Syringomyelia in Patients with Spinal Injuries. El Masri introduced the concepts of “Physiological Instability of the Injured Spinal Cord”, “Time Related Biomechanical Instability of the Injured Spine” and “Micro-instability of the injured spine”. WEM highlighted the importance of the integrated holistic simultaneous adequate management of the injured spine together with all the effects of cord damage from the early hours/days of injury to prevent further mechanical and non-mechanical damage to the injured spinal cord and cauda equina. El Masri demonstrated that with adequate simultaneous. Active Physiological Conservative Management of all the physiologically impaired and malfunctioning organs of the body including the spinal injury, the presence of pin prick sensory sparing is a predictor of spontaneous neurological recovery. El Masri also demonstrated that this neurological recovery occurs irrespective of the degree of Biomechanical Instability on Xrays and irrespective of the degree of Canal encroachment and Cord Compression on CT and MRI. WEM is Peer reviewer for a number of Journals. WEM held the offices of: President of the International Spinal Cord Society, Chairman of the British Association of Spinal Cord Injury Specialists and Executive Member of the BSRM. Founder Member and trustee of SPIRIT Educational Charity in Spinal Injuries and Transhouse Charity that provides interim accommodation between hospital and home for patients. El Masri raised about six million pounds from charity to rebuild and furnish the MCSI. Advisor to WHO‘s & Co-author of the WHO International Perspectives on Spinal Cord Injury which was published in 2013, Member of the NICE Guideline Developing Group in spinal injuries. El Masri received a number of awards including: The Medal of the International Spinal Cord Society, National Hospital Doctor Team Award for Innovation, Paul Harris Fellowship of the Rotary Club Outstanding achievement award from the Chinese Society of Spinal Injuries, Outstanding Consultant Achievement award by the Spinal Injury Association, Hon. Presidency of the Romanian Spinal Cord Society. El Masri was commended in the House of Lords on two

occasions. WEM's is an advocate for the demonstration of evidence based clinical management and the right of the patient to make a fully informed choice between the various methods of treatment including that of the injured spine. El Masri strongly advocates for the management of patients by knowledgeable, well trained, experienced Clinicians supported by a team of Health Care professional in Specialised Spinal Cord Injury Centres with a fit for purpose infrastructure from the early hours or days following injury to enable the team to meet all the medical and non-medical needs of patients with such rare and complex condition.

Traumatic Spinal Cord Injuries (tSCI)-Is the management of the injured spine based on the various radiological presentations evidence-based?

Prior to WWII the majority patients with tSCI died in hospitals. There was however no shortage of Clinicians experimenting with the management of the injured spine.

During WWII L. Guttmann (a well-trained aggressive Neurosurgeon) was given the task of looking after injured soldiers & officers with tSCI at Stoke Mandeville Hospital in the UK. By studying the condition and the causes of death in a large number of patients, he realised that patients died or developed further neurological damage from various complications caused by the multi-organ Physiological impairment and malfunction caused by the neural tissue injury and not from the Spinal Injury (SI). Some died because of additional complications from surgical interventions on the injured spine.

By providing a Holistic Model of Service Delivery that attends to all the patho-physiological medical and non-medical effects of cord damage as well as the injured spine by what can be described as Active Physiological Conservative Management (APCM), Guttmann demonstrated that all complications can be prevented or diagnosed and treated early, some patients exhibit various degrees of neurological recovery and the great majority of patients can live long, healthy, dignified, productive and often competitive lives.

In 1967 Frankel et al studied the neurological outcome of 612 patients treated by APCM and demonstrated that the majority of patients who retained sensory sparing but had no visible or palpable motor sparing following the injury exhibited the recovery of motor power from the motor neurone adjacent to the spared sensory tracts. Surprisingly they found that the neurological recovery occurred irrespective of the radiological presentation on Xrays at admission (within 15 days of injury) and on discharge. They published their results in 1969 in what has been known since as the Frankel Classification. This was the first population outcome study that correlated the presentation and outcomes of patients presenting with sensory and sensory-motor sparing. Their findings have been confirmed by various international groups of clinicians dedicated to the management of patients with tSCI.

Zhenhuan Liu

Nanhai Maternity and Children's Hospital, Affiliated Guangzhou University of Chinese Medicine, China

Biography: Zhen-Huan LIU professor of pediatrics, Pediatric acupuncturist Ph.D. tutor. Zhen has been engaged in pediatric clinical and child rehabilitation for 40 years. Led the rehabilitation team to treat more than 40,000 cases of children with intellectual disability, cerebral palsy and autism from China and more than 20 countries, more than 26800 childrens deformity returned to school and society and became self-sufficient. The rehabilitation effect ranks the international advanced level. Vice-chairman of Rehabilitation professional committee children with cerebral palsy, World Federation of Chinese Medicine Societies. Visiting Professor of Chinese University of Hong Kong in recent 10 years. Zhen is most famous pediatric neurological and rehabilitation specialists in integrated traditional Chinese and Western medicine in China. Zhen has edited 10 books. Zhen has published 268 papers in international and Chinese medical journals.

Scalp acupuncture with functional electrical stimulation for the treatment children with autism spectrum disorder

Background: Autism Spectrum Disorders (ASD) are a series of neurodevelopmental disorders characterized by social disorders, rigid behaviors and narrow interests. The World Health Organization (WHO) estimates that the prevalence of ASD has been increasing over the past 50 years. With one in 48 children, ASD has become a global public health problem. Currently, there is no effective drug treatment for children with ASD, and there is no effective medical treatment. Education of these ASD children by special education methods alone has a poor outcome, with 75% of ASD children failing to achieve normal or cure. And 80% of ASD children suffer from mental retardation, ADHD, epilepsy, emotional sleep disorders and so on. It can cause pain and suffering for ASD children and their parents. The effects may persist into adulthood.

Objective: The purpose of this study was to investigate the effect of head acupuncture therapy on core symptoms, quality of life and communication ability of children with ASD. Our team conducted a controlled study of head acupuncture therapy in 198 children diagnosed with ASD. The clinical diagnostic criteria of children with ASD who were selected for head acupuncture treatment met the DSM-5 criteria. Each child and parent signed an informed consent form.

Methods: 198 children with ASD were randomly divided into two groups. Acupuncture treatment group 89 cases, received head acupuncture therapy and the control group 89 cases received special education and speech therapy for 3 months. Clinical evaluation methods were ATEC, ABC, CARS and Gesell developmental scales. Pre-and post-treatment assessments were performed. The age of the two groups was 3-8 years old, and the gender, degree of illness, comorbidities, family education and rearing methods, course of disease and other factors were statistically analyzed. There was no significant difference between the two groups, and there was a certain comparability between the two groups. Both groups were evaluated on the ATEC, ABC, CARS and Gesell scales before starting rehabilitation. CNRAT method, Zhijiu acupuncture and precise body surface projection in functional language area of cerebral cortex were selected for head acupuncture. Broca and Wennicken area were simultaneously stimulated by acupuncture. Acupuncture is performed every other day. After acupuncture, electrical acupuncture was given to stimulate the language area for 15 minutes, every 10 times of acupuncture, rest for 15 days. A second clinical evaluation was conducted 3 months after acupuncture.

Results: The improvement of core symptoms in the head acupuncture treatment group was better than that in the control group. The initial clinical improvement was in abnormal visual communication, improvement of sleep and mood, and the following clinical effects were alleviation of rigid behavior, improvement of attention, and improvement of verbal and social communication ability. Assessment of these scales reflects a gradual improvement in these core symptoms. But these changes were not significant in the control group.

Conclusion: The research results showed that head acupuncture therapy could significantly improve the core symptoms of ASD children, such as extreme loneliness, eye contact disorder, language repetition, compulsive agreement, and indifference, significantly regulate the abnormal EEG of ASD children, and positively promote the cognitive level of low-functioning ASD children. The clinical efficacy of the treatment of ASD was not closely related to age. Electrocephalic acupuncture can be used as an effective supplement and alternative medicine therapy in the clinical treatment of ASD. The popularization and application of head acupuncture therapy can improve the quality of life of ASD children and reduce the economic burden of society and family.

Since 2004, Nanhai women's and children's hospital affiliated to Guangzhou University of Chinese Medicine has applied our original pediatric neurorehabilitation head acupuncture therapy to treat ASD and achieved good clinical efficacy. In order to further promote the application, our research group obtained the exact clinical effect confirmed by scientific evaluation through the clinical validation study and clinical follow-up of 1000 cases of ASD. We also receive pediatricians from all over the world who come to our hospital in China to study head acupuncture therapy for ASD. Doctors and rehabilitation therapists from Switzerland, Australia, the United States, Germany, Egypt, Russia, Kazakhstan and other countries have come to our hospital to study the clinical application of head acupuncture therapy in ASD.

Keywords: Autism Spectrum Disorder, Acupuncture, Scalp Electroacupuncture.

12TH EDITION OF

International Conference on

NEUROLOGY AND NEUROLOGICAL DISORDERS

HYBRID EVENT

22-24 JUNE 2026

NEUROLOGY

JUNE 22-24 | 2026

ORAL PRESENTATIONS

Aanjaneya Mamgain[1*], Anglina Kisku[2]

[1]National Institute of Pharmaceutical Education and Research, Guwahati, Assam, 781101, India

[2]Department of Pharmacy, DIT University, Dehradun, Uttarakhand, 248009, India

Investigating the therapeutic potential of *Moringa oleifera* bioactive compounds in NF-κB-mediated autism pathogenesis

Nuclear Factor-kappa B (NF-κB) is a key regulator of inflammation and apoptosis implicated in the pathogenesis of Autism Spectrum Disorder (ASD). Aberrant NF-κB activation has been observed in the brain, particularly in microglia, and peripheral blood of individuals with ASD, contributing to neuroinflammation and affecting neuronal function, cell survival, and synaptic plasticity. This study investigates the potential neuroprotective effects of *Moringa oleifera* leaf aqueous extract on NF-κB-associated pathways in ASD. Qualitative analysis and gas chromatography-mass spectrometry revealed the presence of various bioactive compounds in the extract, including alkaloids, tannins, flavonoids, proteins/amino acids, glycosides, reducing sugars, alpha-tocopherol, gamma-tocopherol, undecanoic acid, and vitamin E. In silico studies suggest that these constituents may target NF-κB-related biomarkers such as glycogen synthase kinase-3, COX-1, COX-2, and cytokine receptor complexes, potentially exerting neuroprotective effects. The involvement of NF-κB in ASD pathogenesis highlights its potential as a therapeutic target, and the bioactive compounds in *Moringa oleifera* leaf extract may offer a promising approach to address the underlying inflammatory processes. However, further research is needed to elucidate the causal relationship between NF-κB activation and ASD and to explore the efficacy of *Moringa oleifera* leaf extract as a potential treatment for this neurodevelopmental disorder.

Biography

Dr. Aanjaneya Mamgain, Project Scientist at the National Institute of Pharmaceutical Education and Research, Guwahati. Mamgain hold a B.Pharm. from Dr. A.P.J. Abdul Kalam Technical University, an M.Pharm. in Pharmaceutical Biotechnology from Jamia Hamdard, and a Ph.D. in Pharmaceutics from Indira Gandhi National Tribal University. With 7 years of research experience, Mamgain specialise in nanomedicine, biotechnology, and herbal nanotechnology, with expertise in advanced analytical instruments. Dr. Mamgain has published 2 research papers, 4 book chapters, and 2 Indian patents. Mamgain have presented at 12 national and international conferences, receiving a best e-poster award.

Aditya Seth[1*], Megha Luthra[1], Tushar Marbate[2], Puneet Ohri[1], Ashwani Sharma[1]

[1]Shri Guru Ram Rai Institute of Medical and Health Sciences, India

[2]All India Institute of Medical Sciences, India

A longitudinal study of site prevalence associated clinical and socio-demographic factors, treatment and outcome in non-traumatic intra-cranial hemorrhage patients presenting at a tertiary care facility in NCR, India

Aim/Background: To study prevalence and outcomes associated with clinico-socio-demographic factors in treated NTICH (Non-Traumatic Intra-Cranial Hemorrhage) patients in Institute of Human Behaviour and Allied Sciences (IHBAS).

Materials and Methods:

- **Study Design:** Hospital-based cross-sectional study.
- **Study Site:** Institute of Human Behaviour and Allied Sciences (IHBAS), Delhi.
- **Study Period:** 33 months (January 2022 to September 2024).
- **Study Population:** All newly and previously diagnosed NTICH patients presenting to the Neurosurgery Department.
- **Selection Criteria:** Mandatory written informed consent from patient or caregiver.
- **Inclusion:** Diagnosis confirmed via clinical history and NCCT Brain imaging.
- **Exclusion:** Patients referred out or those who Left Against Medical Advice (LAMA).
- **Sample Size & Technique:** 100 cases selected via total enumeration (census method) to reflect real-time patient load.
- **Data Collection:** Consecutive enrollment based on anatomical classification of hemorrhage on NCCT. Data was gathered using a structured questionnaire covering GCS scores, clinical/biochemical features, socio-demographics, treatment, and outcomes.
- **Data Analysis:** Statistical analysis performed using the latest version of SPSS.

Results: This study analyzes 100 NTICH cases collected via total enumeration over 33 months. Diagnosis and anatomical classification were confirmed using NCCT Brain imaging. The analysis correlates clinico-socio-demographic variables and GCS scores with patient outcomes.

Ethical Status: Final statistical analysis and data presentation are pending Institutional Ethics Committee (IEC) approval. Upon clearance, the full dataset will be processed using SPSS to determine definitive prevalence rates and clinical associations.

Discussion/Conclusion: This study highlights the critical need for structured longitudinal data in managing non-traumatic intracranial hemorrhage within a tertiary care setting. The definitive conclusions and statistical inferences will be established upon receiving ethical clearance.

Keywords: Non-Traumatic Intracranial Hemorrhage (NTICH), Prevalence.

Biography

Dr. Aditya Seth is a dedicated medical professional and current Post-Graduate Resident in MD Community Medicine (Preventive Medicine and Public Health) in Dehradun, Uttarakhand. With a robust clinical foundation, Seth previously served as a Medical Officer in Neurology and a Junior Resident in Neurosurgery, where Seth was recognized for his research on iatrogenic dural tears and stroke. A high achiever who secured a 98.2 percentile in the All India Medical Entrance, Dr. Seth is an active life member of the AIDS Society of India, Epidemiological foundation of India, Indian medical association, IAPSM, etc. Seth has presented multiple papers at national and international levels, focusing on bridging gaps in public health and clinical excellence.

Aleksandra Olejniczak[1*], Klaudia Marek[1], Elżbieta Miller[1], Igor Zubrycki[2]

[1]Department of Neurological Rehabilitation, Medical University of Lodz, Lodz, Poland

[2]Institute of Automatic Control, Lodz University of Technology, Lodz, Poland

Robotics and mirror-therapy-based neurorehabilitation for stroke patients

Aims: Upper limb paresis, which includes disorders of extension and wrist control, is one of the most common and debilitating complications after a stroke. It significantly impacts patients' independence. Conventional rehabilitation methods do not always effectively improve this function long term, which justifies searching for new therapeutic solutions. In response to this need, a device with a soft balloon actuator called the Balonikotron has been developed to assist with wrist extension exercises using a balloon mechanism and audiovisual feedback. This pilot study aimed to evaluate the impact of a four-week therapy program using the Balonikotron on motor function, functional independence, and muscle tone in patients with upper limb paralysis after a stroke.

Material and Methods: A pilot study involving 12 stroke patients with upper limb paresis was conducted. The patients were randomized in a 1:1 ratio to the study group (n=6) or the control group (n=6). Hospitalization lasted four weeks and included a comprehensive neurological rehabilitation program. Both groups underwent a four-week therapeutic program aimed at restoring hand function as part of standard rehabilitation. The study group also participated in therapy using an innovative device with a balloon mechanism and mirror therapy elements. Assessments were performed before and after the intervention using the following scales: The Fugl-Meyer Assessment for the Upper Limb (FMA-UE), the Modified Ashworth Scale (MAS), the Activities of Daily Living (ADL), Modified Rankin Scale (mRS), Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), wrist Range of Motion (ROM), and Barthel Index (BI).

Results: After the intervention, significant improvements in upper limb motor function (as assessed by the FMA-UE, Cohen's d=1.18, p=0.045) and independence (as assessed by the ADL and BI, p=0.02, p=0.009) were observed. The average improvement in the experimental group was 19.17 points for the BI, while the control group's average improvement was only 6.67 points. The descriptive analysis of the MAS scale revealed no increase in spasticity among the patients. In fact, a decrease in spasticity was noted in some patients compared to the initial measurement. The range of wrist extension improved by 14.5° in the experimental group, while it deteriorated by 5.17° in the control group. An upward trend was observed in both the MMSE and MoCA scales, but the differences between the groups did not reach statistical significance (p=0.151 for the MMSE and p=0.187 for the MoCA).

Conclusion: Including robots in the neurological rehabilitation program for stroke patients can significantly support the recovery process. Using additional therapies with specialized devices promotes improvement in upper limb function, including wrist extension, and increases functional independence. The Balonikotron device, which is based on the mirror therapy mechanism, is a valuable addition to the rehabilitation of patients with hand paresis following a stroke.

Biography

Aleksandra Olejniczak is a Physiotherapy student, member of the Student Scientific Society of Neurorehabilitation operating at the Department of Neurological Rehabilitation, Medical University of Lodz, speaker at international conferences, co-author of a publication on the use of robots in neurological rehabilitation of patients after stroke. Research interests include the application of robotics in neurorehabilitation as well as rehabilitation after stroke and in the course of multiple sclerosis.

Dr. Andrea Dincher
Saarland University, Germany

Effects of multimodal therapy on psychological symptoms in Huntington's disease

Introduction: Huntington's disease is a hereditary neurological disorder that is currently incurable. Only the symptoms can be treated, often with multimodal therapy. Some studies suggest that such a multimodal therapy can improve psychological symptoms in Huntington's disease patients, but relatively little is known (Bartlett et al., 2020). This meta-analysis therefore aims to show whether there are indeed effects of multimodal therapy on psychological symptoms in Huntington's disease.

Methods: PRISMA-guidelines (Page et al., 2021) are followed:
Sources: PEDro, PubMed, Web of Science, Scopus.
Search Terms: Multimodal therapy or physical therapy or rehabilitation and Huntington's disease.
Inclusion Criteria: Experimental studies, multimodal therapy, Huntington's disease, publication between 1990 and 2024, human participants.
Methodological Quality: PEDro score (Verhagen et al., 1998). Studies with at least a medium quality (a score≥5) are included into meta-analysis. Standardized mean differences with 95% confidence intervals (SMD<.30=low, >.50=medium, >.80=strong) are shown in forest plots (Verhagen, & Ferreira, 2014) using RevMan 5.4 software (The Cochrane collaboration, 2020).

Results: Of a total of 4833 publications, eight studies met the inclusion criteria and were methodologically analyzed. Four studies achieved the required PEDro total score of ≥5. The effects range from no effect of SMD=.00 for Hospital Anxiety and Depression Scale (HADS) depression subscale to a strong effect of SMD=-1.96 for Beck Depression Inventory (BDI). All effects are in favor to the experimental group.

Conclusions

- Only a few studies achieved at least average methodological quality.
- Various interventions with different durations and intensities show very different effect sizes.
- Programs with a high proportion of exercise seem to be the most effective.
- Further research with high-quality designs is needed.

Biography

Dr. Andrea Dincher Lecturer for special tasks at Saarland University, honorary president of the German Sports Teachers' Association in Saarland, trainer at a sports club for 30 years for apparatus gymnastics, long-distance running, dancing, orthopedics, heart disease, obesity, seniors, and occupational health management. Lecturer for continuing education in the field of neurological rehabilitation and elementary and primary education. Research interests: Training therapy for neurological diseases, child development, and developmental disorders.

Anglina Kisku*, Anurag Raturi, Mandeep Kumar Arora
DIT University, Uttarakhand, India

Modulation of neuroinflammatory pathways and oxidative stress by *Rhododendron arboreum* extract in stress-induced postpartum depression

Postpartum Depression (PPD) is a complex neuropsychiatric disorder frequently precipitated by chronic stress, with its pathophysiology involving the kynurenine pathway, neuroinflammation, and oxidative stress. Conventional antidepressants often exhibit limited efficacy and are associated with adverse effects, underscoring the necessity for safer, plant-based alternatives. This study sought to examine the neuroprotective effects of *Rhododendron arboreum* Organic Extract (RAOE) in rodent models of PPD induced by Chronic Social Stress (CSS) and Unpredictable Chronic Mild Stress (UCMS), with a focus on its influence on neuroinflammatory pathways, oxidative stress, and behavioral outcomes. Postpartum female rats were subjected to UCMS and CSS protocols, followed by oral administration of RAOE. Behavioral assessments, including the sucrose preference test, milk intake measurement, maternal care observation, and maternal aggression assessment, were conducted. Biochemical markers related to oxidative stress and inflammation were measured, alongside evaluations of cell viability and the kynurenine pathway. Histological and toxicity analyses were performed to support safety profiling. RAOE significantly enhanced behavioral performance by improving maternal care, milk intake, and sucrose preference, while reducing maternal aggression. The extract demonstrated potent antioxidant and anti-inflammatory activity, suppressed neuroinflammatory markers, and modulated kynurenine pathway metabolites. In vitro analysis confirmed cell viability and neuroprotective potential. Toxicological studies indicated safety at high doses (LD_{50}>2000mg/kg). RAOE exhibits promising antidepressant-like and neuroprotective effects in stress-induced models of PPD, likely through its antioxidant, anti-inflammatory, and kynurenine pathway-modulating actions. These findings support its potential as a plant-based intervention for postpartum depression, warranting further mechanistic and clinical investigations.

Keywords: Postpartum Depression, Kynurenine Pathway, Neuroinflammation, Antioxidant, Anti-Inflammatory, Cell Viability, Maternal Behavior, Milk Intake, Sucrose Preference, Maternal Aggression.

Biography

Anglina Kisku joined the Faculty of Pharmacy as an Assistant Professor in September 2024. Kisku earned her Ph.D. in Pharmacy from IGNT University, Amarkantak, with M. Pharm in Pharmacology from KIET School of Pharmacy, Ghaziabad, and a B. Pharm from MJP Rohilkhand University, Bareilly, India. Kisku research explores neuropsychiatric disorders, their management through traditional medicines, and innovative drug delivery systems. Kisku has been awarded travel grants, recognized with the Best Poster Award at ICNAND2023, and contributed papers to international conferences. Recently, Kisku secured second position in a DST-STUTI program for her project on millet-based therapy in maternal immune stress–induced autism.

Bhaskar Jyoti Dutta
Royal School of Pharmacy, The Assam Royal Global University, Guwahati, Assam, India

Pterostilbene orchestrates synaptic remodeling and mitochondrial functional reconstitution to attenuate ischemic vascular dementia

Vascular Dementia (VaD), a major contributor to cognitive decline, arises primarily from impaired regulation of cerebral circulation. Pterostilbene (PTE), a natural stilbene, exhibits potent neuroprotective properties, including antioxidative, anti-apoptotic, and cognition-enhancing effects; however, the molecular basis of its protective action in VaD remains poorly defined. Here, we integrated network pharmacology with in-vitro and in-vivo validation to delineate the mechanistic underpinnings of PTE. An ischemic injury model was established in SH-SY5Y cells using Oxygen-Glucose Deprivation/Reoxygenation (OGD/R), while VaD was induced in rats by bilateral common carotid artery occlusion. Cognitive function was assessed by behavioural paradigms, and neuronal integrity, vascular architecture, mitochondrial function, respiratory complex activities, and synaptic plasticity via the cAMP/PKA/CREB signalling cascade were evaluated using histological, biochemical, and molecular assays. Network pharmacology identified the cAMP pathway as a principal mediator of PTE activity. In ischemia injured SH-SY5Y cells, PTE improved viability, reduced oxidative stress, stabilized mitochondrial membrane potential, and elevated ATP production. In VaD rats, PTE enhanced spatial learning and memory, preserved cortical and hippocampal structures, and promoted mitochondrial health, evidenced by upregulation of PGC-1α and TFAM, restoration of respiratory complex activities, and preservation of mitochondrial ultrastructure. PTE also increased expression of synaptic proteins (PSD95, Synaptophysin). Consistently across both models, PTE activated the cAMP/PKA/CREB signalling axis. Collectively, these findings demonstrate that PTE mitigates ischemia-induced cognitive impairment by reversing mitochondrial dysfunction while sustaining synaptic plasticity through cAMP/PKA/CREB activation, highlighting its translational potential as a therapeutic candidate for VaD.

Biography

Bhaskar Jyoti Dutta is a neuropharmacology researcher specializing in neurodegenerative disorders, vascular dementia, and translational nanomedicine. Bhaskar completed his Ph.D. at National Institute of Pharmaceutical Education and Research Hajipur, focusing on mechanistic studies of synaptic dysfunction and phytostilbene-based therapeutics in vascular dementia. Bhaskar expertise includes behavioral neuroscience, molecular pharmacology, proteomics, metabolomics, and intranasal nanoformulations for brain-targeted delivery. Bhaskar has authored multiple publications in reputed international journals, received several national and international research awards, and holds patents in pharmacology and nanotherapeutics.

Bohong Wang*, Jinfeng Shang, Jiakang Jiao, Xin Liu
Beijing University of Chinese Medicine, China

Targeting GDF15 alleviates mitochondrial dysfunction and ferroptosis in cerebral ischemia-reperfusion injury

Growth Differentiation Factor 15 (GDF15) is a well-recognized neurotrophic and regulatory factor. Serum levels of GDF15 are an independent risk factor for stroke and an effective predictor of ischemic stroke outcomes. Currently, there is a lack of research on the mechanism of GDF15 in ischemic stroke. This experiment aims to elucidate the key role of GDF15 in cerebral ischemia. Compared with the control group, the expression of GDF15 increased in OGD/R PC12 cells and tMCAO rats. Exogenous GDF15 significantly improved the damage in OGD/R PC12 cells and tMCAO rats, and this protective effect was related to its antioxidant, anti-inflammatory and anti-ferroptosis properties. In vitro, knockdown of GDF15 by siRNA exacerbated cell damage. The mechanism was verified by transcriptomics, RT-qPCR, Western Blot and immunofluorescence techniques. Kevetrin (a p53 activator) and Pifithrin-α (a p53 inhibitor) were used to detect the key role of GDF15 in the antioxidant and anti-ferroptosis mechanism. The mechanism study showed that GDF15 has a regulatory relationship with p53 and NOX4. p53 can promote the expression of GDF15, and GDF15 can inhibit the nuclear translocation of p53 and reduce the expression of NOX4, thereby regulating mitochondrial electron transport chain (such as SDHB, UQCRC2, ATP5A) and ferroptosis (TF, GPX4, SLC7A11) related targets, reducing ROS production, alleviating oxidative stress damage, improving mitochondrial dysfunction and inhibiting neuronal cell ferroptosis. GDF15 plays an important role in ischemic stroke, and targeting GDF15 is a potential strategy for the treatment of ischemic stroke.

Biography

Wang Bohong, a master's student in clinical Chinese medicine at Beijing University of Chinese Medicine. Bohong research focuses on the pharmacology of traditional Chinese medicine and cerebrovascular diseases. Published a paper as a co first author in the renowned

pharmacology journal, Journal of Ethnopharmacology (IF=5.4), named Shuxuetong injection inhibits pyroptosis in acute ischemic stroke via CD44/NLRP3/GSDMD signal. In addition, Bohong have participated in the publication of 6 Chinese core journal articles and 2 English articles.

Chuck Easttom
Vanderbilt University, United States

Machine learning and Brain Computer Interfaces (BCI)

Machine learning and Brain-Computer Interface (BCI) technologies are converging to create transformative possibilities in neuroscience, medicine, and human-machine interaction. BCIs enable direct communication between the brain and external devices by decoding neural signals, while machine learning provides the computational foundation for interpreting these complex data patterns. This presentation explores recent advances in machine learning methods-particularly deep learning and reinforcement learning-that have dramatically improved the accuracy, adaptability, and real-time performance of BCIs. Applications range from neuro prosthetic control and rehabilitation for individuals with motor impairments to cognitive enhancement, emotion recognition, and adaptive neuro feedback systems. The session also examines challenges such as data scarcity, signal variability, model interpretability, and ethical considerations surrounding privacy and autonomy. By integrating insights from neuroscience, artificial intelligence, and systems engineering, this talk aims to outline both the current state and future directions of intelligent BCIs, emphasizing how machine learning is bridging the gap between thought and action.

Biography

Dr. Chuck Easttom is the author of 45 books, including the book 'Machine Learning for Neuroscience'. Easttom is also an inventor with 27 patents and the author of over 80 research papers. Easttom holds a Ph.D. in computer science, a Ph.D. in Nanotechnology, and a Doctor of Science in Cyber security, and four master's degrees (one in applied computer science, one in education, one in Strategic and Defense studies, and one in systems engineering). Dr. Easttom was a member if the IEEE Brain Computer Interface standards committee. Dr. Easttom is currently an adjunct professor for Georgetown University and for Vanderbilt University.

Daniel Zhang
Canyon Crest Academy, 5951 Village Center Loop Road, San Diego, CA 92130, USA
Pasadena Bio Collaborative Labs, Pasadena, CA, USA

Sex-dependent glial architecture in the prefrontal cortex and its relevance to glioblastoma

Background: Female Glioblastoma (GBM) patients consistently outlive males by 3 to 5 months even after controlling for age, surgical extent, and MGMT methylation status. The biological mechanism driving this survival gap remains unresolved. Nearly all existing research examines glial biology after tumor formation, leaving pre-tumor sex differences in glial architecture unstudied. This study addressed that gap through two complementary analyses.

Methods: Study 1 quantified glial cell density across three subregions of the rat medial Prefrontal Cortex (mPFC)-the Anterior Cingulate Area (ACA), Prelimbic area (PL), and Infralimbic Area (ILA)-using DAPI nuclear morphology and automated image J particle analysis (n=3 per sex). A dorsal-to-ventral gradient analysis compared ACA density to pooled ventral mPFC density by sex using two-way ANOVA with Fisher's LSD post-hoc. Study 2 analyzed GFAP and AIF1 gene expression in 37 human GBM tumor blocks from the Ivy Glioblastoma Atlas Project (Ivy GAP; 19 male, 18 female). A two-way ANOVA examined the effect of sex and EGFR amplification status on the $\log_2$(GFAP/AIF1) expression ratio in 28 blocks with available EGFR data.

Results: In Study 1, male rats showed a statistically significant dorsal-to-ventral glial density gradient (ACA: 78.3 vs. pooled ventral: 175.7 objects/100,000 px^2; p=0.047). Female rats showed a numerically comparable gradient that narrowly missed significance (p=0.068), with large effect sizes in the ACA (Hedges' g=1.91) and ILA (g=1.18), consistent with insufficient power at n=3. In Study 2, neither GFAP nor AIF1 differed by sex in isolation. However, when patients were stratified by EGFR amplification status, amplification was significantly associated with an elevated GFAP/AIF1 ratio in male patients (amplified: 1.420 vs. non-amplified: 0.319; p=0.010) but not in female patients (0.876 vs. 0.658; p=0.552).

Conclusions: These findings suggest that sex-dependent glial differences are expressed through organizational responsiveness to perturbation rather than absolute cell quantity. The female tumor microenvironment appears more resistant to EGFR-driven oncogenic disruption of the astrocyte-to-microglial balance, which may partly explain the persistent female survival advantage in GBM. EGFR-amplified male patients may represent a biologically distinct subgroup with an astrocyte-dominant microenvironment that suppresses cytotoxic T cell infiltration and warrants sex-stratified analysis in future checkpoint inhibitor trials.

Keywords: Glioblastoma, Sex Differences, Glial Biology, Tumor Microenvironment, EGFR Amplification, Prefrontal Cortex.

Biography

Daniel Zhang is a high school junior at Canyon Crest Academy in San Diego, California. Daniel conducts cancer biology research through Pasadena Bio Collaborative Labs, focusing on sex-dependent glial biology and glioblastoma. Daniel broader research experience includes single-cell RNA sequencing of bladder cancer models at UCSC, multi-omics analysis of renal cell carcinoma at the UC Davis Comprehensive Cancer Center, and bladder cancer multi-omics research at Cambridge University. Daniel is interested in translational oncology and sex-stratified approaches to precision medicine.

Danièle Lapointe
Université Laval, Québec, Canada

Study of resilience in cases of incest, father-daughter, step-father and step-daughter in the pre-pubber and puberous period among adult women and mothers: How to overcome the traumatisms of an incestuous relationship?

Sexual abuse of a child is a form of maltreatment. Among recent studies in this area, the Canadian study (Burczycka and Conroy, 2017) indicates that 70.3% are intrafamilial abuse and that of all cases of sexual abuse, 81% of victims are female (Silva and Collin-Vézina, 2017) and that the majority of victims feel deleterious effects (Bilan DPJ-DP, 2017; Koçtürk and Yuksel, 2019). However, over the past thirty years, research has highlighted the possibility of recovery for victims of incest (Berthelot et al., 2019). These "so-called resilient" people used adaptive strategies allowing them to protect themselves from the trauma of the abuse of which they were victims (Barnes and Josefowitz, 2014; de Becker and Maertens, 2015). This thesis reports the results of research carried out with 33 adult women and mothers who experienced incest during childhood and/or adolescence at the hands of their father or stepfather. To do this, a mixed analysis (Student's t test and multiple regression analysis) was used to establish two profiles and to respond to the following three hypotheses: Adaptation strategies, the attachment bond and mentalization. The first results converge with the state of knowledge on the issue, since 21 refers to participants with traditional clinical impacts and 12 refers to participants engaged in a resilience process. Student's t test results showed no difference, but Cohen's (1988) d effect size indicated higher d at subscales and multiple regression analysis showed three unexpected results. The results of the qualitative analysis are in the same direction as those above. To conclude, the present doctoral study corroborates research which shows that resilience is difficult to operationalize.

Biography

Ms. Lapointe (Ph.Ds., M.Sc. Couseling, B. Sc. Ps.) is a clinical psychologist practicing in private practice. For several years, she has been a lecturer at Universite Laval and Universite of Montreal for undergraduate and graduate students. Lapointe also supervises psychologists and psychotherapists, as well as foreign students. Lapointe has participated as speaker in symposia, conferences, sand scientific congresses. Lapointe was an expert in family matters at the Quebec Youth Chamber, and Lapointe was the clinical director of the Traumatys Center. Lapointe as several scientific publications. Lately, Lapointe is also evaluating for the Swiss National Science Foudation (SNSF).

Dixie J Woolston
Mayo Clinic Arizona, United States

Cognition as the fifth vital sign: Integrating neuropsychological assessment and neuroimaging in modern neurology practice

Background: Cognitive symptoms are among the most common reasons patients seek neurological care, yet cognition is often assessed inconsistently in routine clinical practice. As neurological disorders increasingly affect aging populations worldwide, early identification of cognitive dysfunction has become critical for diagnosis, treatment planning, and patient safety. Advances in computerized cognitive screening and multimodal neuroimaging-including structural MRI, Diffusion Tensor Imaging (DTI), and Functional MRI (fMRI)-offer new opportunities to integrate cognitive phenotyping with brain network characterization in everyday neurology practice.

Objective: This presentation reviews current strategies for incorporating structured cognitive assessment into neurology clinics and highlights the complementary role of neuroimaging in evaluating cognitive dysfunction across neurological conditions.

Methods: We review contemporary literature and emerging clinical models integrating brief cognitive screening tools, comprehensive neuropsychological evaluation, and multimodal neuroimaging approaches. Particular emphasis is placed on the clinical utility of computerized cognitive screening platforms, structural MRI markers of neurodegeneration and cerebrovascular disease, diffusion tensor imaging measures of white matter integrity, and functional MRI for mapping language, memory, and executive networks.

Results: Integrating cognitive assessment with neuroimaging enhances diagnostic precision and clinical decision-making across a wide range of neurological conditions. Brief cognitive screening tools can function as efficient triage mechanisms within neurology clinics,

identifying patients who may benefit from comprehensive neuropsychological evaluation or targeted neuroimaging. Multimodal imaging provides additional insight into neural network dysfunction in disorders such as neurodegenerative disease, vascular cognitive impairment, epilepsy, brain tumors, traumatic brain injury, and post-viral syndromes including long COVID. Advanced neuroimaging techniques also play an increasingly important role in pre-surgical brain mapping and treatment planning.

Conclusion: Cognition should be considered a "fifth vital sign" in neurological care. Integrating standardized cognitive assessment with advanced neuroimaging allows clinicians to move beyond symptom-based diagnosis toward network-based understanding of neurological disease. Adoption of scalable cognitive screening workflows and multimodal imaging strategies has the potential to improve diagnostic accuracy, guide treatment planning, and ultimately enhance patient outcomes in modern neurology practice.

Keywords: Cognitive Assessment, Neuropsychology, Neuroimaging, Functional MRI, Diffusion Tensor Imaging, Cognitive Screening, Neurology Practice.

Biography

Dr. Dixie Woolston obtained her PhD in 2006 from University of Texas Southwestern Medical Center. Woolston completed a 2-year fellowship in Clinical Neuropsychology also at UT Southwestern. Woolston has worked in a variety of clinical settings, including the VA, a large private neurology practice, and currently is the Division Chair of Neuropsychology at Mayo Clinic Arizona. Research interests include neuroimaging, Alpha-Stim device, and optimizing brain health.

Er. Kritika

Independent Researcher, India

Securing brain-computer interfaces: Emerging threats and defense strategies

Brain-computer interfaces are on a cross road with BCIs quickly moving out of the research avenues to clinical use and consumer applications with more than 400 ongoing clinical studies around the world and market estimates of more than $5 billion in the next decade. However, as these systems start to reach households and the everyday lives of patients, we have a burning question that the neuroscience community has not really given much thoughtful consideration on, and that is are BCIs safe in the real world environment?

The given talk brings up the next area of neuro-cybersecurity, which is a jaw dropping break in the modern development of BCI as less than 5% of the BCI studies published touch on the issue of security. With BCIs turning into cyber-physical systems operating wheelchairs, prosthetics, and devices to support the vulnerable groups in communication, they also acquire critical vulnerabilities that provide adversaries with access to vulnerable populations, infringe neural privacy, and endanger the integrity of research.

The talk illustrates four key categories of threats, including adversarial attacks with 80-95 percent misclassification rates with imperceptible signal perturbations, privacy violations that allow to extract PINs and medical data out of neural data, model poisoning and introducing hidden front doors in machine learning systems, and physical layer attacks that can use commodity-based hardware to corrupt signals via electromagnetic interference. All the threats are evidenced in published research, and not speculation. The talk will also introduce applications such as practical defense mechanisms such as adversarial training, differential privacy, encrypted communication and real time anomaly detection, all of which retain classification accuracy of over 90 percent with a latency overhead imposed.

There is just time before BCIs can be mass deployed. This talk will help create a fundamental change in the approach of neuroscientists towards the development of BCI by realizing that the future of neural interfaces does not lie in extending the accuracy of decoding but rather in creating secure, trusting systems that patients can expect and regulators can insist. Not whether they will be attacked, but whether we shall get them secured the first thing.

Biography

Er. Kritika is a dynamic and experienced interdisciplinary researcher specializing in the bridging of human, technical, and organizational perspectives of digital risk. Kritika work spans over the disciplines of cybersecurity, generative AI, neuro-security, machine learning, psychology etc. With a substantial portfolio of scholarly publications and keynote engagements, Kritika insights have been widely recognized across various academic and industry platforms. In addition, Kritika also serves as a technical reviewer for academic conferences, journal reviewer for top journals and book and book chapter reviewer.

Gennaro Della Rocca

Consultant Physician, AORN Antonio Cardarelli Hospital, Emergency and Critical Care Department, UOC Neurology & Stroke Unit, Via Antonio Cardarelli 6, 80131 Naples, Italy

Is time born from the brain, or is it the other way around?

This presentation proposes a theoretical model positioning the inner ear-particularly the vestibular system-as a key contributor to neural synchronisation processes and cognitive network regulation. Traditionally viewed through its role in balance and spatial orientation, the vestibular system is here, reinterpreted as a sensory interface for temporal information, potentially modulating circadian rhythms and higher-order cognitive functions.

From an evolutionary perspective, both the vestibular apparatus and cochlea respond to periodic stimuli–movement and sound, respectively-highlighting their shared capacity to detect rhythmic inputs. These cyclical inputs underpin distinct yet complementary functions: Postural control and spatial representation in the vestibular system and language processing and social communication in the cochlear system.

Critically, vestibular projections reach multiple cognitive domains, positioning the inner ear as a temporal coordinator of distributed neural networks. The timing signals it conveys may facilitate plasticity (via long-term potentiation), enable inter-areal communication (via coherence), and promote resonance-based synchronisation even across anatomically unconnected regions. Visual and vestibular afferents function as temporal and spatial reference frames, respectively, which are essential for the accurate reconstruction of external reality.

These mechanisms suggest therapeutic potential: Rhythmic stimulation-whether auditory, vestibular or multimodal-could enhance neuroplasticity and restore connectivity patterns in neuropsychological disorders. By framing rhythm as a shared medium between neural substrates and behavioural functions, this model supports the integration of vestibular-based interventions in cognitive rehabilitation, particularly in conditions involving dysregulated timing, such as Parkinson's disease, neglect, or affective disorders.

Biography

Gennaro Della Rocca graduated in Medicine and Surgery in 1991 and specialized in Neurology in 1996. After working as a medical consultant at the "Villa Camaldoli" Clinic in Naples, where Rocca focused on cognitive rehabilitation, Rocca has been serving since July 2020 at the "Antonio Cardarelli" Hospital (AORN) in Naples in the Neurology & Stroke Unit, primarily dealing with dementia. Rocca professional experience has been centered on dementia and cognitive rehabilitation, and Rocca has authored several scientific papers, including Conchiglia G., Della Rocca G., Grossi D. On a peculiar environmental dependency syndrome in a case with fronto-temporal damage: Zelig-like syndrome. Neurocase, 2007, 13(1):1–5; Abate F., Della Rocca G., et al. The "zig-zag" sign in Progressive Supranuclear Palsy. Parkinsonism & Related Disorders, 2020; 79:86–87; and Della Rocca G. Is the Inner Ear the Brain's Metronome? A New Vestibular Function in a Theoretical Model. Journal of Psychiatry and Cognitive Behaviour, Gavin Publishers, 2025. In September 2024, Rocca published the book Is Time Born from the Brain or Is It the Other Way Around? (Giammarino Publishers), the culmination of ten years of research on the subject.

Hengchao MA[1*], Shijun LI[2], Jun Zhang[1]

[1]Senior Department of Neurosurgery, Chinese PLA General Hospital, China

[2]Department of Radiology, Chinese PLA General Hospital, China

Ultrashort wave modulates brain functional connectivity and behavioral recovery in mTBI rats: A longitudinal 11.7T rs-fMRI study

Background: Mild Traumatic Brain Injury (mTBI) is common, with subtle symptoms and long-term consequences, but the brain network remodeling and its link to behavioral disorders remain unclear. Ultrashort Wave (USW) therapy, a potential non-invasive treatment, has not been systematically studied for its role in regulating this process and aiding recovery.

Methods: Male Sprague-Dawley (SD) rats were randomly assigned to three groups: mTBI model group (mTBI), Ultrashort Wave Intervention group (USW), and control group (Ctrl). An mTBI model was established via a single explosive shock, and the USW group received intervention post-injury. Serum biomarkers (Tau, S100B, NEFL), resting-state functional Magnetic Resonance Imaging (rs-fMRI) at 11.7T, and behavioral assessments (open field test, elevated plus maze test) were conducted on days 4, 14, and 30 post-injury. Functional Connectivity (FC) between Regions of Interest (ROIs) in the whole brain was analyzed using the SIGMA standard brain atlas. Longitudinal changes in FC were modeled using a Linear Mixed-Effects Model (LMM), and FC×group interaction analysis was performed to investigate whether ultrashort wave intervention altered the relationship between FC and behavioral outcomes in mTBI rats.

Results: Compared to the Ctrl group, the mTBI group exhibited early post-injury motor deficits and anxiety-like behaviors, accompanied by elevated serum levels of Tau, S100B, and NEFL. ultrashort wave intervention significantly improved motor and exploratory behaviors in the mTBI rats, alleviated anxiety-like symptoms, and accelerated the recovery of serum S100B and NEFL levels to control levels. Longitudinal comparisons based on

the LMM revealed that, compared to the mTBI group, the USW group exhibited a reversal in the time slope of FC changes, approaching that of the Ctrl group. Further FC×Group interaction analysis suggested that the behavioral improvements induced by ultrashort wave intervention were likely associated with the remodeling of specific neural circuits. Notably, the strength of connectivity between the Left Dentate Gyrus-Left Secondary Auditory Cortex Ventral Part showed a significant positive interaction with Slow Movement Time (β=26.258, p=0.0046), and a significant negative interaction with Immobility Time (β=-59.574, p=0.0195). Additionally, the strength of connectivity between the Left Basal Forebrain Region -- Left Granule Cell Level of the Cerebellum showed a negative interaction with Center Zone Distance (β=-333.396, p=0.0444). These results suggest that USW may regulate the relationship between specific functional connections and behavioral outcomes, thus facilitating neurobehavioral recovery following mTBI.

Conclusions: USW intervention may promote neurobehavioral recovery following mTBI by modulating the remodeling of brain network connectivity. This study provides experimental evidence supporting ultrashort wave as a potential non-invasive therapeutic strategy for mTBI, with implications from whole-brain network alterations to behavioral improvements.

Biography

Dr. Hengchao Ma is a PhD researcher in the Senior Department of Neurosurgery, Chinese PLA General Hospital, Beijing, China. Hengchao work centers on 11.7-T Ultra-High-Field (UHF) MRI in traumatic brain injury, leveraging quantitative and advanced neuroimaging to characterize microstructural and functional alterations and to develop imaging biomarkers that support mechanistic studies and clinical translation.

Jinfeng Shang[1*], Yiran Cui[2], Jingjuan Wang[1], Xin Liu[1]

[1]Beijing University of Chinese Medicine, China

[2]Beijing Hospital of Traditional Chinese Medicine, Capital Medical University, China

Chrysin targets ceruloplasmin to modulate zinc homeostasis in cerebral ischemia reperfusion injury

As a plant flavonoid compound, chrysin comes from *Scutellaria baicalensis* Georgi (Huangqin), which has a powerful immunoprotective effect to suppress immune inflammatory responses, and plays an outstanding role in the treatment of various brain diseases. This study aims to explore whether chrysin regulates zinc homeostasis by reducing oxidative stress and targeting Ceruloplasmin (CP), thereby alleviating Cerebral Ischemia-Reperfusion Injury (CIRI). In vivo and in vitro experiments were conducted on tMCAO model SD rats and OGD/R PC12 cell model. The therapeutic effects of chrysin on CIRI were evaluated by TTC staining, Nissl staining, neurological function scores, cell counting kit-8 and transmission electron microscope. To investigate the mechanism, TPEN was used to reduce the level of zinc ions in vivo, adeno-associated virus and siRNA were used to increase and decrease the expression of ceruloplasmin, respectively, and HIF-1α was overexpressed by dimethyloxallyl glycine. Molecular dynamics simulation, infrared spectroscopy, DARTS, and CETSA experiments were used to explore the binding characteristics of chrysin and CP in a zinc environment. RT-qPCR, immunohistochemistry, and Western blot were used to detect mRNA and protein expression levels.

Results: Chrysin significantly reduced the cerebral infarction rate and increased the survival rate of cells, while reducing zinc ion and oxidative levels in vivo. Mechanistically, both chrysin and the zinc chelator TPEN could reverse the promoting effect of zinc overload on CIRI, and overexpression of CP synergistically enhanced the therapeutic effect of chrysin on tMCAO rats, while CP knockdown exacerbated OGD/R injury. Zinc ions enhanced the affinity of chrysin and CP by stabilizing the coordination bonds of the chrysin-CP complex, which was confirmed by ex vivo experiments. In addition to directly binding zinc ions, chrysin directly binds CP, enhancing its activity and zinc-chelating

capacity, thereby reducing zinc overload. Concurrently, chrysin suppresses HIF-1α expression, lowering CP levels and restoring physiological balance. Additionally, HIF-1α is the direct upstream regulatory molecule of CP. This study is the first to demonstrate the molecular mechanism by which chrysin regulates zinc homeostasis and oxidative stress through targeting CP in CIRI.

Biography

Shang Jinfeng, a 2024 Ph.D. student in Traditional Chinese Medicine at Beijing University of Chinese Medicine, holds honors including "Jilin Virtue Youth," "Light of the Ordinary Nominee," and recognition among the National Scholarship's Top 100 Graduate Students (2022-2023). He received the National Scholarship, National Endeavor Scholarship, and First-Class Academic Scholarship. His research on cerebral ischemia, drug intervention, and renowned TCM experience inheritance includes participation in 4 national and 13 university projects, resulting in 6 SCI and 15 Chinese core journal publications (cumulative impact factor >50).

João Rafael De Oliveira Rocha da Silva

Connect Life Rehabilitation and Performance, Ubatuba, São Paulo, Brazil

Rehabilitation of patients with chronic musculoskeletal pain

Chronic pain is defined as persistent pain lasting more than three months, resulting in alterations in the brain's functional synaptic network and its gray matter dimensions. It causes hypersensitivity to nociceptive stimuli and increased excitability of the nodal stress mechanism, causing the individual to remain in a state of alert (hyperactivation of the nervous system) during normal daily activities, increasing their levels of stress, anxiety, and fear.

It can be classified as primary, with no known etiology, or secondary, pertinent to a specific pathological process and clinical diagnosis. Despite the great relevance of studies addressing the importance of physical exercise in these individuals, the understanding of correct assessment and prescription during clinical practice still does not seem very clear.

Scientific evidence is focused on establishing which exercise modality would be most appropriate; however, we observe a lack of information on recurrent neuro functional and biomechanical alterations in this population, which we can classify as a pathological pattern that should not be neglected. In previous studies, we observed that chronic pain directly impacts cardiac rehabilitation and adherence to physical exercise, significantly increasing disability and mortality in this population.

We also observed that individuals with chronic pain exhibit patterns of altered motor control and kinesiophobia. Since any type of chronic musculoskeletal pain can lead to functional disability, musculoskeletal pain in the lower and upper limbs directly impacts gait, work activities, and physical activities, including exercise and activities of daily living.

Previous studies have shown that the pathophysiology of chronic pain is responsible for alterations in neuromuscular reflexes, causing changes in motor control due to factors such as muscle inhibition, muscle strength deficits, altered body perception, and alterations in the sensorimotor system. Multimodal physiotherapy is a clinical approach where the physiotherapist uses more than one technique or therapeutic resource in the same session. This approach will address the treatment and prevention of chronic musculoskeletal pain, considering neuro functional improvement as well as the locomotor system, focusing on the assessment and functional improvement of these individuals.

It utilizes evidence-based practice, drawing from studies we have previously published highlighting the importance of manual therapy and the optimization of motor control through therapeutic exercise in pain management.

Keywords: Chronic Pain, Physiotherapy, Manual Therapy, Motor Control, Therapeutic Exercise.

Biography

Pt. João Rafael Rocha da Silva has been a clinical physiotherapist for over 15 years, with a postgraduate degree in rehabilitation applied to sport from the Department of Orthopedics and Traumatology at the Escola Paulista de Medicina CETE-UNIFESP, also having a postgraduate degree in Improvement in assessment and interdisciplinary treatment in Pain at the Hospital das Clínicas of the Faculty of Medicine of the University of São Paulo HC-FMUSP. Silva recently published five studies related to the treatment of Pain, which were presented at more than five international conferences and congresses. Scientific reviewer for international journals.

Katrīna Kate Būka[1*], Iveta Haritončenko[2]
[1]Riga Stradiņš University, Faculty of Medicine, Riga, Latvia
[2]Department of Neuroangiography, Riga East Clinical University Hospital (RAKUS), Riga, Latvia

Multilocular extracranial arterial dissection with cerebral infarction in a young woman

Background: Extracranial cervical artery dissection is a significant cause of stroke in young patients without traditional cardiovascular risk factors; however, its clinical presentation is often nonspecific, which may delay timely diagnosis. This paper presents a clinical case of a young woman with multilocal extracranial arterial dissection complicated by cerebral infarction.

Case Presentation: We describe the clinical case of a 30-year-old woman who was admitted with a two-week history of headache and pulsatile tinnitus in the left ear, as well as transient numbness on the left side of the body. Neuroimaging (CT, CTA, and digital cerebral angiography) revealed a cerebral infarction in the territory of the left middle cerebral artery and multiple extracranial arterial dissections: Occlusion of the left internal carotid artery due to dissection, dissection of the right internal carotid artery with approximately 50% luminal stenosis, and bilateral vertebral artery dissections. Neurological deficit was minimal (NIHSS 0, mRS 1).

Initial management was conservative, consisting of anticoagulant and antiplatelet therapy, due to a high perioperative risk. Follow-up CTA demonstrated recanalization of the left internal carotid artery with formation of a large dissecting aneurysm, prompting the decision for endovascular treatment. Percutaneous transluminal angioplasty with implantation of a flow-modulating stent was performed, resulting in restoration of the arterial lumen and near-occlusion of the dissecting aneurysm.

Conclusions: This case illustrates the complex course of multiple cervical artery dissections in a young patient with cerebral infarction. An individualized treatment strategy-beginning with conservative therapy and followed by selective endovascular intervention-allowed for a favorable clinical and radiological outcome. The case highlights the importance of a multidisciplinary approach and dynamic imaging follow-up in the management of such patients. Radiologists and neurologists should be aware of the possibility of arterial dissection in young individuals presenting with unexplained headache or tinnitus and consider appropriate diagnostic investigations and therapeutic strategies.

Biography

Katrīna Kate Būka is a fifth-year medical student at Riga Stradiņš University, Faculty of Medicine, Latvia. Būka has a strong academic interest in neurology and neurovascular diseases, with particular focus on cerebrovascular pathology and endovascular treatment strategies. Būka clinical training includes rotations in neurology and interventional neuroradiology at Riga East Clinical University Hospital. Būka is actively involved in clinical case analysis and research activities related to stroke in young patients. Through participation in scientific conferences, Būka aims to deepen her understanding of evidence-based neurovascular management and contribute to improved patient outcomes.

Klaudia Marek[1*], Joanna Piwnik[2], Elżbieta Miller[1]

[1]Department of Neurological Rehabilitation, Medical University of Lodz, Lodz, Poland

[2]Department of Biostatistics and Translational Medicine, Medical University of Lodz, Lodz, Poland

Spasticity management and the use of radial shock waves in post-stroke patients

Aims: Recent studies predict that the number of people diagnosed with stroke in the European Union will rise by 27% by 2047, driven by an aging population and improved survival rates. One of the most common complications following stroke is upper limb spasticity. As spasticity progresses, patients face challenges such as reduced joint mobility, stiffness, contractures, and pain, which significantly impair daily functioning and increase the burden on families and healthcare providers. Although various treatments exist, such as physical therapy, exercise therapy, pharmacological interventions, and surgical procedures, heir effectiveness remains limited and often associated with adverse effects. This study aims to assess the efficacy of radial shock wave therapy in improving upper limb motor function, reducing muscle tone, and alleviating pain in patients with post-stroke upper limb spasticity.

Material and Methods: This study examined 62 stroke patients with spasticity (MAS≥2) who completed the entire treatment regimen. The patients were randomized in a 1:1 ratio into two groups: A control group and a study group. Hospitalization, during which the patients received radial Shock Wave Therapy (rESWT), lasted six weeks. Treatment included kinesiotherapy, physical therapy, one-on-one sessions with a physical therapist, occupational therapy, psychological care, speech therapy, and medical care. Patients in the study group received an additional four rESWT treatments at weekly intervals. The results were measured using the FMA-UE, VAS, ADL, and MAS scales. Assessments were performed before the first rESWT treatment, immediately after the first treatment, and six days after the fourth treatment.

Results: In the study group, changes were observed in all analyzed parameters: FMA-UE ($p<.0001$), VAS ($p<.0116$), MAS ($p<.0001$), and ADL ($p<.0001$) at intervals.

- In the study group, an increase in FMA-UE and ADL values was noted already in the first week after the procedure compared to the baseline level, and after 5 weeks these parameters showed further statistically significant improvement compared to the results obtained one week after the intervention.
- In the case of MAS, a decrease in values was observed one week after the procedure compared to the pre-procedure status, while after 5 weeks this decrease was significantly greater than after the first week.
- The VAS FU and VAS for the wrist did not show significant changes in the first week after the procedure, but after 5 weeks a significant decrease was noted compared to the baseline values (VAS FU: $p=0.0200$/VAS wrist: $p=0.0268$).

Conclusion: The combination of intensive neurorehabilitation with rESWT in stroke patients with MAS≥2 muscle spasticity may be effective in reducing forearm flexor muscle spasticity, improving hand function, and increasing functional independence. Improvements in upper limb motor function and reductions in muscle tone occur independently of changes in pain perception among patients.

Biography

Klaudia Marek has done Master of Physiotherapy, PhD candidate at the Medical University of Lodz (Poland), academic and research staff member at the Department of Neurological Rehabilitation. Leader and supervisor of the Student Scientific Society of Neurorehabilitation, bringing together over 100 physiotherapy students. Marek research interests focus on neurological rehabilitation, particularly post-stroke recovery, robotics in neurorehabilitation, and spasticity management.

Linda K Friedman[1,2*], Richard J. Zeman[1,2], Hong Peng[1]

[1]NeuroInfusions LLC, United States

[2]Valley Stream, NY, 11581, Dept. Cell Biology and Anatomy, New York Medical College, Valhalla, NY, 10595, Unites States

Neuroprotective, anticonvulsant, and proconvulsant effects of cannabinoids following neurotrauma

Traumatic Brain (TBI) injuries result in profound local hypoperfusion, ischemia, chronic inflammation and refractory seizures (Post-Traumatic Epilepsy (PTE)), and restrict drug delivery to the site of impact so that peripheral treatment alone would have limited access to the site of injury during the most critical phases of neurotrauma. Cannabidiol (CBD), the major non-psychotropic cannabinoid, has anti-convulsant, anti-inflammatory, anti-nociceptive, antioxidant, and immuno-suppressive properties not fully understood. In pre-juvenile rats, microinjection of CBD attenuated Kainate (KA)-induced seizures to a greater extent than intraperitoneal injection, indicating that local drug administration was more effective. In adult rats after experimental TBI, our modified CBD-infused implant applied extradural with oil injection supplementation restored vestibulomotor and cognitive functions compared to systemic treatment alone. We questioned whether the CBD or the low concentrations of THC in the extract was responsible for behavioral and cellular recovery. We hypothesized that an optimal ratio of Cannabidiol (CBD) to Tetrahydrocannabinol (THC) is required to protect against neuropathological consequences following TBI greater than either substance alone. Varied CBD:THC extract concentrations were compared with hemp CBD lacking THC (CBD0). Neurons, glia, and Parvalbumin Interneurons (PV-INs) were evaluated. Weight loss was observed following high doses of THC dominant cannabis, THC100:1. Neuroscores and vestibulomotor performance were restored optimally with CBD:THC300:1-10:1. However, THC dominant treatments resulted in early onset to spontaneous seizures post-TBI. In a non-reward T-maze, the CBD10:1 group had the highest alternation rates; TBI+vehicle, CBD0, CBD1:1, and THC100:1 treatment groups had the lowest. The novel object recognition memory task showed CBD300:1 treated animals had the best performance, while TBI or THC100:1 treated groups had the worst. The Forced Swim Test (FST) showed immobility time was highest after TBI and lowest after THC100:1 treatment. The Elevated Plus Maze (EPM) revealed the CBD0 group spent

the most time in closed arms. Both tests indicate that reduced anxiety was THC dependent. All combinations resulted in reduced injury but CBD10:1 and THC20:1 gave the most protection and THC100:1 the least. Reduced anxiety level was THC dependent but higher doses were pro-convulsant cautioning THC dosing. Reduced GFAP labeling was highest with CBD dominant cannabis supporting its neuroprotective role against inflammation. Rescue of diminished bilateral PV-INs was observed within the hippocampus and medial Prefrontal Cortex (mPFC) with CBD dominant treatment (CBD300, CBD0) supporting their anticonvulsant effect. Loss of PV-INs with THC dominant treatment supports their proconvulsant effect. Thus, CBD and THC have different beneficial therapeutic effects indicating an optimal concentration ratio is critical for optimal neuropathological therapeutics.

Keywords: Cannabinoids, Neurotrauma, Neuroprotection, Post-Traumatic Epilepsy.

Annotation: Traumatic Brain (TBI) injuries result in profound local hypoperfusion, ischemia, chronic inflammation, and metabolic dysregulation which restrict drug delivery to the site of impact so that peripheral treatment alone would have limited access to the site of injury during the most critical phases of neurotrauma. Cannabidiol (CBD), the major non-psychotropic cannabinoid, has anti-convulsant, anti-inflammatory, anti-nociceptive, antioxidant, and immuno-suppressive properties that may treat both primary and secondary injury associated with TBI. Recently we found that direct delivery to the wound site and the ratio of CBD to Tetrahydrocannabidol (THC) (CBD:THC) may be critical for motor and cognitive recovery. Determining the optimal ratio of CBD:THC will allow clinicians to treat patients with optimal cannabinoid combinations to combat excitotoxic cascades that quickly follow TBI and prevent the onset to PTE.

Biography

Dr. Linda K Friedman is a Associate Professor of Neuroscience, New York Medical College, grew up in Massachusetts and received her Bachelor's degree from the University of Louisville in 1980. She obtained her MA and PhD degrees from the City University of New York, Mount Sinai in 1988. Friedman was a postdoc at Downstate Medical Center, Brooklyn and at Albert Einstein College of Medicine in 1988-1994 and was appointed Instructor Professor in 1994-1996. Friedman was appointed to Assistant and Associate Professor at NJ Neuroscience Institute Seton Hall University 1997-2012, then transferred to New York Medical College in 2012 to present. Friedman has focused on pre-clinical models to study neurotoxicity, delayed neurodegeneration, novel drug therapy, that may lead to neuroprotective and cognitive benefits. Friedman has many awards and honors: 1992, 1997, Annual American Epilepsy Society, Seattle, Junior Investigator Awardee; Red Ribbon Best Poster Award in Basic Research at the 114th Annual Osteopathic Association Convention. 2012, Platform Presentation Selection: Annual American Epilepsy Society Convention. Chosen in Top 10%; 2021: Platform Presentation Selection: "Chronic postnatal subconvulsive activity alters mood, cognition, seizure severity, NeuN antigenicity and polyphosphoinositide hydrolysis within limbic structures of juvenile rats". Friedman LK, Tenth and 11th International Meetings on Metabotropic Glutamate Receptors. AMPA and mGluR expression after chronic anticonvulsant treatment and epilepsy. Taormina Sicily-Italy.

Mandeep Kumar Arora*,
Tripti Bisht, Anglina Kisku
Department of Pharmacy, DIT University, Dehradun, Uttarakhand, India

Neuroprotective potential of a traditional Uttarakhand plant against Lambda-Cyhalothrin (LCT)-induced neural impairment: A pharmacological investigation

Neurodegenerative disorders are a group of diseases characterized by the progressive loss of structure or function of neurons, including brain cells, leading to impaired movement, cognition, and other neurological functions. Several factors contribute to the progression of neurodegeneration. Compelling evidence indicates that pesticide exposure is associated with an increased risk of neurodegenerative diseases, as many pesticides are identified as neurotoxic. Neurodegeneration was induced by Pyrethroid insecticides, particularly Lambda-Cyhalothrin (LCT) which are extensively used due to their high potency as insecticides and relatively low mammalian toxicity at recommended doses. However, accumulating evidence suggests that LCT exposure induces neurotoxicity through mechanisms involving oxidative stress, mitochondrial dysfunction, neuroinflammation, and neuronal apoptosis. Crude extract of *Pyracantha crenulata* was prepared to mitigate the effect of pesticide using cold maceration method. To evaluate the neuroprotective efficacy of PCEA through a combination of behavioural, biochemical, molecular, and histopathological analyses. Behavioural assessments, including locomotor activity, cognitive performance (Morris water maze), and recognition assessment, were conducted to determine functional recovery following PCEA treatment. Biochemical estimations focused on key markers of oxidative stress (Malondialdehyde (MDA), reduced Glutathione (GSH)), cholinergic function (acetylcholinesterase activity), and nitrosative stress (nitric oxide levels). Furthermore, the modulatory effects of PCEA on neuroinflammatory cytokines (TNF-α, IL-6) and Neurotrophic Factors (BDNF) were assessed to elucidate its anti-inflammatory and neurorestorative properties. This study represents the first comprehensive investigation into the neuroprotective potential of *P. crenulata* against LCT-induced neurotoxicity, using acute (6 days; 4mg/kg, 8mg/

kg) and chronic (28 days; 2mg/kg) exposure models. Our findings not only establish dose-dependent neurotoxicity thresholds for LCT but also highlight *P. crenulata's* ability to mitigate oxidative damage, suppress neuroinflammation, and preserve neuronal architecture.

Biography

Mandeep Kumar Arora is an accomplished academic and researcher with over 20 years of experience, including 15 years in academia and research, along with international industry exposure in the UK and India. Arora specializes in neuropharmacology, vascular pharmacology, and pharmacovigilance, focusing on oxidative stress, neuroinflammation, and mitochondrial dysfunction in neurological disorders. Arora credentials include 45+ publications in high-impact journals, 150+ cumulative impact factor, 1500+ citations, h-index of 16, and 21 Indian patents. Arora has led funded projects, supervised Ph.D. and M. Pharm scholars, and earned prestigious awards including "Teacher of the Year 2021" and the Scientific Excellence Award, demonstrating outstanding leadership and innovation.

Milton Cesar Rodrigues Medeiros[1*], Marilia Mancebo[2], Milton Takeshi Medeiros[2]

[1]Santa Casa de Arapongas, Full Member of Brazilian Academy of Neurology, Brazil

[2]UNIFAA, RJ, Brazil

Transcranial Direct Current Stimulation (tDCS) for chronic migraine: A randomized clinical trial with 20 patients

Background: Chronic migraine is a disabling and often treatment-resistant neurological disorder. Transcranial Direct Current Stimulation (tDCS) is a non-invasive neuromodulation technique with emerging evidence for pain management. To assess the efficacy and safety of anodal tDCS applied over the primary motor cortex (M1) in reducing pain intensity and migraine frequency in patients with chronic migraine, compared to sham stimulation. Twenty adults (12 women and 8 men; age range 27-46) diagnosed with chronic migraine (ICHD-3) were randomized into two groups: Active tDCS (n=10) and sham (n=10). The active group received 2mA of anodal tDCS over M1 (C3) for 20 minutes/day, 5 days/week for 4 weeks. The sham group received stimulation for only 30 seconds. Pain intensity (Visual Analog Scale) and monthly migraine frequency were assessed at baseline, post-treatment, and 5 weeks follow-up. The active tDCS group showed a mean pain intensity and migrane frequency reduction. No adverse events were reported. Anodal tDCS over M1 significantly reduced pain intensity and migraine frequency in chronic migraine patients, with effects sustained for five weeks post-treatment. tDCS was safe, well-tolerated, and may represent a valuable non-pharmacological intervention.

Introduction: Chronic migraine is a highly disabling neurological condition characterized by the presence of headache on at least 15 days per month for more than three months, with at least eight days having features of migraine without aura. It represents the most severe end of the migraine spectrum and affects approximately 1-2% of the global population, disproportionately impacting women. The pathophysiology of chronic migrane is multifactorial, involving both peripheral and central mechanisms. A pivotal role is played by dysfunction in the trigeminovascular system, which leads to the release of Calcitonin Gene-Related Peptide (CGRP), a potent vasodilator and pro-inflammatory neuropeptide. This cascade promotes

neurogenic inflammation, peripheral sensitization, and activation of second-order neurons in the trigeminocervical complex. Over time, repeated migraine attacks can induce maladaptive neuroplastic changes, including central sensitization and decreased efficacy of descending pain modulatory systems, especially involving the periaqueductal gray and thalamus. Structural and functional neuroimaging studies have consistently demonstrated alterations in key brain regions in patients with chronic migrane, including increased iron accumulation in pain-modulating structures and abnormal functional connectivity in the default mode and salience networks. These findings support the hypothesis that chronic migrane is not merely an extension of episodic migraine but a distinct disorder with unique central nervous system alterations. Chronic migrane frequently coexists with psychiatric comorbidities such as depression, anxiety, and sleep disturbances, as well as metabolic conditions like obesity and insulin resistance. These comorbidities not only complicate diagnosis and management but also contribute to disease chronification. Moreover, medication overuse headache arising from the excessive use of analgesics, triptans, or combination drugs is present in nearly half of patients with chronic migrane and further reinforces the vicious cycle of pain and medication dependency.

It presents significant disability and burden and is often refractory to both acute and preventive pharmacological strategies. Neuromodulation techniques, including tDCS, are promising alternatives or adjuncts to pharmacotherapy, particularly for patients with medication overuse, intolerance, or inadequate response.

Methods: This was a double-blind, randomized, sham-controlled trial involving 20 adults, age range 27-46 (12 women, 8 men) with chronic migraine based on ICHD-3 criteria. All patients signed the informed consente form. Participants were randomly assigned to receive either active or sham tDCS. There were 10 people in each group (active and sham), matched by age. The active protocol involved anodal stimulation at 2mA over the left primary motor cortex (C3 when we use the 10-20 system of the electroencephalogram) or M1, for 20 minutes daily, five days a week, for four weeks. The sham protocol mimicked the procedure but with current discontinued after 30 seconds. Primary outcomes included changes in pain intensity, assessed by the Visual Analog Scale (VAS), and monthly migraine frequency. Assessments were conducted at baseline, immediately post-intervention (four weeks) and after 5-week of the last stimulation.

Results: At week 4, the active group exhibited a mean VAS pain reduction of 76.6% (SD=5.8) compared to 8.5% (SD=9.1) in the sham group ($p<0.001$). The migraine attack frequency dropped by 49.8% (SD=8.1) in the active group, compared to 11.0% (SD=14.9) in the sham group ($p<0.001$). These benefits persisted at the 5-week follow-up. No adverse effects were reported in either group.

Discussion: The present findings demonstrate that anodal Transcranial Direct Current Stimulation (tDCS) applied to the primary motor cortex (M1) significantly reduces both pain intensity and monthly migraine frequency in patients with chronic migraine. The reduction in mean Visual Analog Scale (VAS) scores by 76.6% and attack frequency by nearly 50% is not only statistically significant but clinically meaningful, especially considering the typically refractory nature of chronic headache.

These results are consistent with prior studies exploring neuromodulation in migraine. While earlier work by Antal et al. (2011) applied cathodal tDCS over M1 and observed reductions in migraine days and pain intensity, our study suggests that anodal stimulation which increases cortical excitability, may be equally or even more efficacious, particularly when applied with a higher intensity (2mA) and longer duration (20 minutes per session). This polarity-dependent variability underscores the complexity of tDCS mechanisms and supports the growing view that individualized montage selection may be necessary for optimal outcomes. The robust and sustained effects observed at the five week follow-up suggest enduring neuroplastic changes in cortical and subcortical pain networks. M1 stimulation is hypothesized to modulate the activity of thalamocortical pathways and the descending pain modulatory system, particularly through enhanced connectivity with the periaqueductal gray, anterior cingulate cortex, and dorsolateral prefrontal cortex. These mechanisms may underpin both the antinociceptive and frequency-modulating effects observed.

Importantly, the intervention was well tolerated, with no adverse events reported, consistent with the established safety profile of tDCS in neurological populations. This aspect is crucial, particularly for patients with contraindications to pharmacotherapy, history of medication overuse headache, or intolerance to preventive agents such as topiramate or β-blockers. In this regard, tDCS offers a non-pharmacological, low-cost, and portable option for chronic migraine management, which may be integrated into broader multidisciplinary care programs.

Our findings also have implications for the biopsychosocial model of migraine, especially considering that many patients suffer from comorbid anxiety, depression, and sleep disturbances. tDCS may potentially influence these domains by modulating large-scale brain networks implicated in mood and arousal regulation, though this hypothesis warrants targeted investigation.

Despite the promising outcomes, several limitations should be acknowledged. The sample size, while sufficient for preliminary efficacy analysis, was small and may limit generalizability. Additionally, the absence of neurophysiological or imaging biomarkers precludes definitive conclusions about the mechanistic underpinnings of the observed changes. Future trials should consider incorporating measures such as EEG, fMRI, or quantitative sensory testing to explore how tDCS modifies pain-related brain activity and connectivity.

Another area for future research involves optimizing stimulation parameters, such as electrode placement (e.g., bilateral M1 vs. M1-DLPFC montages), session frequency, and duration of treatment to achieve durable remission. Moreover, combination therapies involving tDCS and behavioral interventions like cognitive behavioral therapy or mindfulness-based stress reduction may synergistically target both neurophysiological and psychological contributors to chronic migraine.

Conclusion: This study adds to the growing body of evidence supporting the use of tDCS as an effective, safe, and non-invasive treatment for chronic migraine. Anodal stimulation of the primary motor cortex resulted in significant reductions in pain intensity and migraine frequency, with benefits sustained at follow-up. These findings suggest that tDCS may facilitate neuroplastic remodeling of pain-related neural circuits and serve as a valuable

adjunct or alternative to pharmacologic treatments. Larger, mechanistically informed trials are warranted to validate these outcomes and optimize stimulation protocols for broader clinical application.

Keywords: Chronic Migraine, tDCS, Neuromodulation, Pain Management, Non-invasive Stimulation, Clinical Trial.

Biography

Dr. Milton C. R. Medeiros is a titular member of the Brazilian Academy of Neurology. Graduated in medicine from the State University of Londrina in 1994 (UEL). Medical residency in neurology at UEL, graduated as specialist in 1997. Member of the Brazilian Academy of Neurology since 2002. Member of the headache and pain scientific departments at the Brazilian Academy of Neurology. Dr. Milton is also a member of the scientific department of the cognitive neurology and aging. Dr. Milton is a writer. Dr. Milton latest book is "Fuja do Alzheimer agora mesmo", publisher Viseu, still an exclusive edition in Portuguese.

Min Zhang[1,2,4,5*], Yu-Ping Zhang[1,2,3], Ling-Yan Zhang[1,2,4,5], Xi-Yun Liu[1,2], Shi-Chao Li[1,2,6], Xiao-Yu Wang[1,2], Xiao-Peng Ren[1,2,4,5], Xin-Yi Zhou[1,2], Cheng-Shuai Zhao[1,2]

[1]Department of Pathophysiology, Hebei Medical University, 361 Zhongshan East Road, Shijiazhuang, 050017, Hebei, People's Republic of China

[2]Hebei Key Laboratory of Critical Disease Mechanism and Intervention, 361 Zhongshan East Road, Shijiazhuang, 050017, Hebei, People's Republic of China

[3]Department of Pathophysiology, Hebei North University, 11 Zuanshi South Road, Zhangjiakou, 075000, Hebei, People's Republic of China

[4]Hebei Key Laboratory of Forensic Medicine, Hebei Collaborative Innovation Center of Forensic Medical Molecular Identification, Shijiazhuang, 050017, Hebei, People's Republic of China

[5]Laboratory of Neural and Vascular Biology, Ministry of Education, Hebei Medical University, Shijiazhuang, 050017, Hebei, People's Republic of China

[6]Laboratory Diagnosis Center for Infectious Disease, The Second Hospital of Hebei Medical University, Shijiazhuang, Hebei 050000, People's Republic of China

Cerebral Ischemic Preconditioning (CIP) promotes brain ischemic tolerance through N6-methyladenosine suppression of ACSL4-induced ferroptosis

Cerebral Ischemic Preconditioning (CIP), a phenomenon in which a brief, sub-lethal ischemic episode confers robust protection against a subsequent severe stroke, is a powerful intrinsic mechanism for enhancing brain ischemic tolerance. Ferroptosis is now recognized as a critical pathogenic component in ischemic brain injury. Concurrently, the dynamic N^6-methyladenosine (m^6A) RNA modification has emerged as a crucial epitranscriptomic regulator of diverse cellular processes, including cell death pathways. The potential role of m^6A signaling in modulating ferroptosis within the context of CIP-induced neuroprotection remains largely unexplored. This study aimed to investigate the hypothesis that m^6A-dependent mechanisms mediate the anti-ferroptotic effects of CIP. Utilizing complementary in vivo (mouse transient middle cerebral artery occlusion, MCAO) and in vitro (oxygen-glucose deprivation, OGD in HT22 neuronal cells) models, we found that both CIP and pharmacological ferroptosis inhibition significantly attenuated ischemia-induced neuronal ferroptosis. We identified acyl-CoA synthetase long-chain family member 4 (ACSL4) as a central player. Similarly, in HT22 cells, mild OGD Preconditioning (OGDP) attenuated the OGD-triggered upregulation of ACSL4. Crucially, overexpression of ACSL4 abolished the protective effect of

OGDP against ferroptosis, confirming its essential role. Mechanistically, we discovered that OGD downregulated the m^{6}A "eraser" protein FTO (fat mass and obesity-associated protein) while upregulating the m^{6}A "reader" proteins IGF2BP1 and IGF2BP3. This shift in the m^{6}A regulatory machinery enhanced the stability of ACSL4 mRNA, promoting its expression and subsequent ferroptosis. OGDP reversed this cascade by restoring FTO expression, reducing IGF2BP1/3 levels, and thereby accelerating m^{6}A-dependent decay of ACSL4 mRNA. Collectively, our results demonstrate that CIP attenuates ACSL4-mediated ferroptosis via the m^{6}A-FTO-IGF2BP1/3 axis to establish ischemic tolerance, revealing a novel and promising epitranscriptomic target for therapeutic intervention in ischemic stroke.

Biography

Min Zhang, born in 1968, PhD, Professor and Doctoral Supervisor. Zhang have been engaged in research on the induction and mechanisms of cerebral ischemic tolerance since 2005. Zhang have presided over several projects from the National Natural Science Foundation of China and provincial natural science foundations. Zhang have published more than 50 academic papers, including 18 SCI papers as the first author or corresponding author. Zhang papers have been cited 49 times at most, with 41 non-self-citations. Zhang have received five Hebei Provincial Science and Technology Progress Awards, including one first prize in 2025, ranking first.

Mustafa A Khan
Sevaro Health Inc., United States

Resolution of the CSF-SBP phase paradox: A mathematical framework for cerebral pressure dynamics

Objective: To resolve the clinical paradox where CSF pressure peaks before systolic blood pressure yet both drop to zero simultaneously during cardiac arrest.

Background: CSF pressure demonstrates 50-100ms phase lead over systolic blood pressure during normal cardiac cycles, yet both pressures zero simultaneously during cardiac arrest. This paradox lacks theoretical explanation. Previous models invoking temporal delays explain phase relationships but not simultaneous collapse, while direct coupling models explain zeroing but not phase lead.

Design/Methods: We developed a geometric energy conservation framework incorporating the Monro-Kellie doctrine. Through energy partitioning among arterial, transverse, and CSF components with normalization constraints, we derived pressure relationships. Geometric factors $K=(PT/PL)(VT/V_C)$ and $k=(VL/VC)[1+mk(Pt/PL)]$ emerge from fundamental conservation principles.

Results: The linear relationship $P_C=P_L(K-k)$ resolves both paradoxical observations. Time derivatives show $dP_C/dt=(dPL/dt)(K-k)+PL[d(K-k)/dt]$, where the second term creates phase lead through geometric factor evolution. During cardiac arrest, as $PL\rightarrow0$, then $PC\rightarrow0$ simultaneously, explaining instantaneous coupling. The framework predicts measurable coupling constants and compliance-related changes.

Conclusions: This geometric energy conservation framework provides the first unified resolution of the CSF-SBP phase paradox. The linear coupling relationship explains both phase lead and simultaneous zeroing through geometric factors rather than temporal delays. Clinical applications include non-invasive ICP estimation and personalized pressure management based on individual coupling constants.

Biography

Dr. Mustafa Khan is a board-certified stroke neurologist at Sevaro Health Inc. and the developer of Prince Wave Field Physics, a theoretical framework grounded in the de Broglie relation $\lambda p=h$ applied to cerebral hemodynamics. Khan work integrates wave-mechanical principles with clinical neurology, offering novel explanations for cerebral autoregulatory failure, perfusion collapse, and precision stroke management, including a mechanistic reinterpretation of the Cushing reflex as diastolic collapse.

Mustafa A Khan
Sevaro Health Inc., United States

Mathematical proof reveals Cushing's triad as diastolic collapse: The 2:1 ratio as a precision diagnostic marker and novel therapeutic target

Objective: To derive the precise hemodynamic mechanism of cushing's triad using fundamental physics principles and validate quantitative predictions against clinical data.

Background: Cushing's triad has been misunderstood as protective hypertension for over a century. Using de Broglie principles and energy conservation, we hypothesized that the triad represents forced hemo-dynamic redistribution, not a protective reflex.

Design/Methods: Mathematical analysis of cerebral hemodynamics under ICP constraints. Energy conservation equations ($\delta P_L + \delta P_t \approx 0$) were applied to derive pressure relationships. Clinical validation used retrospective analysis of blood pressure patterns in TBI patients with Cushing's triad versus IIH patients with elevated ICP.

Results: Mathematical derivation reveals $\delta SBP \approx -2\delta DBP$ with $\delta MAP \approx 0$, predicting systolic pressure rises exactly twice the diastolic drop while MAP remains constant. Clinical validation (n=400+) confirmed 99% of cases show this 2:1 ratio (e.g., 120/80→160/60). Five mathematical conditions must be met: ICP dominance ($\delta P_C \approx \delta P_T$), pressure opposition ($\delta P_t = -\delta P_L$), transverse flow failure ($\delta m_t/\delta m_L \to 0$), volume collapse ($\delta V_t/\delta V_L \to 0$), and tissue injury. This explains why TBI at ICP 30-35mmHg triggers the triad while IIH at 45mmHg does not. The triad originates from the compressed normal hemisphere, not injured tissue. Terminal progression shows coronary hypoperfusion at DBP<50, leading to cardiac arrest.

Conclusions: Cushing's triad represents mathematically-forced diastolic collapse with compensatory systolic rise. The 2:1 ratio provides an objective diagnostic trigger for immediate EVD placement. Understanding diastolic hypotension as primary pathology opens novel therapeutic targets: Agents maintaining DBP without raising ICP, perforator-specific vasodilators, and ICP-MAP constraint breakers. This quantitative framework transforms a mysterious reflex into targetable pathophysiology with immediate applications for automated detection systems and pharmaceutical development.

Biography

Dr. Mustafa Khan is a board-certified stroke neurologist at Sevaro Health Inc. and the developer of Prince Wave Field Physics, a theoretical framework grounded in the de Broglie relation $\lambda p=h$ applied to cerebral hemodynamics. Khan work integrates wave-mechanical principles with clinical neurology, offering novel explanations for cerebral autoregulatory failure, perfusion collapse, and precision stroke management, including a mechanistic reinterpretation of the Cushing reflex as diastolic collapse.

Dr. Ragni Kumari
Assistant Professor & Convenor (Head), Department of Optometry, Faculty of Paramedical Sciences,
Uttar Pradesh University of Medical Sciences (UPUMS),
Saifai, Etawah-206130, Uttar Pradesh, India

The impact of acquired brain injuries on vision: Patterns, assessment, and rehabilitation

Acquired Brain Injuries (ABIs), including stroke and Traumatic Brain Injuries (TBIs), frequently result in visual system impairments ranging from basic sensory deficits to complex perceptual dysfunctions, significantly affecting independence, safety, and quality of life. This narrative review synthesizes current knowledge on the types, mechanisms, assessment, and management of vision impairments associated with ABI and highlights existing gaps in care and research. A comprehensive literature search was conducted in PubMed, Scopus, and Google Scholar, including studies addressing post-ABI visual deficits, their pathophysiology, rehabilitation approaches, and outcomes in both adult and paediatric populations. Visual impairments following ABI encompass visual field deficits (e.g., homonymous hemianopia), oculomotor dysfunctions, cortical visual impairment, and higher-order visual perceptual disorders such as visual neglect and agnosias. Assessment requires interdisciplinary collaboration using perimetry, visual evoked potentials, neuroimaging, and cognitive testing. Rehabilitation strategies include compensatory training, prism adaptation, vision therapy, and assistive technologies, though the evidence base remains variable and standardized care pathways are limited. Early screening, interdisciplinary management, and personalized rehabilitation are crucial for optimizing recovery, and further research is needed to establish evidence-based interventions and integrate visual care into comprehensive neurorehabilitation services.

Keywords: Acquired Brain Injury, Visual Impairment, Cortical Visual Impairment, Visual Field Loss, Traumatic Brain Injury, Stroke Rehabilitation, Neuro-Ophthalmology.

Biography

Dr. Ragni Kumari is an Assistant Professor and Head of the Department of Optometry at Uttar Pradesh University of Medical Sciences (UPUMS), with over 17 years of experience in the fields of optometry, public health, clinical education, and community outreach. Dr. Kumari holds a Ph.D. in Public Health and is a Certified Professional in Medical Health Professions Education. Dr. Kumari has authored more than 135 research papers and serves as a reviewer and editorial board member for several prestigious journals, including PLOS Global Public Health, Cureus, Asian Journal of Pediatric Research, as well as publications under BPI, RPC, and Science Domain International. In addition to her academic roles, Dr. Kumari is the Managing Director of Diya Foundation, where Dr. Kumari leads initiatives aimed at providing eye care services to underserved communities. Dr. Kumari tireless work in public health has earned her numerous accolades, including the International Women Researcher Award (2021), the Most Determined Researcher Award (2025) and research excellence award (2026). Dr. Kumari was also honored with the Best Faculty Award on the foundation day of UPUMS and the Best Speaker Award at a conference focused on underprivileged children. Dr. Kumari's research and professional focus lie in bridging clinical practice with academic excellence and advancing public health initiatives, particularly in the field of eye care, within India.

Rahul Hajare
Sandip University, School of Pharmaceutical Sciences, India

A computational approach to evaluate the impact of chemical pathways on brain activity on a broad scale

One of the most important problems in modern neuroscience is determining how pharmacological substances affect brain function. There is currently no methodical way to assess these effects in whole-brain models, which usually concentrate on macroscopic phenomena, whereas pharmaceutical interventions function at the molecular level. Here we tackle this problem by introducing a computational method for brain simulations based on mean-field models with biophysical foundations that incorporate membrane conductances and synaptic receptors, as seen in the anesthetic example. We demonstrate that, as seen experimentally in profound anesthesia, anesthetics that target GABAA and NMDA receptors can cause the brain to change to generalized slow-wave patterns. In order to support our ideas, we show that these slow-wave states are less sensitive to outside stimuli and have functional connectivity that is limited by anatomical connection, which is consistent with observations made in anesthetic states in other animals. Our method offers a strong framework for comprehending how pharmacological actions at the molecular level affect whole-brain dynamics since it is based on mean-field models that take molecular reality into account.

Biography

Dr. Rahul Hajare received his B.Pharm. degree in 2002 from Amravati University in India, where he studied pharmaceutical chemistry. After that, he joined the research team at Nagpur University's Institute of Pharmaceutical Research, Education, and Quality Assurance. In 2012, he graduated from Vinayaka Mission University with a PhD after completing two years of postdoctoral research at the National AIDS Research Institute (ICMR) in Pune, India, under the supervision of renowned and highly respected scientist Dr. Ramesh Paranjape. In Nashik, Dr. Rahul is named a professor at Sandip University, School of Pharmaceutical Science. In SCI (E) journals, he has over 70 research publications to his credit. The Scholar Hindu University of America, Florida, presents Dr. Rahul Hajare with an award.

Dr. Raul Villamarin Rodriguez
Vice President, Woxsen University, Hyderabad, India

AI-assisted collective analysis of neurological biomarkers: A study for enhanced diagnostic accuracy in neurodegenerative disorders

Neuromorphic collective intelligence system for enhanced neurological diagnostic accuracy.

Background: Traditional neurological diagnosis relies heavily on individual clinician expertise, leading to significant variability in diagnostic accuracy across institutions and specialties. The complexity of neurological disorders, particularly neurodegenerative diseases, often requires integration of diverse clinical perspectives and computational analysis of multimodal biomarker data that exceeds individual cognitive processing capabilities.

Objective: To develop and validate a Neuromorphic Collective Intelligence System (NCIS) that integrates human neurological expertise with artificial intelligence through advanced brain-computer interfaces for improved diagnostic accuracy in complex neurological disorders.

Approach: The NCIS framework combines non-invasive neural interfacing using high-density EEG arrays with federated learning algorithms that preserve individual cognitive boundaries while enabling collective diagnostic reasoning. The system employs a linguistic-neural translation layer that maps neural activity patterns from participating neurologists to semantic representations in the artificial system's knowledge base. This enables seamless integration of human intuitive pattern recognition with computational analysis of neuroimaging, electrophysiology, and biomarker data.

The Architecture Maintains Three Operational Layers: Local cognitive models that adapt to individual neurologist expertise patterns, task-specific integration models for different diagnostic challenges, and a global coordination layer managing information flow while preventing cognitive overload. Fairness-verified algorithmic governance ensures equitable weighting of diverse specialist perspectives based on subspecialty expertise and case complexity.

Anticipated Outcomes: NCIS is expected to significantly improve diagnostic accuracy for challenging neurological cases, particularly in differential diagnosis of neurodegenerative disorders and early-stage disease detection. The system should reduce diagnostic variability between institutions and provide enhanced diagnostic support for community hospitals with limited subspecialty access.

Biography

Dr. Raul Villamarin Rodriguez is a distinguished cognitive technologist and Vice President of Woxsen University, where he leads pioneering research at the intersection of AI, cognitive science, and behavioral intelligence. Dr. Rodriguez's work focuses on developing human-centric systems that enhance decision-making, personalization, and ethical AI deployment. Dr. Villamarin is the architect of several patented frameworks, including the Behavioral GPS™, Unified Healthcare Intelligence System (UHIS), and the N-PACE academic engine-deployed across education, healthcare, aviation, and urban ecosystems. Dr. Rodriguez's research is deeply interdisciplinary, combining neurocognitive modeling, predictive analytics, and ethical system design. Dr. Rodriguez's has published over 190 academic works, including books, peer-reviewed journals, and policy papers, and is an invited speaker at premier global platforms such as Web Summit, ICAO, World Summit AI, and LEAP. Dr. Rodriguez's serves on international boards for higher education, technology ethics, and AI governance, advocating for sustainable, inclusive, and culturally intelligent innovation across the digital economy.

Rishabh Singh[1*], Rishi Sharma[2], Chandrasekhar Singha[3], Ininaki Lyngdoh[4]

[1]Paediatric Intensive Care Unit Fellow, Madhukar Rainbow Children's Hospital, New Delhi, India
[2]Paediatric Neurology Consultant, Madhukar Rainbow Children's Hospital, New Delhi, India
[3]Head of Paediatric Intensive Care Unit, Madhukar Rainbow Children's Hospital, New Delhi, India
[4]Paediatric Intensive Care Unit Fellow, Madhukar Rainbow Children's Hospital, New Delhi, India

A rare presentation of transverse myelitis - transverse myelitis plus syndrome

Introduction: Transverse Myelitis is characterized by abrupt onset progressive weakness with sensory disturbances. Pathogenesis is usually implicated with direct viral invasion or vasculitis or an autoimmune response. TM Plus syndrome is association with another neuroinflammatory condition, like Multiple Sclerosis (MS), Neuromyelitis Optica Spectrum Disorder (NMOSD), or Acute Disseminated Encephalomyelitis (ADEM), presenting more severe symptoms affecting brain areas or widespread areas. We present a case report with an unusual paradigm of clinical signs and symptoms confirmed subsequently on radiological imaging and treated as Transverse myelitis Plus syndrome.

Methods: Case Report A 7 Years old child presented with complaints of Fever for 5 Days, Weakness of lower limbs X 2 days R>L in the form of high stepping gait s/o of an asymmetrical ascending motor weakness. On examination had GCS 15/15, with Single Breath Count 18. Power affected in Right Hip flexors 1/5 and left hip flexors 3/5, with power at knee Joint > 2/5 in both sides and Ankle Joint >2/5 with absent Knee Joint Reflexes, other deep tendon reflexes were preserved. No sensory loss or bladder bowel involvement

Investigations: CSF analysis showed no albumin- cytological dissociation, with a negative Autoimmune panel.

Nerve Conduction study showed F wave latency Bilaterally with absent H reflex, suggestive of Demyelinating motor polyneuropathy.

MRI BRAIN and WHOLE SPINE- Long segment Intramedullary T2 Hyperintensity in entire dorsal cord – Holocord myelitis – central cord likely Acute Transverse Myelitis with nerve root enhancement cauda equina

Result: In View of Asymmetrical nature of the disease MRI Whole Spine was done s/o Transverse Myelitis given pulse dose of Methyl prednisolone for 5 days at 30mg/kg/day then shifted to oral prednisolone at 2 mg /kg for two weeks followed by tapering.

Conclusion: Hence, Transverse Myelitis Plus Syndrome is a rare presentation which in our case had asymmetrical ascending motor weakness with no significant sensory involvement although NCS s/o demyelinating polyneuropathy.

Biography

Experienced and compassionate Paediatric Senior Resident with a holistic approach to patient care. Skilled in managing diverse cases involving multi-system involvement in intensive areas. Collaborative team player with a keen interest in research and experience in Behavioural disorders and Public Health. Proficient in optimizing patient care, managing multiple cases, and supervising new medical professionals. Strong background in working with minority and low-income populations.

Robert B. Slocum Ph.D
Narrative Medicine Program Coordinator, University of Kentucky HealthCare, Lexington, Kentucky, USA

Narrative medicine applications for neuro-oncology patient identity and quality of life

Brain cancer and its treatments bring a unique threat to the patient's identity and quality of life by challenging their essential identity in significant ways, possibly including impaired cognitive skills, loss of memory, reduced coordination, altered feeling states, and limited capacity for self-expression. These impairments may have a devastating and worsening impact on the patient as the cancer progresses and may be exacerbated by the side effects of treatment. This presentation will consider possible applications for Narrative Medicine (NM) to help these patients retain and rediscover self-identity. NM encourages patients to engage their stories of illness and treatment through guided conversations and emotional writing, with attentive listening at the heart of NM sessions. Patient experiences may be shared in conversation with a NM provider, or written in a patient journal and discussed at a later time. NM sessions were incorporated into the care of patients with brain tumors at the University of Kentucky Neuro-Oncology Program. NM made visible contributions for patients discussed in the case histories of this study. Understanding the patient's story is critical for evaluating the significance of impairments due to brain cancer and treatment relative to the patient's unique sense of self and quality of life. NM is at the nexus of clinical management and quality of life concerns for brain cancer patients. Insights from NM sessions may also help the treatment team as they assess patient needs, attitude, and abilities. This presentation will help the audience to understand the personal and unique impact of brain cancer on each neuro-oncology patient and the need for Narrative Medicine (NM) interventions and appropriate responses by all treatment team members to ameliorate the impact of these symptoms for improved patient quality of life.

Biography

Robert B. Slocum is the Narrative Medicine Program Coordinator at University of Kentucky HealthCare. He holds doctorates in law (Vanderbilt), ministry (University of the South), and theology (Marquette). Dr. Slocum has experience in pastoral ministry as well as academic teaching and administration. Dr. Slocum has taught undergraduate courses in religious studies and ethics. Dr. Slocum is an Assistant Professor (voluntary faculty, Internal Medicine) at the University of Kentucky College of Medicine (COM). Dr. Slocum teaches a fourth-year COM elective on the narrative basis for patient care and resilient practice. Dr. Slocum is a member of the Hospital Ethics Committee. Dr. Slocum is the author, editor, or co-editor of 14 books, including a journal of reflections. Dr. Slocum 36 articles have appeared in theological or medical journals and as book chapters, and he has made presentations at more than two dozen theological and medical conferences. Dr. Slocum has also published short fiction and poetry. Dr. Slocum is interested in the clinical application of narrative and the significance of narrative for identity formation. Dr. Slocum sees Narrative Medicine as a bridge between medical humanities and clinical practice.

Roberto Grau[1,2]*, Marcos Francisco[1,2]
[1]Kyojin S.A., Rosario, Argentina
[2]Juventas 4life S.L., Madrid, Spain

The gut-microbiota-brain connection: *Bacillus subtilis* DG101 regulates the aging-related insulin-IGFR1-FOXO route and the oxidative stress-related P38MAPK-NRF2 signaling to protect against Parkinson' and Alzheimer' diseases

Neurodegenerative diseases (Parkinson and Alzheimer's diseases) represent a pandemic that affects millions of humans around the globe, and for which there is no cure or effective treatments or medications.

The gut microbiota, the trillions of microorganisms inhabiting the intestinal lumen, connect with the central nervous system and affects its functioning. One member of the gut microbiota, the bacterium *Bacillus subtilis* Natto (BSN) DG101, is recognize for its beneficial (probiotic) effects on human health. Here, we present our recent investigations in the animal model *Caenorhabditis elegans* colonized by biofilm-forming BSN DG101 against the onset and progression of PD and AD. C. elegans is resistant to oxidative injury of dopaminergic neurons caused by treatment with the neurotoxin 6-hydroxydopamine (6-OHDA). Biofilm-forming BSN DG101-colonized *C. elegans* display dopamine-dependent behaviors indistinguishable from those of 6-OHDA-untreated worms colonized by gut commensal *E. coli* OP50. Life expectancy is longer and dopaminergic neurons are more strongly protected in biofilm-forming BSN DG101-colonized *C. elegans* dat-1p: CAT-2 worms, which exhibit early dopaminergic decay, than in biofilm-deficient BSN DG101- or *E. coli* OP50-colonized dat-1p: CAT-2 worms. Increases in healthy life expectancy and behavioral fitness are also observed in biofilm-forming BSN DG101-colonized worms overexpressing human alpha-synuclein and Parkin synthesis-deficient worms. The BSN DG101-controlled insulin/IGF-1 signaling (ILS), whose downregulation prevents aging-related PD, is not involved in protecting against oxidative damage-related PD. We demonstrate

that biofilm-forming BSN DG101 activates PMK-1 (p38 MAPK)/SKN-1 (Nrf2) signaling, which exerts antioxidant effects to protect *C. elegans* from oxidative injury-induced PD. Additionally, transgenic *C. elegans* model of AD (i.e., worms expressing the toxic forms of human Aβ-amyloid peptide) colonized by BSN DG101, but not worms colonized by gut commensal *E. coli* OP50, were fully protected and displayed cognitive responses and life expectancies indistinguishable from the behavior and life span of wild-type *C. elegans.* This research points to the important role of the microbial gut biofilm in neuroprotection and opens the possibility of novel strategies against PD, AD and other neurodegenerative diseases involving the gut – brain connectome and the human probiotic BSN DG101.

Biography

Dr. Roberto Grau is a Biochemist and PhD in Biological Sciences (Faculty of Biochemical and Pharmaceutical Sciences, National University of Rosario, UNR); International Master's Degree in Human Microbiota (Technological University of Spain); Professor of the Diploma Program in Microbiota and Nutrigenomics at the University of Mendoza; advanced studies in Philosophy (Faculty of Humanities and Arts, UNR). Postdoctoral Fellow in Molecular Biology of Signal Transduction in Bacteria (The Scripps Research Institute, Department of Molecular and Experimental Medicine, San Diego, California, USA). Former university professor and researcher, Latin American Fellow of the Pew Charitable Trust in Biological Sciences (San Francisco, USA); Fulbright International Scholar (Washington, DC, USA). With 43 years of experience in scientific research; recipient of more than 20 national and international awards for his teaching and scientific career; Director of over 40 scientific research projects funded by national and international government agencies and private companies. Dr. Grau has published over 50 original articles in prestigious journals and given over 150 presentations at scientific conferences, supervised dozens of postgraduate and doctoral theses, trained over 50 professionals in science, and been a visiting professor at more than 15 universities and research institutes worldwide (France, Spain, Germany, India, Japan, Brazil, Cuba, Chile, USA, UK). Dr. Grau has founded and/or catalyzed the creation of half a dozen biotechnology companies, including Kyojin S.A. in Argentina and Juventas 4life SL in Spain. Dr. Grau is a member of the EURECAT-Barcelona / Metrofood EU 2024-2025 program; the Health Start Madri+D 2025-2026 program; and the Madrid Science Park Foundation (Complutense and Autonomous Universities of Madrid) 2026. Current Research Topic: Role of Microbiota and Probiotics in Neuroprotection, Antitumor Therapies and Healthy Longevity.

Sara Adel Ahmed
Faculty of Medicine, Ain Shams University, Cairo, Egypt

Nutrition and neurodegeneration: The role of dietary interventions in Alzheimer's disease

Background: Chronic inflammation plays a critical role in most of challenging diseases, including rheumatoid arthritis, cancer, heart disease, diabetes, asthma, and even neurological diseases such as Alzheimer's and depression. Many studies proved how different dietary components can modulate key pathways to inflammation including sympathetic activity, oxidative stress, transcription factor Nuclear Factor Kappa B (NF-κB) activation, and proinflammatory cytokine production.

Diets that induce inflammation are high in refined starches, sugar, saturated and trans-fats, and low in omega-3 fatty acids, natural antioxidants and fiber from fruits, vegetables, and whole grains. Polyphenols, present in many dietary foods such as berries, green tea, and turmeric, exhibit antioxidant and anti-inflammatory properties by inhibiting NF-κB signaling. Probiotics and prebiotics help maintain gut integrity, reducing systemic inflammation linked to conditions such as IBD and metabolic syndrome.

High-fat meals especially trans-fat can stimulate low-grade endotoxemia, a rise in bacterial endotoxins, inflammatory antigens that are typically found circulating at low concentrations in blood. High-fat meals can also induce NF-κB activation in PBMCs. Different research highlights the anti-inflammatory potential of omega-3 fatty acids, found in fish oil and flaxseeds, which modulate inflammatory mediators such as prostaglandins and cytokines. Not only that but the n-6: n-3 ratios worked together to enhance inflammation beyond the contribution provided by either variable alone.

Neurological diseases represent a major global health burden, characterized by progressive impairment of cognitive, motor, and functional abilities. Among these, Alzheimer's disease is the most common cause of dementia, marked by progressive memory loss, cognitive decline, and behavioral changes. Its pathophysiology involves amyloid-β plaque deposition, tau protein hyperphosphorylation, neuroinflammation, and oxidative stress.

Emerging evidence highlights the critical role of nutrition in both the prevention and management of Alzheimer's disease. Dietary patterns rich in antioxidants, omega-3 fatty acids, vitamins (particularly B-complex, D, and E), and polyphenols have been associated with reduced neurodegeneration and improved cognitive function.

Biography

Sara Adel Ahmed is a Clinical nutrition consultant and Ass Prof in faculty of medicine Ain Shams University. ISSA certified and Barcelona diploma. Sara is an MD in pharmacology in faculty of medicine, teaching to undergraduates and postgraduates' pharmacology. Sara approached her believes in clinical nutrition through working with patients in hospitals in different specialties and applied how nutrition can integrate with all disciplines. Sara tried to raise the community awareness about nutrition through community webinars and events. Finally, Sara is also the founder and president of the International Medical Sports Nutrition Association which serves sports nutrition to support athletic performance and avoidance of injuries in sports.

Sathvik Loke[1*], Michael C. Jin[2] MD, MS
[1]Illinois Mathematics and Science Academy, USA
[2]Stanford University, USA

Advancing parkinson's disease management: From dopaminergic therapy to deep brain stimulation and beyond

Parkinson's Disease (PD) is a progressive Parkinson's disease characterized by degeneration of dopaminergic neurons in the substantia nigra, resulting in motor dysfunction, cognitive decline, and substantial long-term socioeconomic burden. Despite advances in symptomatic treatment, durable disease management remains limited by medication-related complications, delayed diagnosis, inequitable access to advanced therapies, and the absence of widely implemented disease-modifying interventions. This narrative review synthesizes current and emerging approaches to PD management through a translational framework integrating pharmacologic therapy, neuromodulation, biomarker development, and healthcare systems analysis.

Levodopa, administered in combination with carbidopa, remains the cornerstone of treatment but is constrained by a progressively narrowing therapeutic window, motor fluctuations, dyskinesias, and polypharmacy-related complications. Deep Brain Stimulation provides significant symptomatic improvement in appropriately selected patients, though long-term outcomes depend on multidisciplinary evaluation, postoperative optimization, and economic accessibility. Beyond symptom management, PD imposes substantial direct and indirect costs through caregiver burden, reduced productivity, psychiatric comorbidity, and long-term care needs.

Emerging diagnostic strategies, including blood-and cerebrospinal fluid–based biomarkers, advanced molecular imaging, and artificial intelligence–assisted neuroimaging, show promise for earlier detection and individualized intervention. By integrating biological mechanisms, clinical therapeutics, surgical innovation, diagnostic technologies, and

systems-level economic considerations, this review highlights the need for a comprehensive, multimodal, and patient-centered approach to improve long-term outcomes and support sustainable PD care.

Keywords: PD, Dopamine Replacement Therapy, Levodopa, Deep Brain Stimulation, Neurodegeneration, Biomarkers, Precision Medicine.

Biography

Sathvik is a rising junior at the Illinois Mathematics and Science Academy with aspirations of becoming a neurosurgeon. Sathvik is passionate about neuroscience, particularly studying the human brain and researching neurodegenerative diseases and behavioral sciences. Sathvik manuscript on the future of Parkinson's disease has been accepted for publication in the Journal of Research High School. Sathvik is also applies machine learning to address modern medical challenges and explore innovative healthcare solutions. Outside of academics, Sathvik enjoys watching football, playing tennis and soccer, and spending time with friends.

Sergey Suchkov[1-14]*, Eric J. Sorenson[19], Aleksandr Gabibov[15], Noel Rose[16,17], Holland Cheng[18]

[1]N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia
[2]China Hong Kong Innovation International Business Association, Hong Kong
[3]InMedStar, Russia
[4]New York Academy of Sciences, USA
[5]EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU, Belgium
[6]ISPM (International Society for Personalized Medicine), Tokyo, Japan
[7]PMC (Personalized Medicine Coalition), Washington, DC, USA
[8]AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland
[9]ACS (American Chemical Society), Washington, DC, USA
[10]AHA (American Heart Association), Dallas, TX, USA
[11]ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA
[12]ISER (International Society for Eye Research), Anchorage, AK, USA
[13]Secretary General, United Cultural Convention (UCC), Cambridge, UK
[14]The Russian Academy of Natural Sciences, Moscow, Russia
[15]Institute for Bioorganic Chemistry, Russian Academy of Sciences, Moscow, Russia
[16]Harvard Medical School, Boston, MA, USA
[17]Center for Clinical Autoimmunity, John Hopkins Medical School University, Baltimore, MD, USA
[18]The College of Biological Sciences, UC Davis, CA, USA
[19]Dept for Neurology, Mayo Clinic, Rochester, MN, USA

Antibody-proteases as translational tools of the next-step generation to be applied through biodesign-driven translational biotech in personalized and precision neurology practice

Multiple Sclerosis (MS) is a complex neurodegenerative pathology featuring with inflammation, demyelination, and neurodegeneration. Due to the heterogeneity of MS-related clinical and even subclinical subtypes, their diagnosis becomes challenging and the best treatment cannot be easily provided to patients and/or persons-at-risk.

Molecular biomarkers which are easily quantifiable, enable individual decisions and are an important step on the way to a personalized therapy, come from the areas of immunology due to the causal pathology-driven mechanisms and can excellently complement other disease characteristics. For a long time, numerous potential biomarkers that provide meaningful information related to the development of MS-related pathology, have been identified. But MS continues to be extremely unpredictable, with more unanswered questions than absolute certainties, the finding of it seems still a long way off. Therefore, to date, there remains no single reliable biomarker that can provide information on the prognosis of MS, distinguish between the different clinical courses of MS, and also predict responses to a specific form of treatment.

Abs against myelin basic protein/MBP endowing with proteolytic activity (Ab-proteases with functionality) are of great value to monitor demyelination to illustrate the evolution of multiple sclerosis (MS). Anti-MBP autoAbs from MS patients exhibited sequence-specific proteolytic cleavage of MBP, which, in turn, markedly differed between: (i) MS and healthy controls; (ii) Different clinical MS; (iii) EDSS scales of demyelination scores to correlate with the disability of MS patients to predict the transformation prior to changes of the clinical courses.

Ab-mediated proteolysis of MBP was shown to be sequence-specific whilst demonstrating five sites of preferential proteolysis to be located within the immunodominant regions of MBP and to fall inside into 5 sequences fixed. Some of the latter (with the highest encephalitogenic properties) were proved to act as a specific inducer of EAE and to be attacked by the MBP-targeted Ab-proteases in MS patients with the most severe (progradient) clinical courses. The other ones whilst being less immunogenic happened to be EAE inducers very rare but were shown to be attacked by Ab-proteases in MS patients with moderate (remission-type) clinical courses.

The activity of MBP-targeted Ab-proteases was first registered at the subclinical stages 1-2 years prior to the clinical manifestations of MS. About 24% of the direct MS-related relatives were seropositive for low-active Ab-proteases from which 22-28% of the seropositive relatives established were being monitored for 2 years whilst demonstrating a stable growth of the Ab-associated proteolytic activity. Moreover, some of the low-active Ab-proteases in persons at MS-related risks (at the subclinical stages), and primary clinical and MRT manifestations observed were coincided with the activity to have its mid-level reached. Registration in the evolution of highly immunogenic Ab-proteases would illustrate either risks of transformation of subclinical stages into clinical ones, or risks of exacerbations to develop.

The activity of Ab-proteases in combination with the sequence-specificity would confirm a high subclinical and predictive (translational) value of the tools as applicable for personalized monitoring protocols.

The translational potential of this knowledge is in the rational design of new diagnostic tools and new targeted therapeutics based on principles of artificial biocatalysts and biodesign and to exploit the role of the key pathways in influencing disease. The last years have seen a major upturn in the fortune of Therapeutic Abs, approved for clinical use. This success can be related to the engineering of mAbs into chimeric Abs, or humanized ones, which have had a major effect on immunogenicity, effector function and half-life.

Emerging have created a vast range of novel, Ab-based therapeutics, which specifically target biomarkers of disease. Ab-proteases can be programmed and re-programmed to suit the needs of the body metabolism or could be designed for the development of principally new catalysts with no natural counterparts. The latter would suit the needs of the body metabolism or could be designed for the development of principally new catalysts with no natural counterparts. So, Ab-Protease Engineering would offer the ability to enhance or alter their sequence-specific activity to expand the clinical utility of the absolutely new tools to suit the neurodegenerative pathology standards.

The dearth of knowledge about the pathology of MS and the clinical variation in MS subtypes makes it implausible to establish a single ideal biomarker which guarantees the full evaluation of the disease. Moreover, there remains a need for a panel of validated biomarkers that are capable of predicting and monitoring the efficiency of the growing number of treatment strategies available, with the aim of reducing the recurrence of relapses, and stopping the progression and disability of patients with MS. So, the next important step in the direction of the innovations-based approach should be early adoption of Ab proteases as biomarkers of the next step generation in clinics for future medical interventions! Neurodegenerative diseases are promisingly suited models for PPM because of the rapidly expanding design-driven innovations including ABZYMES technologies and the development of pathology-valuable biomarkers and the potential modifying treatments. And we have made the first step in this direction to improve the management of MS. With advances in our understanding of Ab-protease functions and properties coupled with improvements in immune and protein engineering we can expect that Ab-protease therapeutics will gain regulatory approval and make significant contributions in healthcare in the near-term.

Further studies on targeted Ab-mediated proteolysis may provide a translational tool for predicting demyelination conditions illustrating the crucial feature of neurodegenerative pathology, and thus the disability of the MS patients and/or persons-at-risk.

Biography

Sergey Suchkov was born in the City of Astrakhan, Russia, in a family of dynasty medical doctors. In 1980, graduated from Astrakhan State Medical University and was awarded with MD. In 1985, Suchkov maintained his PhD as a PhD student of the I.M. Sechenov Moscow Medical Academy and Institute of Medical Enzymology. In 2001, Suchkov maintained his Doctor Degree at the National Institute of Immunology, Russia. From 1989 through 1995, Dr Suchkov was being a Head of the Lab of Clinical Immunology, Helmholtz Eye Research Institute in Moscow. From 1995 through 2004 - a Chair of the Dept for Clinical Immunology, Moscow Clinical Research Institute (MONIKI). In 1993-1996, Dr Suchkov was a Secretary-in-Chief of the Editorial Board, Biomedical Science, an international journal published jointly by the USSR Academy of Sciences and the Royal Society of Chemistry, UK.

Srikar D[1], Dr. T Kishore Kumar[2*]

[1]Research Scholar (PT), Department of ECE, NIT Warangal, India

[2]Professor, Department of ECE, NIT Warangal, India

AI based detection of iron deficiency anemia using conjunctiva of images

Anemia is an iron deficiency that often results in the decrease of Red Blood Cells (RBC). One of the worldwide public health issues affecting children and pregnant women is anemia. The main cause of anemia is increased RBC destruction, blood loss, and defective cell production. It also occurs when the amount of RBC within the body decreases, or when the Hemoglobin level of blood is below the normal threshold. Achieving a high level of accurate detection of anemia is a challenging task in existing models. Anxiety and expense are two difficulties in the invasive method of identifying anemia, which prevents advancements in health. Hence, it is essential to develop non-invasive methods for diagnosing anemia that reduce expenses and increase detection efficacy. This proposed model helps to detect iron deficiency anemia from the conjunctiva images of humans using the deep learning model. The early detection of iron deficiency anemia is helpful to take proper medical facilities to enhance the quality of life of the human. The conjunctiva images required to perform the anemia detection are collected from the benchmark dataset. The collected conjunctiva images are directly passed to the Nested Dilated Efficient Attention Network (NDEAN) for detecting iron deficiency in humans. The developed model is used to find the anemic state of the human in a more efficient way. Finally, the effectiveness of the NDEAN is determined via the validation process among several performance indices.

Keywords: Iron Deficiency Anemia Detection, Conjunctiva Images, NDEAN-Nested Dilated Efficient Attention Network.

Biography

Prof. T Kishore Kumar is currently a professor in the Department of ECE and a former head, Department of ECE and Institute Computer Center, National Institute of Technology Warangal, India. Prof. Kishore current areas of research include signal processing, artificial intelligence and biomedical signal processing, machine learning for speech, image and medical applications and AI in healthcare.

Srikar. D[1], Dr. T. Kishore Kumar[2*]
[1]Research Scholar(PT), Department of ECE, NIT Warangal, India
[2]Professor, Department of ECE, NIT Warangal, India

Explainable AI based dilated convolutional self-attention based bidirectional gated network for epilepsy seizure detection

Epilepsy affects 50 million people worldwide and has a significant influence on their quality of life. Epilepsy prevalence varies depending on a variety of conditions, although it is more frequent in underdeveloped countries. This underscores the crucial need for advances in treatment and prevention approaches to improve people's quality of life around the world. Therefore, the proposed study introduces a novel Explainable AI (XAI) based hybrid deep learning model for detecting epilepsy seizures. The most important processes in this study are collecting data, preprocessing, extracting features, and detection. Initially, input EEG signals are acquired from a publically available dataset, then pre-processing is performed to improve signal quality by removing artifacts using the Windowing-based Modified Notch Impulse Response Filter (WMNIRF) approach. Then, the necessary features are extracted in the feature extracted stage using an Improved Pseudo-Wigner Ville Distribution (IPWVD) and Extended Fast Fourier Transform (EFFT) methods. From the extracted features, the most optimal feature sets are selected to reduce the feature dimensionality problem using Maximum Information Coefficient based Frilled Lizard Optimization (MIC-FLO) approach. Finally, based on the selected features, the epilepsy seizures are detected by proposing a novel Dilated Convolutional Self-Attention based Bidirectional Gated Network (DC-SA-BiGN) model. Finally, for making the proposed deep learning based detection model as more interpretable, Shapley Additive Explanations (SHAP) is utilized and it can analyse the features that more necessary for seizure detection. In addition, the proposed model attain accuracy of 96%, precision of 97%, recall of 96%, and specificity of 96%.

Biography

Prof. T Kishore Kumar is currently a professor in the Department of ECE and a former head, Department of ECE and Institute Computer Center, National Institute of Technology Warangal, India. T Kishore current areas of research include signal processing, artificial intelligence and biomedical signal processing, machine learning for speech, image and medical applications and AI in healthcare.

Tatiana Korolenko*, Alexandr Pupyshev, Viktor Belichenko
Scientific Research Institute of Neurosciences and Medicine, Novosibirsk, Russia

Effect of autophagy induction on neurons and glial cells of db/db mice with diabetes T2 and neurodegeneration development

Type 2 diabetes is associated with the formation of features of Alzheimer's Disease (AD). A common mechanism appears to be the impairment of autophagy, making its stimulation a potential target for AD treatment. A good opportunity to study the correction of diabetes and neurodegeneration is provided by db/db mice, a model of diabetes and obesity that develop signs of AD with age. In our previous work, we have found that db/db mice are amenable to treatment with the disaccharide trehalose, which activates autophagy via an mTOR-independent pathway. The aim of this study was to evaluate the effect of trehalose in old db/db mice. In 3-month-old mice, trehalose reduced obesity, attenuated hyperglycinaemia, significantly activated autophagy in the brain, weakened neuroinflammation and oxidative stress, and restored cognitive impairment. It remains unclear to what extent the therapeutic effect of trehalose depends on the age of mice and on the activation of autophagy gene transcription and ultrastructural changes in neurons and glia cells. The therapeutic effect of treatment with 3% trehalose in drinking was investigated on 5-month-old db/db mice. Trehalose did not induce a significant decrease in the body mass or blood glucose and cholesterol levels but it decreased the expression of the insulin receptor gene Insr. There was an increase in the lipofuscin levels in cortical neurons and glial cells, while trehalose did not attenuate the accumulation of the marker.

Conclusions: Thus, a differential effect of trehalose treatment was obtained for 5-month-old db/db mice compared young mice, consisting in the absence of activation of autophagy gene transcription or attenuation of lipofuscin accumulation. Apparently, the therapeutic effect of trehalose on the disturbances in db/db line mice decreases with age and becomes less effective at 5 months of age.

Biography

Dr. Tatiana Korolenko studied Medicine and Chemistry at the Novosibirsk Medical University, Russia (former USSR) and graduated as MS in 1965. Dr. Korolenko then joined the research group of Prof. R.I. Salganik, Institute of Cytology and Genetics, Russian Academy of Sciences, Novosibirsk, Russia; then Prof. Y. Natori at the Tokushima University, Japan and later Laboratory of Prof. H. Fritz at the Ludwig-Maximilians-Universität in Munchen, Germany. Dr. Korolenko received her PhD degree in 2020 at the Novosibirsk Medical University; after 1-year postdoctoral fellowship she obtained the position of Professor at the Novosibirsk Medical University. Dr. Korolenko has published more than 70 research articles in SCI (E) journals).

Valida A Isanova
Professor, Kazan State Medical University, Russia

Pathogenetic physical rehabilitation of neurological patients by kinesotherapy in the anti-gravity "Atlant" rehabilitation costyume

Scientists have expanded the notion of the brain plasticity. If necessary, even specialized motor neurons can be taught to perform functions completely alien to them.

Neurophysiologists from the University of California, Berkeley (USA) described an experiment in which the rat neurons were reconfigured. It can be said that the rats have learned to "think" differently.

With the help of straining devices, installed in the pneumosuite “Atlant” along the antagonistic muscles of body and limbs, the myotatic reflex on extension is triggered in each segment, which activates the motor centres functions in all CNS levels. Mechanism of action: In response to proprioceptive stimulation caused by the approximating effect of the suit, stimulating the mechanisms of autoregulation of the α-γ-motoneuron system, which affects the restoration of the muscle tone, motor functions are activated.

The Kinesio therapeutical method in “Atlant” creates the conditions for the patient’s active participation in mobility rehabilitation process. The patients gain walking skills and other daily important moving skills, which become possible only with the adequate position of the body and limbs. Through the available objective trainings with the usage of pneumosuit “Atlant”, the patients establish social activity and communication, normal for various functional systems in the motor-cognitive interaction. The methods of our rehabilitation system are based on the mechanisms and principles of afferent proprioceptive stimulation of the myotatic stretch reflex developed by the US clinical neurophysiologist, G. Kabat.

Biography

Valida A. Isanova, In 2000 Valida Isanova was transferred to a position of professor of department of neurology and neurosurgery of the Kazan State Medical University where continues to work to the present. Doctor of medical sciences, Professor Valida Isanova personally studied pathogenetic methods of nervous system diseases' rehabilitation in clinics of Germany, England, Switzerland, Austria, China. One of achievements is developed for restoration of motor and cognitive impairments the author's technology "The method of kinesiotherapy of neurologic patients with impairments of motor functions and Rehabilitation Pneumosuit (RPS) "Atlant" in the method" (domestic analog of PNF). These technologies in neurorehabilitation were included into an educational program of professional development of neurologists and neurosurgeons in Kazan State Medical University. The techniques developed by professor Isanova were reported in the oral presentations at the international conference "Movement: Brain, body, cognition" in Oxford in 2017 and at the international Neurology, Brain Disorders conference "Neurology and brain disorders" in Rome in 2018. 3rd Edition of International Conference on June 24-26, 2019 Paris, France.

Jie Zhou[1,2], Yingli Yang[1,2,3], Jiacheng Tian[1,2], Cong Liu[1,2], Jiafei Chen[4], Mei Yang1,2, Mengran Zhang[1,2,3], Yu Duan[1,2], Tian Zhang[1,2], Yapei Sun[1,2], Qin Yu[1,2], Zhiqin Xia[1,2], Xinglin Wan[1,2], Weixia Duan[1,2*], Shangcheng Xu[1,2,5]

[1]Chongqing Key Laboratory of Prevention and Treatment for Occupational Diseases and Poisoning, China

[2]The First Affiliated Hospital of Chongqing Medical and Pharmaceutical College, China

[3]Nanjing Medical University, China

[4]Army Medical University (Third Military Medical University), Chongqing 400038, China

[5]Chongqing Medical University, China

Diquat exposure induces brainstem demyelination and encephalopathy via upregulating the mitochondrial calcium uniporter

Diquat (DQ) is a widely used new herbicide that poses a great threat to the environment, ecological systems and human health. Although the Central Nervous System (CNS) is a sensitive target of DQ exposure, the major brain regions, pathological changes and underlying mechanisms of DQ damage to the CNS remain obscure. We demonstrated that the brainstem was the primary region where DQ damaged the CNS. DQ exposure damaged both neurons and glial cells and disrupted neurotransmitter metabolism. DQ caused brainstem demyelination, as indicated by the loss of myelin sheaths, decreased levels of myelination biomarkers, and abnormal myelin morphology. Mechanistically, the expression of the Mitochondrial Calcium Uniporter (MCU) was increased in the DQ-exposed brainstem, and MCU knockdown mice were less sensitive to DQ-induced demyelination and CNS injury by attenuating disturbances in brain energy metabolism via the AMPK pathway. Moreover, the inhibition of MCU efficiently improved DQ-induced mitochondrial dysfunction in vitro. Overall, this study is the first to reveal that the brainstem is the key injured brain region and that demyelination is the prominent pathological feature induced by DQ exposure. The MCU is a potential therapeutic target for DQ-induced demyelination and CNS injury. These novel findings expand our understanding of DQ-induced CNS injury and offer a promising therapeutic strategy.

Biography

Dr. Duan earned her Ph.D. in Medicine from the Third Military Medical University in China in 2015. Dr. Duan currently serves as a Chief Physician, Associate Professor, and Master's Supervisor. Dr. Duan works at the First Affiliated Hospital of Chongqing Medical and Pharmaceutical College and is recognized as a promising young scientist. Dr. Duan previously conducted a one-year academic visit at Heidelberg University. Dr. Duan primary research focuses on the pathogenic mechanisms of pesticide poisoning and corresponding prevention/treatment measures. Dr. Duan has led 11 research projects, including grants from the National Natural Science Foundation of China, and has published over 30 academic papers.

Zhanna Semenova[1*], S. V. Meshcheryakov[1], A. G. Gagloev[1], I. V. Melnikov[1], V. I. Lukyanov[1], M. I. Daskovsky[2] E. A

[1]Research Institute of Emergency Pediatric Surgery and Traumatology-Dr. Roshal's Clinic, Department of Health of the City of Moscow, Russia

[2]FSUE SRC "Kurchatov Institute", VIAM, Russia

Comparative analysis of autologous bone and custom hydroxyapatite implants (Custombone) for post traumatic skull defects in paediatric patients

Introduction: The concept of reconstruction post-traumatic skull defects is based on restoring anatomical relationships to maximize recovery of brain function, i.e., it is considered as a mandatory stage of surgical rehabilitation. The choice of implant, taking into account age-related features, is limited for the pediatric patients. According to the literature, the use of autobone is accompanied by a high percentage of resorption. Hydroxyapatite-based implants are considered as an alternative material.

Objective: To compare the effectiveness of autologous bone and custom hydroxyapatite implants (Custombone) in paediatric patients with post traumatic skull defects.

Material and Methods: The study included 85 patients aged 1.5 to 17 years with post-traumatic skull defects >5cm in diameter. Autograft was used in 57 cases, and in 28 cases-an individual Custombone implant. The follow-up period ranged from 1 to 10 years.

Results: Early-stage infectious complications were 2.5% (2 patients) in patients with autobone. Complete resorption requiring repeated surgical treatment was 6 observations (10.1%). When using Custombone, no complications were observed in the early and late postoperative period. However, the use of the implant was not possible in children under 3 years due to the mismatch between the thickness of the implant and the thickness of the child's bone, and the high cost limited its widespread use. According to the literature, the average osteointegration coefficient of Custombone is 37.4% (Wim Maenhoudt, 2018). In our observations, partial osteointegration was diagnosed in 9 patients (31%).

Conclusion: Both materials have distinct advantages and limitations:

1. **Autologous** Bone: Risk of complications (infections, resorption).

2. **Custombone:** No early/late complications, but age restrictions and incomplete osteointegration (31 % partial vs. 37.4 % average).

The findings support the need for further development of hydroxyapatite based implants to improve osteointegration and adapt them for younger patients. Supported by a grant No. 0511-9/25 from the Moscow Department of Health.

Biography

Semenova Zhanna Borisovna is a Chief of Pediatric Neurosurgeons Health Department of Moscow and Central Federal District of the Ministry of Health of the Russian Federation. Head of the Department of Neurosurgery and Neurotrauma of the Research Institute of Emergency Pediatric Surgery and Traumatology-Clinic of Dr. Roshal. Professor at the Department of Pediatric Neurosurgery, Russian Medical Academy of Continuig Professional Education. Member of the European Multidisciplinary Academy of Neurotrauma (EMN). Academic Secretary of the Association of Neurosurgeons of Russia. President of the Society for Pediatric Neurosurgery of the Russian Federation. Member of the Editorial Board of the journal Burdenko's Journal of Neurosurgery. Awards and titles: 2016-Holder of the golden Arty Order "For Honor and Dignity". 2016National Award for the best doctors of Russia. 2021-Prof. V. V. Lebedev Medal "For merits in emergency neurosurgery".

Zhenhuan Liu
Nanhai Maternity and Children Hospital, Affiliated to Guangzhou University of Chinese Medicine, China

Neuroimaging-based evaluation of scalp acupuncture for neural repair and reorganization in children with cerebral palsy

Objective: To investigate the effect of and Acupuncture on brain plasticity and motor development in children with cerebral palsy. Investigate effect on mechanism of apoptosis of brain nerve cells, regulating the expression of neurotrophic factors, promoting the remodeling of nerve synaptic structure and motor development in young rats with cerebral palsy. Two: To evaluate the effect and mechanism of acupuncture on cerebral palsy. Three: The nerve repair effect of acupuncture on cerebral palsy.

Methods: In this study, 146 cases of brain injury and 1078 cases of cerebral palsy were included by randomized controlled study with ICF Gross motor function measure, Peabody fine motor function, Gesell, muscle tension, joint activity, activity of daily living transcranial doppler, Skull B ultrasound, Brain Nuclear Magnetic Resonance Imaging MRI, Positron Emission Tomography SPECT, Diffusion tensor tractography evaluation method.

Results: The recovery rate of extracellular space (92.3%) was significantly higher than that of the control group (70.8%) ($P<0.05$), Transcranial Doppler, TCD total efficiency (79.3%) was significantly higher than that in the control group (51.8%) ($P<0.05$). Acupuncture to promoting the development of neurological and cognitive movement under 6 months children, effectively reduce the neurological sequelae. The total effective rate of the children with cerebral palsy was 87% in the acupuncture group, which was significantly higher than that of the control group ($P<0.01$). The total effective rate of Brain MRI was 59.55% in the acupuncture group and 13.25% higher than that in the control group ($P<0.01$). The total effective rate was 91.3% in the 1year follow-up group, which was significantly higher than that in the control group ($P<0.01$).

the FA value of white matter fiber bundle was significantly higher than that of acupuncture at 60 times (P<0.05). The recovery rate of ultrasonous brain injury (86.7%) in acupuncture group was significantly higher than that in control group (64.4%) (P<0.05). The recovery rate of brain SPECT in acupuncture group was 96.4%, which was significantly higher than that in the control group (P<0.01).

Conclusion: Acupuncture rehabilitation not only promote the development of white matter and gray matter in children with cerebral palsy, but also promote the brain function of children with cerebral palsy remodeling and compensation, and promote social adaptation, language and other cognitive function development, children with cerebral palsy movement and Fine motor function development and recovery, improve the children's self-care ability.

Keywords: Cerebral Palsy, Acupuncture, Nerve Repair, Remodeling, Motor Function.

Biography

Zhenhuan LIU professor of pediatrics, Pediatric acupuncturist Ph. D. tutor. LIU has been engaged in pediatric clinical and child rehabilitation for 40 years. Led the rehabilitation team to treat more than 40,000 cases of children with intellectual disability, cerebral palsy and autism from China and more than 20 countries, More than 26800 childrens deformity returned to school and society and became self-sufficient. The rehabilitation effect ranks the international advanced level. Vice-chairman of Rehabilitation professional committe children with cerebral palsy, World Federation of Chinese Medicine Societies. Visiting Professor of Chinese University of Hong Kong in recent 10 years. LIU is most famous pediatric neurological and rehabilitation specialists in integrated traditional Chinese and Western medicine in China. LIU has edited 10 books. LIU has published 268 papers in international and Chinese medical journals.

12TH EDITION OF

International Conference on

NEUROLOGY AND NEUROLOGICAL DISORDERS

HYBRID EVENT

22-24 JUNE 2026

NEUROLOGY

JUNE 22-24 | 2026

POSTER PRESENTATIONS

Anwita Golem
Blue Valley School District, United States

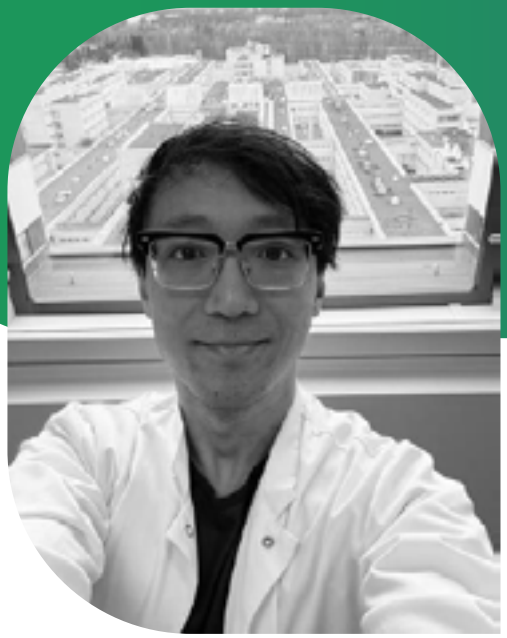
Genomic disruption of the NRXN1 gene: Implications for synaptic biology and neuropsychiatric disorders

Introduction: Advances in high-resolution genomic technologies-including chromosomal microarray, next-generation sequencing, and targeted CNV assays-have substantially enhanced our ability to detect clinically meaningful structural variation in patients with suspected neurodevelopmental and neuropsychiatric disease. Within this landscape, deletions involving the NRXN1 gene have emerged as among the most recurrent and consequential findings encountered in clinical cytogenomic testing. As a presynaptic cell-adhesion molecule essential for synapse assembly and neurotransmission, NRXN1 plays a central role in neural circuit formation. A synthesis of 173 published studies demonstrates that NRXN1 haploinsufficiency is consistently associated with autism spectrum disorder, global developmental delay, speech and language impairment, epilepsy, and schizophrenia, while rare biallelic losses produce severe phenotypes resembling Pitt–Hopkins–like syndromes.

From a mechanistic perspective, NRXN1-related disease largely reflects disruption of trans-synaptic signaling within neurexin-neuroligin pathways, which are critical for maintaining excitatory–inhibitory balance and synaptic plasticity. Exonic deletions involving the 5' region and α-isoform promoter show the strongest functional effects and highest penetrance. Yet, in routine clinical practice, the phenotypic expression of NRXN1 deletions is strikingly variable-ranging from asymptomatic parental carriers to profound neurocognitive impairment in affected children-underscoring the need for careful interpretation within a broader genomic and clinical context.

These observations reinforce the importance of including NRXN1 in diagnostic microarray and sequencing workflows, especially for patients presenting with early developmental delays, ASD features, unexplained epilepsy, or psychiatric symptoms. Given the incomplete penetrance and frequent presence of additional contributing variants, genetic counseling must emphasize variable expressivity.

Collectively, current evidence positions NRXN1 as a key node in neurodevelopmental circuitry and a high-yield target for diagnostic evaluation, mechanistic investigation, and future therapeutic refinement. As the field advances toward precision-based frameworks, a deeper understanding of NRXN1 biology will be essential for improving diagnostic accuracy, prognostic clarity, and individualized patient management across the lifespan.



Emilia K. Pesonen, MB, MSc[1], Sonja Portin, MB[1], Tommi K. Korhonen, MD, PhD[1], Juha-Matti Isokangas, MD, PhD[2], Mikael von und zu Fraunberg, MD, PhD[1], Cheng Qian, MD, PhD[1*]

[1]Department of Neurosurgery, Oulu University Hospital & University of Oulu, Oulu, Finland

[2]Department of Radiology, Oulu University Hospital, Oulu, Finland

Intra-arterial nimodipine and outcomes in delayed cerebral ischemia after aneurysmal subarachnoid haemorrhage

Objective: Delayed cerebral ischemia (DCI) caused by cerebral vasospasm remains a major cause of morbidity after aneurysmal subarachnoid hemorrhage (aSAH). Intra-arterial nimodipine (IAN) is increasingly used as rescue therapy in patients with vasospasm refractory to induced hypertension, although data regarding outcomes and repeated infusions remain limited. This study evaluated functional outcomes and mortality in patients treated with IAN after a SAH.

Methods: We retrospectively identified all patients with DCI after aSAH treated with IAN in a tertiary center between 2017 and 2024. Clinical and radiological data, treatment characteristics, and outcomes were analyzed. Primary endpoints were six-month mortality and unfavorable functional outcome defined as modified Rankin Scale (mRS) score >2 at 3–6 months. Logistic regression analyses were used to identify factors associated with mortality and unfavorable outcomes.

Results: Fifty-two patients were included with a mean age of 53 years; 65.4% were female. At 3–6 months, 46.2% of patients achieved functional independence, while 53.8% had unfavorable outcomes. Six-month mortality was 10.0%. Higher Hunt and Hess grade, Glasgow Coma Scale score of 3–8 on admission, intraventricular hemorrhage, and repeated IAN administration were associated with unfavorable functional outcome. Older age and pre-existing anticoagulant and/or antiplatelet therapy were associated with increased mortality. Post-treatment infarcts were identified in 26.9% of patients, while infarcts developing despite IAN treatment occurred in 15.4%. Repeated IAN administration did not significantly increase six-month mortality but was associated with poorer functional outcomes and a higher likelihood of permanent cerebrospinal fluid shunt placement.

Conclusions: Despite representing a subgroup of severely affected aSAH patients with symptomatic vasospasm, nearly half of the patients treated with IAN achieved functional independence after 3–6 months. Mortality rates were lower than those reported in several population-based aSAH cohorts. Repeated IAN administration was associated with less favorable functional outcomes, likely reflecting greater disease severity.

Biography

Cheng Qian, MD, PhD, is a neurosurgeon at Oulu University Hospital and the University of Oulu, Finland. His research interests focus on neurovascular surgery, aneurysmal subarachnoid hemorrhage, delayed cerebral ischemia, and intraoperative navigation technologies. He is actively involved in clinical and translational neurosurgical research.

Elizabeth Farber[1*], Katelyn Gamson[1], Akanksha Sharma[1]

[1]Sutter Health California Pacific Medical Center (CPMC), United States

When left is right: Right-hemisphere language dominance enabling safe resection of a left-frontal glioblastoma in a right-handed patient

Background: Language is left-hemisphere dominant in approximately 90–95% of right-handed individuals (1–3). As a result, it is often assumed that left-frontal gliomas in right-handed patients involve critical language areas and are therefore not safe for surgical resection. However, maximal safe resection has been consistently associated with improved survival in glioblastoma and diffuse adult-type gliomas (4–8). Preoperative functional mapping techniques, including task-based functional MRI and Wada testing, can identify individual language lateralization and guide surgical planning (9–12). We describe a case in which presumed left-hemisphere language dominance did not hold, and formal assessment of hemispheric language dominance directly guided surgical planning and enabled safe tumor resection.

Case Presentation: A 68-year-old right-handed woman with limited English proficiency presented with transient right facial droop and subtle cognitive slowing. MRI of the brain revealed a 3.5-cm peripherally enhancing, centrally necrotic left-frontal subcortical mass. Biopsy confirmed glioblastoma, IDH-wildtype, CNS WHO grade 4. Preoperative imaging showed medial basal ganglia invasion and extension into the left insula and frontal operculum, suggesting tissue involvement of the regions critical for motor control, speech articulation, and expressive language, initially rendering extensive resection unsafe.

Intervention: Wada testing showed preserved language after left-hemispheric propofol injection but profound aphasia after right-hemispheric injection, confirming right-hemisphere language dominance (12). Memory was intact bilaterally. The patient was therefore deemed safe to undergo extensive surgical resection. She underwent left frontotemporal craniotomy

for resection of brain tumor with brain motor mapping and 5-ALA fluorescence guidance. Intraoperatively, deeper dissection revealed tumor extension toward the lateral ventricle and proximity to the Sylvian fissure and insula adjacent to the corticospinal tract. Circumferential tumor resection was performed while preserving critical pathways. Near-total resection was achieved, leaving a minimal rim along eloquent white matter.

Outcomes: Postoperatively, the patient retained full motor strength, had no language deficits, and maintained baseline cognition. She started adjuvant therapy with temozolomide and concurrent radiation therapy four weeks post-operatively.

Conclusion: Right-hemisphere language dominance in a right-handed patient allowed safe maximal resection of a left frontal glioblastoma. This case highlights the importance of individualized presurgical language mapping to expand surgical options and optimize outcomes in glioblastoma patients (5, 9, 13). Language discordance may further contribute to disparities, as limited English proficiency can affect nuanced discussions about functional risk and influence access to advanced presurgical evaluation.

Biography

Elizabeth Farber, MD, MS, is completing her intern year at California Pacific Medical Center and will soon begin her neurology residency at Stanford Medicine. She earned her MD from UC San Diego and an MS in Neuroscience from George Mason University. Recognized for academic achievement, leadership, and service, her interests include clinical research, medical education, and improving access to care for underserved and refugee communities. She values collaboration, teaching, and compassionate, patient-centered care.

Hanna Pawluk[1*], Renata Kołodziejska[1], Grzegorz Grześk[2], Mariusz Kozakiewicz[3], Grzegorz Kozera[4], Mateusz Pawluk[1], Magdalena Grześk-Kaczyńska[5], Natalia Soja-Kukieła[6], Alina Woźniak[1]

[1]Department of Medical Biology and Biochemistry, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[2]Department of Cardiology and Clinical Pharmacology, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[3]Department of Geriatrics, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[4]Medical Simulation Center, Medical University of Gdańsk, Poland

[5]Department of Allergology, Clinical Immunology and Internal Medicine, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[6]Statistical Analysis Centre, Nicolaus Copernicus University in Toruń, Poland

The role of interleukin 6 in ischemic stroke

Ischemic stroke is one of the leading causes of mortality and long-term disability worldwide, resulting from the sudden occlusion of a cerebral vessel and subsequent hypoxia of neural tissue. This event triggers a complex pathophysiological cascade involving excitotoxicity, oxidative stress, and activation of the inflammatory response. The immune response plays a crucial role in both the progression of tissue damage and the initiation of repair mechanisms.

Interleukin 6 (IL-6) is a key pro-inflammatory cytokine involved in the acute-phase response following ischemic stroke. Its concentration reflects immune system activation and may have prognostic and pathophysiological significance, both in neural tissue injury and in repair processes.

In the studied group of patients, significant temporal variability in IL-6 levels was observed. The highest values were recorded 24 hours after admission, indicating a peak of the inflammatory response in the early phase of stroke. Subsequently, IL-6 levels significantly decreased by day 7, reaching values close to baseline.

Post-hoc analysis revealed significant differences between measurements at time points 0–1 and 1–7 days, confirming the dynamic nature of the inflammatory response. Additionally, IL-6 levels were significantly higher compared to the control group at all time points, indicating sustained inflammatory activation in post-stroke patients.

With respect to the modified Rankin Scale (mRS) at admission, higher IL-6 levels were observed in patients with more favorable outcomes, suggesting a complex, potentially biphasic role for this cytokine, not only as a mediator of injury but also as a component of the reparative response.

IL-6 demonstrates a clear temporal profile following ischemic stroke, with a peak within the first 24 hours, supporting its role in the acute inflammatory response. Persistently elevated levels relative to controls indicate prolonged immune activation. The ambiguous relationship with clinical outcomes suggests that IL-6 may exert both pro-inflammatory and potentially neuroprotective effects, warranting further investigation.

Biography

Hanna Pawluk is a Researcher and lecturer at the Department of Medical Biology and Biochemistry, Nicolaus Copernicus University Medical College in Toruń. Author of research related to ischemic stroke, oxidative stress, and inflammation.

Hyung Jin Lee*, Hee Jong Ki, Ji Ho Yang

Daejeon St. Mary's Hospital, College of Medicine, The Catholic University of Korea, South Korea

Case report: Neuroimaging changes of nonketotic hyperglycemia chorea patient with status epilepticus attack

Background: We present the neuroimaging findings of case of Non-Ketotic Hyperglycemia (NKH) associated with seizures and chorea. Seizures of NKH patients are usually focal motor seizures, rarely progress to status epilepticus with secondary generalization.

Case Presentation: An eighty-three-year-old male came to emergency department with seizure. He also had been suffered abnormal movement like chorea for 2-3weeks. He had been diagnosed with diabetes mellitus, but was not on proper treatment for recent several weeks. Random blood sugar was 532 mg/dl. Arterial blood gas showed mild acidosis (pH 7.31) and ketone bodies were not detected in urine. Non-contrast computerized tomography brain on day 1 showed left side hyperdense lentiform and caudate nuclei. Diffusion & SWI MRI on day 1 showed focal abnormal signal intensity of left side basal ganglia. He was started on insulin and valproic acid at ER. He was transferred to other hospital for glycemic control. Five days later, He revisited our hospital due to uncontrolled seizure with unconsciousness. CT was repeated on day 6, 18 and MRI was done on day 7, 35 after glycemic control. Patient recovered clinically well and showed rapid radiological resolution, too.

Conclusion: We show rare NKH patient having seizure and chorea with serial imaging study.

Biography

Hyung Jin Lee is professor in catholic University, Korea as neurosurgeon. Lee specialized at vascular disease and neurointervention. Lee received his master's and doctoral degrees from the Catholic University of Korea, College of Medicine. Lee also completed a research fellowship at the University of Washington, St. Louis, USA. Lee is currently a full member of the Korean Neurosurgical Society, the Korean Society of Cerebrovascular Surgeons, and the Korean Neuroendovascular Society. Lee previously served as the Director of the Department of Neurosurgery and as the Chair of the Institutional Review Board (IRB) at Daejeon St. Mary's Hospital.

Valeriy E. Babelyuk[1], Igor Lvovych Popovych[2*], Ruslan S. Vastianov[3], Tetyana A. Korolyshyn[1], Galyna I. Dubkova[1], Yuriy G. Dobrovolsky[4], Svitlana M. Holubinka[1], Walery Zukow[5]

[1]Clinical Sanatorium "Moldova" of State Chancellery of the Republic of Moldova, Truskavets', Ukraine

[2]Kozyavkin International Rehabilitation Clinic, Truskavets', Ukraine
[3]National Medical University, Odesa, Ukraine
[4]Yuriy Fedkovych National University, Chernivtsi, Ukraine
[5]Nicolaus Copernicus University, Torun, Poland

Neurotropic and biophysics effects of Kyokushin Karate Kata (KKK)

Introduction: In Eastern martial arts, non-contact mental influence on a partner is described, often associated with the concept of "energy-informational exchange". The Kyokushin Karate Kata (KKK) practice is particularly noteworthy in this context.

Objective: To explore the neurophysiological and biophysical mechanisms underlying the effect of KKK on brain activity of the sender and the physiological state of the receiver-in both animal models (rat's hippocampal slice) and non-biological systems (distilled water).

Materials and Methods: Three experiments were conducted: (1) EEG, HRV, and EPI/GDV (electro-photonic imaging/gas discharge visualization) were recorded in KK master and Reiki practitioner; (2) Synaptic efficacy in rat's hippocampal slice was measured during remote mental interventions; (3) Light activity of distilled water, induced by gas discharge, was analyzed after exposure to KKK and canonical Christian prayer. Data analysis included EEG and EPI signal processing, entropy calculations, correlation, canonical, multiple regression and discriminant analysis.

Results: KKK practice, but not Reiki significantly increased EEG delta, alpha and theta Spectral Power Density (SPD), to the greatest extent in temporal and parietal loci, while decreased beta SPD to the greatest extent in frontal, prefrontal and central loci, reduced both spectral and EPI entropy, and enhanced EPI energy characteristics. Remote KKK influence induced a notable increase in rat's Hippocampal Synaptic Efficacy (HSE) (+7.5±5.1%) while Reiki was ineffective (-0.5±4.8%) (Mean±SD). Strong canonical correlations were identified between sender's parameters during sessions and changes in rat's HSE as the receiver: R=0.936; 0.931; 0.913; and 0.959 with EEG SPD EEG entropy, EPI entropy and EEG&EPI in total, respectively. Both KKK and Christian prayer increased the light activity of distilled water, but only in cases where they decreased EEG entropy and increased SPD of delta-rhythm and energy of the third virtual Chakra.

Conclusions: Findings support the propozed hypothesis of a dual-biophotonics (energy-mediated) and negentropic (information-mediated) mechanism underlying KKK's effects, observable through objective neurophysiological and biophysical parameters. Compared to other mental practices, KKK demonstrated more pronounced (Reiki) or equal (Prayer) systemic influence.

Keywords: Kyokushin Karate Kata, Reiki, Prayer, EEG, EPI/GDV, Entropy, Energy-Informational Interaction, Rat's Hippocampal Slice, Synaptic Efficacy, Water Light Activity, Neurophysiology.

Biography

Igor L. Popovych was born in 1957. After graduating with honors in 1979 from the Ternopil' Medical Institute, Igor worked as a doctor in a sanatorium (Berezhany) from 1980 to 1983. In 1981, Igor entered the correspondence postgraduate study at the O. O. Bogomolets Institute of Physiology of the NAS of Ukraine, from where in 1983 Igor was sent to the laboratory for studying the mechanisms of physiological action of mineral waters (Truskavets'). Junior RF (1983-90), RF (1990-93), senior RF (1993-2000), leading RF (2000-2010), head (2010-2016). Igor retired due to age in 2017. Since 2008 (part-time) and currently a senior RF at the Kozyavkin International Rehabilitation Clinic.

Crysvane Araújo de Oliveira Lima[1,2], Polliany da Silva Mendonça[1,2], Joelson Germano Crispim[1,2], Roger Luis da Silva[1,2], Ryan Cordeiro Silva[1,2], Lucas Felipe de Melo Alcantara[1,2], Filipe Silveira Duarte[1,2], Moacyr Jesus de Melo Rego[1,2], Maira Galdino da Rocha Pitta[1,2], Michelly Cristiny Pereira[1,2], Michelle Melgarejo da Rosa[1,2*]

[1]Center for Therapeutic Innovation-Suelly Galdino (NUPIT-SG), Recife, Brazil

[2]Department of Biochemistry, Federal University of Pernambuco, Recife, Brazil

Medial prefrontal cortex, dopamine and glutamate modulation in regulating reward-seeking and frustration responses

Adaptive behavior depends not only on learning from rewards, but also on the brain's capacity to respond when expected rewards fail to occur. Reward omission is a powerful trigger of negative emotional states such as frustration and anxiety and is increasingly recognized as a key mechanism underlying maladaptive behaviors in neuropsychiatric and neurological disorders. The Medial Prefrontal Cortex (mPFC) plays a central role in integrating reward expectations with emotional regulation, yet the specific contributions of its dopaminergic and glutamatergic signaling remain poorly understood. In this study, we investigated how dopamine D2 and NMDA receptor signaling within the mPFC regulates reward-seeking behavior and emotional responses under conditions of reward omission. Using a cued sucrose-seeking task, we show that intact D2 and NMDA receptor activity in the mPFC is critical for sustaining reward-seeking behavior when expected rewards are omitted, revealing a key mechanism supporting behavioral persistence in the face of negative prediction errors. In contrast, only D2 receptor blockade disrupted cue-driven reward-seeking behavior, indicating a selective role for dopaminergic signaling in cue-reward associations. Notably, selective NMDA receptor blockade and functional silencing of the mPFC reduced anxiety-like behavior following reward omission, dissociating motivational persistence from emotional reactivity. These findings highlight a nuanced and bidirectional role of the mPFC, in which

dopaminergic and glutamatergic pathways differentially regulate motivation and affective responses when expectations are violated. Together, our results provide new mechanistic insight into how the prefrontal cortex orchestrates behavioral and emotional adaptation to unmet expectations. These findings have direct relevance for conditions characterized by impaired reward processing and maladaptive persistence, including depression, anxiety disorders, addiction, and neurodegenerative diseases affecting frontal circuits.

Biography

Dr. Melgarejo holds a PhD in Neurobiology/Neurosciences from the Leibniz Institute for Neurobiology, Magdeburg, Germany, where she investigated the molecular mechanisms underlying brain plasticity and homeostatic synaptic plasticity. Dr. Melgarejo earned a Master's degree in Biochemistry from the Federal University of Santa Maria (UFSM), with a focus on memory and behavior, and a Bachelor's degree in Pharmacy from the same institution. Dr. Melgarejo is currently an Associate Professor in the Department of Biochemistry at the Federal University of Pernambuco (UFPE), a researcher at the Suely Galdino Center for Therapeutic Innovation Research (NUPIT). Internationally, she has served as a Visiting Professor at Stanford University, working in the laboratory of Dr. Michelle Monje, a world-renowned leader in neuro-oncology. Dr. Melgarejo also completed a postdoctoral fellowship in 2023 at the Department of Neurobiology and Anatomy, McGovern Medical School, The University of Texas Health Science Center. Additionally, she was a Visiting Professor at the German Cancer Research Center (DKFZ) in Heidelberg. Dr. Melgarejo's research focuses on: (1) Neuromodulation of psychological and cognitive disorders, including major depressive disorder and anxiety disorders; (2) The development of natural and synthetic therapeutic alternatives for psychological, neurological, and neuro-oncological disorders; and (3) The interaction between the central nervous system and tumorigenesis.

Renata Kołodziejska[1*], Hanna Pawluk[1], Grzegorz Grześk[2], Mariusz Kozakiewicz[3], Grzegorz Kozera[4], Mateusz Pawluk[1], Magdalena Grześk-Kaczyńska[5], Natalia Soja-Kukieła[6], Agnieszka Tafelska-Kaczmarek[7], Alina Woźniak[1]

[1]Department of Medical Biology and Biochemistry, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[2]Department of Cardiology and Clinical Pharmacology, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[3]Department of Geriatrics, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[4]Medical Simulation Center, Medical University of Gdańsk, Poland

[5]Department of Allergology, Clinical Immunology and Internal Medicine, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland

[6]Statistical Analysis Centre, Nicolaus Copernicus University in Toruń, Poland; 7Department of Organic Chemistry, Faculty of Chemistry, Nicolaus Copernicus University, Poland

The role of TNF-α in ischemic stroke

Ischemic stroke is one of the leading causes of mortality and long-term disability worldwide. Its pathogenesis is associated with the sudden occlusion of a cerebral artery, resulting in a critical reduction of blood flow and oxygen supply to brain tissue. This triggers a complex cascade of pathophysiological processes, including energy failure, glutamate-mediated excitotoxicity, oxidative stress, and activation of the inflammatory response.

Neuroinflammation Plays A Dual Role in Stroke: It contributes to neuronal damage while also initiating repair processes. These mechanisms involve numerous cytokines and immune mediators, including Tumor Necrosis Factor Alpha (TNF-α), a key proinflammatory cytokine that regulates immune responses, modulates blood–brain barrier permeability, and influences neuronal survival.

In the analyzed patient group, significant temporal changes in TNF-α levels were observed. The highest concentrations were recorded 24 hours after stroke onset, corresponding to the peak of the acute inflammatory response. Both 24-hour and 7-day measurements differed

significantly from baseline values. However, no significant difference was found between day 1 and day 7, suggesting a sustained but relatively stable inflammatory activation.

Compared with the control group, TNF-α levels were significantly elevated only at 24 hours, whereas no statistically significant differences were observed at baseline or on day 7. This indicates that the increase in TNF-α is transient and primarily confined to the early acute phase.

No significant correlations were identified between TNF-α levels and clinical outcomes assessed using the NIHSS and mRS scales, suggesting limited utility of this cytokine as a prognostic biomarker.

In summary, TNF-α demonstrates a transient increase during the acute phase of ischemic stroke, peaking within the first 24 hours. Its elevated levels do not persist in later stages and do not show consistent associations with clinical outcomes. These findings suggest that TNF-α primarily serves as a marker of the early inflammatory response, with limited prognostic value in later stages of the disease.

Biography

Renata Kołodziejska is a Researcher and lecturer at the Department of Medical Biology and Biochemistry, Nicolaus Copernicus University Collegium Medicum in Toruń. Renata is actively engaged in scientific research on the molecular mechanisms underlying ischemic stroke, with a particular focus on oxidative stress and inflammatory pathways. Author and co-author of publications contributing to the understanding of stroke pathophysiology.

Sharafath Hussain Zahir Hussain*, Kehinde O. Sunmboye
Medicine, Rheumatology, University Hospitals of Leicester NHS Trust, Leicester, GBR, United Kingdom

Progressive multifocal leukoencephalopathy mimicking a cerebral vasculitis flare

Introduction: Progressive Multifocal Leukoencephalopathy (PML) is a rare opportunistic infection caused by reactivation of the John Cunningham (JC) virus in immunocompromised patients and may present with focal neurological deficits that mimic stroke.

Case Presentation: We report a case of a 68-year-old woman with a background of primary cerebral vasculitis, which was diagnosed two years ago. She appeared to have had a recurrence of her symptoms with new onset expressive dysphasia, right-sided upper limb weakness, and right-sided facial weakness during a rheumatology clinic visit. The patient was on maintenance azathioprine for her cerebral vasculitis at the time of presentation. She had received a total of 2g of rituximab through intravenous infusion, with a two-week interval between doses. Additionally, she had undergone intravenous cyclophosphamide treatment (15mg/kg) following the standard vasculitis regimen for induction remission therapy, which had been administered at the time of her diagnosis two years prior.

Initial imaging on non-contrast Computed Tomography (CT) of the head after admission to the emergency department did not show any acute neurological findings. Further imaging studies revealed changes in the right parietotemporal white matter T2 hyperintensity with similar changes in the left frontal and left parietal lobes suggestive of Progressive Multifocal Leukoencephalopathy (PML). A Magnetic Resonance Imaging (MRI) scan of the brain conducted three months earlier had been unremarkable. Cerebrospinal Fluid (CSF) Polymerase Chain Reaction (PCR) testing confirmed the presence of polyoma John Cunningham (JC) virus Deoxyribonucleic Acid (DNA).

Conclusion: This case highlights that progressive multifocal leukoencephalopathy should be considered an important differential diagnosis in immunocompromised patients presenting with new stroke-like features.

WE WISH TO MEET YOU AGAIN AT OUR UPCOMING EVENTS

13th Edition of International Conference on
Neurology and Brain Disorders
October 19-21, 2026 | Boston, Massachusetts, USA
https://neurologycongress.com/

13th Edition of International Conference on
Neurology and Neurological Disorders
June 2027 | Hybrid Event
https://neurology.magnusconferences.com/

Questions? Contact
Phone: +1 (702) 988-2320 | Whatsapp: +1 (540) 709-1879
E-mail: neuro@magnusconference.com