magnus Group

11TH EDITION OF INTERNATIONAL CONFERENCE ON

NEUROLOGY AND NEUROLOGICAL DISORDERS

JUNE
05-07
ROME,ITALY
2025

VENUE:
NH Villa Carpegna
Via Pio IV, 6, 00165 Roma RM, Italy

BOOK OF ABSTRACTS

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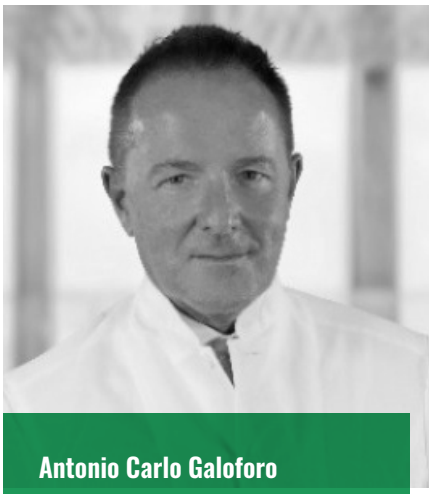

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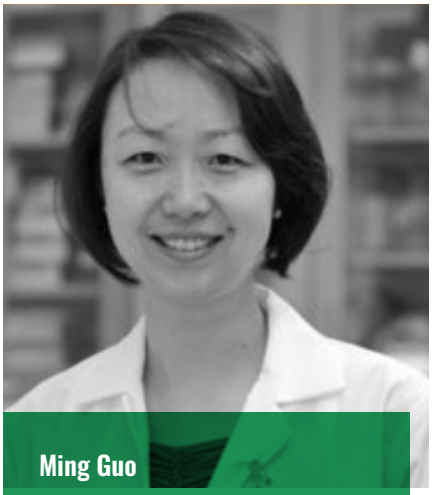

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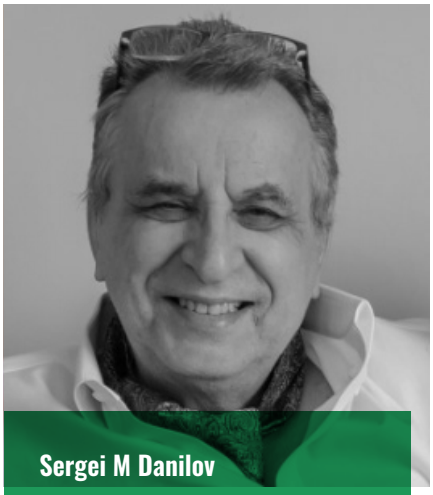


Sergei M Danilov
University of Illinois, United States

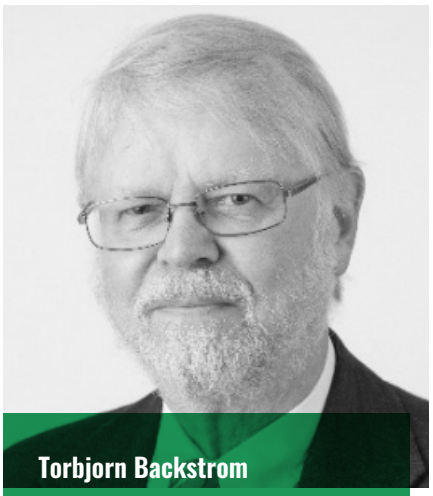
Keynote Speakers

Sergey Suchkov

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

Stefano Sandrone

Imperial College London, United Kingdom



Torbjorn Backstrom

Umea University Hospital, Sweden

W S El Masri

Keele University, United States

Yong Xiao Wang

Albany Medical College, United States

Zhenhuan Liu

University of Chinese Medicine, China

Thank You All...

Welcome Message

Dear members and attendees of Neurology 25,

It is my great honour and privilege to warmly welcome you all to Neurology 25, our distinguished gathering of minds in the heart of Rome this June 2025. I extend my sincere gratitude to each of you for your dedication and hard work in preparing and presenting your groundbreaking research and insights at this esteemed conference. A special welcome and thanks also go to those who have joined us to support your colleagues and to engage with the wealth of knowledge shared by our presenters.

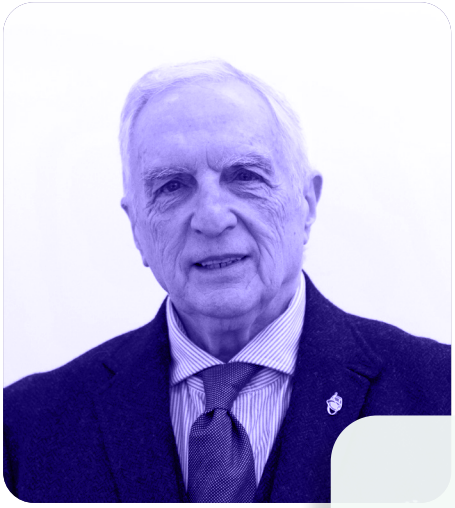
Neurology 25 promises to build on the success of previous conferences, offering an even broader range of scientific sessions and categories. This year's event is set to be one of our most dynamic and inspiring yet, with numerous opportunities for new and exciting collaborations to emerge. I am confident that these exchanges will significantly advance our collective understanding and inspire future research in the field of neurology and neuroscience.

I would like to express my heartfelt thanks to the dedicated organizing committee for their tireless efforts in making this conference a reality and ensuring it will be a memorable experience for all.

I wish you all an enriching and enjoyable time at Neurology 25. May your presentations be well-received and may you forge valuable new connections. In closing, I kindly encourage everyone to stay engaged throughout the conference and support your fellow presenters, especially those making their international debut. Let’s make this a truly inclusive and supportive environment for sharing and learning.

Ken Ware

NeuroPhysics Therapy Institute and Research Centre, Australia



Welcome Message

Welcome to the 11th Edition of International Conference on Neurology and Neurological Disorders. This event will offer an opportunity to be acquainted to the latest breakthroughs and advancements in neurology under the theme "Frontiers of Neurology: Bridging Science, Medicine, and Technology". The event has a comprehensive programme promising to uncover the complexities of neurological science, as well as providing a platform for enriching dialogues and collaborations.

Neurology 2025 will explore an extensive range of topics, from neurodegenerative diseases and clinical neurophysiology to brain injuries, cognitive neuroscience and much more. Cutting-edge subjects such as neuroinformatics, human brain mapping, and the convergence of neuroscience with artificial intelligence will be highlighted. The diverse agenda covers molecular genetics, brain pathology, neuroimmunology, and much more, offering a holistic view of contemporary neurological science.

I would be very pleased to welcome you in Rome in 2025!

Professor Luiz Moutinho

University of Suffolk, England.

Welcome Message

Dear Colleagues, Scientists & Clinicians, Biodesigners & Biotechnologists, Partners & Friends,

On behalf of the Organizing Committee, I look forward to welcoming you to the 11th Edition of International Conference on Neurology and Neurological Disorders, which is to be hold in Rome as one of the ancient, historic, well-known, prestigious and attractive City in the World.

Building on the success of the preceding meetings, the Conference will feature a highly interactive, stimulating and multidisciplinary Program including initiatives to unveil the secrecy of Personalized & Precision Medicine (PPM) and Neurology (PPN) and aiming at making bridges between Academia, Clinical Practice, Bioindustry and Business. So, the scope and quality of the scientific exchange makes this Conference the elite neurology-related and brain research, practice and instructional meeting in the world.

Today PPN is becoming the strategic mainstream of brain research & clinical neurology practice, and significantly influences the future of the care based on the evidence-based medical research, the needs and the challenges in daily neurology, and the strategies of neuro-supported biopharma as well. Making progress in the field of PPN is thus one of the most significant global challenges of our time. And advances in fundamental, translational and clinical research are beginning to transform the clinical neurology to make it personalized!

The Conference provides a unique opportunity for university leaders, educators, neurologists and experts from all over the world to convene and share novel ideas on crucial issues and trends in the area of PPN and clinical neurology as a whole. From prevention through optimal care and, finally, to rehabilitation the whole range of neurological diseases will be presented with world-known faculty, eminent researchers, highly experienced clinicians and talented educators.

The Conference will provide enough space for discussion, meetings and workshops in order to allow a close contact between speakers and visitors and will secure the ideal forum to stimulate ideas and establish collaborations. Your active contribution will thus be crucial to the success of the Conference! Together, we will become a stronger voice and global force with the ultimate goal of succeeding in decreasing the worldwide burden of the illness.

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 also hope and trust that you will enjoy your visit this Grand Event and to the unique and exciting City of Rome as well!

Warmest and productive wishes and hope to meet and to see you soon in Rome!

Dr. Sergey Suchkov, MD, PhD

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

Centro de Estudios de la Fotosíntesis Humana, Aguascalientes, México

Welcome Message

Dear congress visitors; It is an honor and pleasure to write a few welcome notes. Welcome friends from the neuroscience community to 11th Edition of International Conference on Neurology and Neurological Disorders, and I hope we can gather in Rome, Italy in June 05-07, 2025. To share the latest advances, new knowledge, new ideas and new technologies in neuroscience. To contribute to the advancement of international neuroscience and the treatment and rehabilitation of neurological diseases.

I will give a keynote speech. The title of my lecture is “Neuroimaging by Evaluation Nerverenovate and Neuroplasticity of Acupuncture in Children with Cerebral Palsy I wish the conference a complete success.

Zhenhuan LIU

Professor of Pediatric Neurologist

Guangzhou University Chinese Medicine, China

ABOUT
MAGNUS GROUP

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 2025

We are pleased to welcome you to the **11th Edition of the International Conference on Neurology and Neurological Disorders (Neurology 2025)**, hosted in **Rome, Italy**, and online from **June 5–7, 2025**. As participants gather from across the globe, this hybrid event serves as a collaborative platform to share new findings, foster partnerships, and drive innovation in neurology. With this year's theme, "*Frontiers of Neurology: Bridging Science, Medicine, and Technology*," the conference celebrates the convergence of research, clinical practice, and emerging tools shaping the future of brain health.

The content within this abstract book reflects the depth and diversity of the discussions taking place during the event. Spanning fields such as neurodegeneration, neuroimmunology, neurotechnology, and rehabilitation, the presented work showcases the dedication of scientists and clinicians working to understand, treat, and prevent neurological disorders. Attendees will have the opportunity to engage with pioneering ideas and approaches that are redefining how we view and manage conditions affecting the nervous system.

We hope this event not only informs but also inspires. As you explore the abstracts and participate in sessions, may you find opportunities for connection, insight, and collaboration that continue beyond the conference halls. Your contributions and curiosity are what make Neurology 2025 a vibrant, impactful gathering.

ABOUT

CPD Accreditation

APPROVED PROVIDER #780643

Continuing Professional Development (CPD) credits are valuable for Neurology 2025 attendees as they provide recognition and validation of their ongoing learning and professional development. The number of CPD credits that can be earned is typically based on the number of sessions attended. You have an opportunity to avail 1 CPD credit for each hour of Attendance.

Some benefits of CPD credits include:

Career advancement: CPD credits demonstrate a commitment to ongoing learning and professional development, which can enhance one's reputation and increase chances of career advancement.

Maintenance of professional credentials: Many professions require a minimum number of CPD credits to maintain their certification or license.

Increased knowledge: Attending Neurology 2025 and earning CPD credits can help attendees stay current with the latest developments and advancements in their field.

Networking opportunities: Neurology Conference provide opportunities for attendees to network with peers and experts, expanding their professional network and building relationships with potential collaborators.

Note: Each conference attendee will receive 24+ CPD credits.

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KEYNOTE PRESENTATIONS

Antonio Carlo Galoforo[3*], Miriam Ciani[1], Catia Scassellati[2], Roberta Zanardini[1], Cristina Geroldi[4], Cristian Bonvicini[1]

[1]Molecular Markers Laboratory, Irccs Istituto Centro San Giovanni Di Dio Fatebenefratelli, Brescia, Italy

[2]Biological Psychiatry Unit, Irccs Istituto Centro San Giovanni Di Dio Fatebenefratelli, Brescia, Italy

[3]Oxygen-Ozone Therapy Scientific Society (Sioot), Gorle, Italy; University of Pavia, Pavia, Unicamillus Rome, Italy

[4]Alzheimer Unit, Irccs Istituto Centro San Giovanni Di Dio Fatebenefratelli, Brescia, Italy

Oxygen-ozone therapy and cognitive frailty: A non-pharmacological approach to potentially resolve immune and inflammatory dysfunctions

As the world's population ages, Cognitive Frailty (CF) is becoming one of the most serious health problems and elucidating its biological mechanisms along with prevention and treatments becomes increasingly important also considering the associated health costs. We thus performed a clinical randomized trial where CF subjects received a non-pharmacological therapy based on the regenerative properties of Ozone (O_3) known to act on immune/inflammation processes, strongly altered in CF.

A cohort of 75 patients was stratified in non-, mildly- or severely frail rate and treated with placebo, Oxygen (O_2) or O_2-O_3. The serum levels of 27 peculiar pro- and anti-inflammatory cytokines and chemokine cell signalling molecules were measured by using the Bio-Plex Pro

Biography

Prof. Antonio Carlo Galoforo graduated in Medicine and Surgery in Brescia, one of the most accredited experts in oxygen and ozone therapy, of which has been a member of the Scientific Society since 1992. Head of oxygen ozone therapy at excellent polyclinics where works daily and contact person for the Lombardy Region for hearings on the use of ozone in medicine and the environment. Speaker at various national and international conferences. Author of numerous national and international works, Master Professor in oxygen ozone therapy at the University of Pavia and Unicamillus International Medical University in Rome. Prof. Antonio Carlo Galoforo is a reference person accredited by the WHO for studies relating to the use of Ozone for Buruli Ulcer treatment; Founder and President of O3FORAFRICA Onlus; Head of Oxygen Ozone Therapy of Affidea Italia Centers and Bianalisi Group; Academician of European Academy for Economic and Cultural Relations. Winner of two Research Projects from Minister of Health.

Human Cytokine 27-plex immunoassay. The student's t-test and Analysis of Variance (ANOVA) followed by Tukey's post hoc test were used for comparison of means between the groups.

Preliminary analyses evidenced the implication, at different levels, of some molecules in relation to the frailty rate. Noteworthy, we observed modulations of immune (i.e interleukin, IL-9) and inflammation (i.e IL-1β) biomarkers at baseline and after treatment. Correlations between clinical CF profiles and peripheral levels of the considered biomarkers are ongoing in order to predict the response to O_2-O_3 therapy.

Although preliminary, these results confirm that the immune-inflammation systems are involved in the aetiopathogenetic mechanisms of CF, and that the related molecules could be potential therapeutic targets/biomarkers for the O_2-O_3 therapy. These data will further permit to validate a potential new non-pharmacological treatment approach for this condition.

Mrs Jacqueline Tuppen RN, BSc

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

Who cares …… for the carers

The House of Commons Inequalities in Dementia Care Debate 2024 highlighted that in the whole of the UK, the number of people with dementia is estimated at 850,000 and in England, around 540,000 people care for an individual with dementia.

These numbers are replicated around the world. For example, in Australia, estimates in 2023, suggested that there were at least 140,900 informal carers of people with dementia who live in the community.

The 2024 USA Alzheimer's facts and figures found an estimated 6.9 million Americans age 65 and older are living with Alzheimer's dementia. In 2023, 11.5 million family and other caregivers of people living with Alzheimer's or other dementias provided an estimated 18.4 billion hours of unpaid help.

An Alzheimer's society survey 2023 found that 1 in 10 provide unpaid care and 80% of unpaid carers are taking on the majority of caring responsibilities for loved ones living with dementia.

In the UK an Admiral Nurse survey in 2023, found that carers needed help and support with a variety of issues that included coordinating care, communicating with the variety of different specialists and support networks plus accessing information to help them understand the condition.

Carers are people in their own right, with their own physical and emotional needs, and with their own obligations. It is important to ensure that carers supporting someone living

Biography

Mrs Jacqueline Tuppen, graduated in 1997 from the University of Greenwich, London with a BSc Hons and Specialist Practitioner, worked with the local Community Mental Health team, ending as their Acting Community Services Manager. Mrs Jacqueline became an Admiral Nurse in 2008, and retired from the NHS in 201l, as an independent specialist nurse practitioner, started-COGS Club for people in the early stages of Dementia and continues to work for Dementia UK as a sessional Admiral Nurse on their Dementia Helpline. Mrs Jacqueline Tuppen published articles, presented at a variety of events in the UK, France, Eire and Italy.

with dementia can continue to live their own lives as fully as possible.

Whilst there are a variety of organizations that provide support for carers in the UK, this presentation will specifically discuss 2 interventions–one professional, non pharmacological intervention and a low cost community, non pharmacological intervention that support carers and people living with dementia in the UK. These interventions are recommended to the delegates as being new and adaptable to other countries carers needs.

Jonathan Eskenazi

Neurological Institute of Los Angeles, United States

Traumatic brain injury in 2025

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.

Biography

Dr. Jonathan Eskenazi, MD, is a well-rounded, double board-certified Neurologist and Brain Injury Specialist, born and raised in Lima, Peru. He completed both his undergraduate and medical education at Cayetano Heredia University in Lima. He went on to finish his preliminary internal medicine residency at Jewish Hospital of Cincinnati, Ohio, followed by a Neurology Residency at Cedars-Sinai Medical Center in Los Angeles, where he served as Chief Resident. Dr. Eskenazi holds double board certifications: one in Neurology from the Board of Neurology and Psychiatry, and another in Brain Injury Medicine from the American Board of Physical Medicine and Rehabilitation. He has also completed accredited courses and mini fellowships in clinical neurophysiology and neuro-stimulation for Parkinson's disease and epilepsy.

He currently serves as the Chief Medical Officer at the Neurological Institute of Los Angeles and is a member of the Stroke Team at Cedars-Sinai Medical Center. Dr. Eskenazi has served as Vice-Chair of the North American Neuromodulation Society Resident Fellowship Section and has been a

member of the Board of Directors for the California Neurology Society. He holds academic appointments at both the UCLA School of Medicine and Cedars-Sinai Medical Center.

Dr. Eskenazi often shares that neurology runs in his blood and has been a lifelong passion. Coming from three generations of neurologists, his upbringing deeply influenced his interest in neuroscience and continues to inspire his career. Pursuing a comprehensive approach to neurology, he strongly advocates for the development of a Multicenter Neuroscience Institute in Los Angeles, with a focus on serving underserved areas. He is currently recruiting patients for a prospective study on the use of transcranial magnetic stimulation in individuals with traumatic brain injury and depression. He has expressed that the most rewarding aspect of being a physician is the ability to listen, engage, and make a meaningful impact on patients' lives—both clinically and personally. A dedicated community advocate, he is deeply committed to improving patient outcomes and takes pride in contributing to positive changes in people's lives. Outside of his medical work, Dr. Eskenazi enjoys traveling and exploring different cultures through their food and traditions. He often reflects on an important life lesson: that people tend to cherish what they have worked hard to achieve far more than what comes easily. For him, the value of success lies in the dedication and effort it takes to earn it.

Ken Ware

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

Perception and individuality

The way we perceive our world directly influences our responses to it. There is significant variability in how individuals perceive and subjectively evaluate their environment, even among people experiencing the same conditions. In a healthy human system, perception and response to the environment are generally accurate. However, when subjective sensory perception errors occur, they can lead to motor response errors. The accumulation of these sensory perception errors can manifest in a variety of psychophysical diseases and disorders.

It is crucial to have reliable methods for measuring the acuity of a patient's environmental perception, particularly within stable environments where moment-to-moment measurements can be taken against a consistent and reliable backdrop. The detection of sensory perception errors under controlled conditions is interconnected with the psychophysical conditions of the individual across all scales. Assisting patients in correcting these errors can lead to the emergence of enhanced functional behaviours across various domains.

Neurophysics Therapy has proven highly effective in helping patients with complex neurological diseases and disorders. This approach facilitates the elimination of rogue patterns in sensory-motor communication, promoting the neurological network operations necessary for normal functioning within the dynamic landscape of the real world.

Keywords: Neurophysics Therapy, Perception, Sensory Perception Errors, Neurological Diseases and Disorders, Psychophysical.

Biography

Ken Ware was founder of Neurotricional Sciences Pty Ltd and Neurophysics Therapy and Research and 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 Ware is a Former Mr. Universe 1994, National powerlifting and Bodybuilding champion and record holder and published relative publications in 'Frontiers in Clinical Physiology' - 'World Journal of Neuroscience' – 'World Journal of Cardiovascular diseases'. Ken Ware 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.

Luiz Moutinho

University of Suffolk, United Kingdom

Futurey on neurology

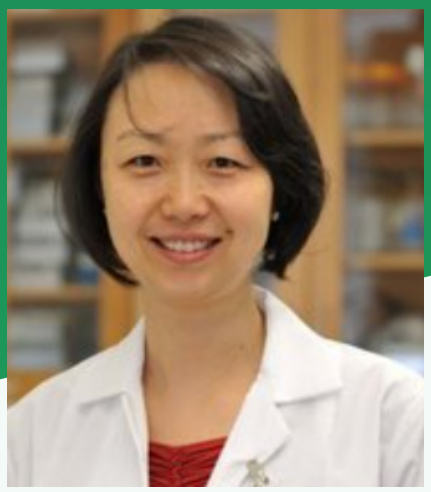
The presentation starts by addressing the fields of neurobiology, especially concentrating on the future of neurobiology and the future, connectivity and connectomics. The discussion will also include genetics and neurobiology. Artificial Intelligence and Machine Learning will be intertwined in the discussion. A new era for the neuroscience of social behaviour will be explored. Ethical considerations will also be highlighted. Conceptualisations of metabolomics and precision medicine will be advanced then as well as neurotrophic factors and phenotypic responses. Other key areas to be presented involve cognitive neuroscience, the future of neuropharmacology and neuropsychology. The human element in neuropsychology is debated. The interaction between technology, neuroscience and psychological theory is explored. Another important concept to be discussed is human cognition. The integration between cognitive science and artificial intelligence with be presented. All these areas are discussed and analysed in terms of developments and trends. Behavioural neuroscience is presented then and covered from its future to the ethical and societal implications. Neuroinformatics, Computational Neuroscience and Brain-inspired Computing are presented next. Other topics will also be dissected like, Neuroeducation, Human-like Intelligence, Brain-Computer Interfaces, Neurodiversity, Neural Interfaces and Augmentation, Brain Simulation and Deep Neural Networks.

Biography

Professor Luiz Moutinho (BA, MA, PhD, MAE, FCIM) is the Global Dean for Innovation at the Kotler Business School, as well as, the Visiting Professor of Marketing at Faculty of Arts, Business and Applied Social Science, Univ. of Suffolk, Ipswich, England, UK, at The Marketing School, Portugal, an Adjunct Professor of Marketing, GSB, FBE, University of the South Pacific, Suva, Fiji. In 2020, Prof Luiz Moutinho was elected as the member of The Academia Europaea. In 2017, 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, Luiz Moutinho was professor of BioMarketing and Futures Research at the DCU Business School, Dublin City University, Ireland. This was the first Chair in the world on both domains-BioMarketing and Futures Research. Previously, and for 20 years, appointed as the Foundation Chair of Marketing at the Adam Smith Business School, University of Glasgow, Scotland. Luiz Moutinho completed his PhD at the University of Sheffield in 1982 and 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. Also 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, was the director of the Doctoral Programmes at the Confederation of Scottish Business Schools and at the Cardiff Business School between 1993 and 1996. Prof Luiz 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. Prof Luiz Moutinho has another 4 associate editorships as well as being in the editorial boards of another 47 international academic journals. 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. Prof Luiz 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 and 171419 academic citations, the h-index of 59 and the i10-index of 166 (Google Scholar, April,27th, 2024).

Biography



Ming Guo

UCLA David Geffen School of Medicine, United States

Parkinson's disease, aging and mitochondria

Parkinson's Disease (PD) is the second most common neurodegenerative disorders. Mutations in PINK1 and PARKIN lead to inherited forms of PD. My lab was one of the first in the world to report the function of the PINK1 gene, and to show that PINK1 and parkin function in a common pathway to regulate mitochondrial integrity and quality. Mitochondrial morphology (dynamics) is controlled by two opposing actions, mitochondrial fusion, which is regulated by mitofusin, and mitochondria fission, which is controlled by drp1. We discovered that the PINK1/parkin pathway degrades mitofusin (inhibits mitochondrial fusion), and promotes Drp1 (mitochondrial fission). Our work provided compelling evidence that mitochondrial dysfunction underlies PD pathogenesis. In addition to mitofusin and Drp1, we have identified multiple other regulators of mitochondrial health that interact with PINK1/parkin in PD models. These include MUL1 (degrades Mitofusin in parallel of the PINK1/parkin pathway), VCP (degrades Mitofusin), Atg1 (increases Drp1 in addition to promoting autophagy) and clueless/CluH (a new regulator of Drp1). We have also developed the first transgenic model of mitochondrial DNA (mtDNA) heteroplasmy related to cellular aging and have identified factors that allow us to remove up to 95% damaged mtDNA. These genes and pathways may serve as therapeutic targets for PD and have the potential to reverse aging.

Robert B Slocum

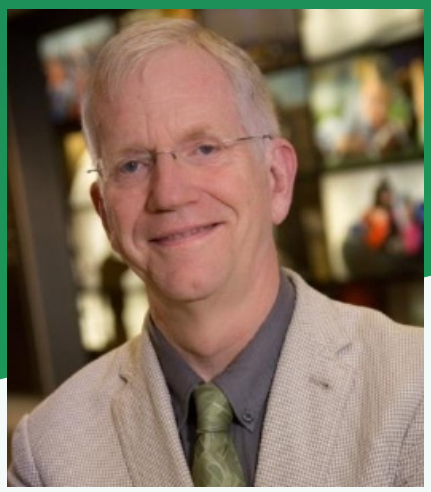
University of Kentucky HealthCare, United States

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

Patients with Psychogenic Non-Epileptic Seizures (PNES), also known as Functional Seizures (FS), have involuntary paroxysmal episodes that resemble epileptic seizures but without organic etiology. Patients with PNES are frequently misdiagnosed and mistreated for epileptic seizures. Accurate diagnosis may be delayed for many years. PNES 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. Many patients with PNES have a history of sexual, physical, or emotional abuse or other traumatic or overwhelming experiences. Some patients with PNES have been accused of faking symptoms or malingering, and stigmatized by health care providers, coworkers, family members, and others in society. Patients with PNES may have family histories of poor interpersonal communication and conflict resolution, with inherited codes of silence and shame concerning sensitive or traumatic subjects. PNES 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.

Narrative Medicine (NM) visits draw out the patient's narrative of illness or injury and treatment in the context of their entire life story. The focus is to discover topics and areas in the patient's narrative that the patient needs to explore. Narrative writing exercises have proven helpful for patients facing a variety of traumas and major stresses in

Biography



Robert B. Slocum is the Narrative Medicine Program Coordinator at University of Kentucky HealthCare and 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, also 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), 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 and the author, editor, or co-editor of 14 books, including a journal of reflections. His 36 articles have appeared in theological or medical journals and as book chapters, and has made presentations at more than two dozen theological and medical conferences. Robert B. Slocum has also published short fiction and poetry, interested in the clinical application of narrative, the significance of narrative for identity formation, and sees Narrative Medicine as a bridge between medical humanities and clinical practice.

situations similar to those faced by patients with PNES. NM sessions encourage patients to communicate more effectively about their unspeakable distress and reclaim their lives from the communication disorder of PNES. A case study illustrates NM applications to help a patient with PNES to communicate about a traumatic past that has been avoided and address psychogenic symptoms.

Sergei M. Danilov MD, PhD

Division of Pulmonary and Critical Care, Department of Medicine, University of Illinois at Chicago, IL 60612, USA

ACE-dependent Alzheimer's Disease (AD)

An analysis of 1200+ existing missense ACE mutations revealed that >400 are predicted to be damaging and led us to hypothesize that heterozygous carriers of these loss-of-function (LoF) ACE mutations (which result in low ACE levels) may be at risk for the development of late-onset Alzheimer's Disease (AD) [Danilov, 2024].

The 1st stage of this ACE-dependent AD project is characterization of blood ACE levels, catalytic properties, and conformations (ACE phenotyping) using a wide set of mAbs to ACE that were developed in our lab. We already have performed ACE phenotyping in >200 carriers of 80+ different ACE mutations and 200+ controls [Kryukova, Biomedicines, 2024, PloS One, 2024, unpublished]. We found that several of the relatively frequent AD-associated ACE mutations (present in at least 2% of the population) are truly damaging and, likely transport-deficient, resulting in plasma ACE levels only ~50% of controls. Some other AD-associated ACE mutations were not associated with a decrease in blood ACE levels, and likely do not affect ACE surface expression. Thus, their mechanism of association with AD is likely different, such as via catalytic changes. However, both these types of ACE mutations may result in reduced degradation of amyloid beta peptide Aβ42, an important component for amyloid deposition, and may pose a risk factor for the development of AD. Therefore, a systematic analysis of blood ACE levels in patients with ACE mutations has the potential to identify individuals at increased risk of late-onset AD.

Biography

Sergei M. Danilov, MD completed his PhD and postdoctoral studies from the National Cardiology Research Center, Moscow, Russia. Sergei M. Danilov is the Principal Investigator and Head of the laboratory of ACE biology in the Division of Pulmonary and Critical Care, (Department of Medicine in the University of Illinois at Chicago). Sergei M. Danilov's laboratory developed more than 40 mAbs to ACE and has published more than 200 papers on ACE biology and ACE immmunochemistry in highly respected journals and has been serving as an editorial board member of Biomedicines.

The 2nd stage of this project will include 1) Cell-based in vitro model (HEK cells transfected with cDNA of different ACE mutations) in order to find transport-deficient ACE mutations, which may be amenable to rescue of impaired trafficking of mutant ACE to the cell surface; 2) medico-genetic analysis of 50-100 families of carriers with the most damaging and transport-deficient ACE mutations. This stage will identify prospective candidates for a future limited clinical trial of preventive or therapeutic interventions to delay the development of ACE-dependent AD.

The 3rd stage of the project could be a limited clinical trial in individuals with several transport-deficient ACE mutations (starting with the most frequent damaging ACE mutation, Y215C) aiming to enhance mutant ACE protein traffic, as we previously demonstrated for the transport-deficient ACE mutation, Q1069R, using a combination of chemical and pharmacological chaperones and proteosome inhibitors [Danilov, PloS One, 2010].

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The foundation and architecture of Personalized & Precision Medicine (PPM) in clinical neurology: Towards curative and neurodegenerative disease-modifying treatment for multiple sclerosis

A new systems approach to diseased states and wellness result in a new branch in the healthcare services, namely, Personalized and Precision Medicine (PPM). PPM is an emerging approach to healthcare that aims to optimize medical treatment by tailoring it to the specific characteristics of each patient, person-at-risk and/or healthy person. PPM is based on the idea that individuals differ in many aspects, including their genetics, phenotype, lifestyle, and environment, and that these differences can have a significant impact on disease development and response to treatment. Therefore, by taking into account

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 awarded with MD. In 1985, maintained his PhD as a PhD student of Sechenov University and Institute of Medical Enzymology. In 2001, maintained his Doctor Degree at the National Institute of Immunology, Russia. From 1989 through 1995, was 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). At present, Dr. Sergey Suchkov, MD, PhD, is: Professor of the Russian University of Medicine, Moscow, Russia. Meber of the Russian Academy of Natural Sciences, Moscow, Russia. Dr. Suchkov is a member of the: New York Academy of Sciences, USA. American Chemical Society (ACS), USA; American Heart Association (AHA), USA; European Association for Medical Education (AMEE), Dundee, UK; EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU.

these individual differences, PPM can help to identify the most effective treatment strategies for each patient (canonical treatment) and/or person-at-risk (preventive and prophylactic approaches).

Neurodegenerative Disorders (NDDs) 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. And the considerations make it clear that PPM could transform clinical care in the field of NDDs, and could lead to a new treatment framework for NDDs diseases.

The potential benefits of PPM in the area of NDDs are significant. By identifying biomarkers and disease subtypes, PPM can help to diagnose the disease earlier and more accurately, as well as predict disease course and response to treatment. Furthermore, by developing targeted therapies, PPM can improve treatment efficacy and reduce the risk of adverse effects.

Precision diagnostics is a critical component of PPM in NDDs and is essential for the successful implementation of PPM in the field of NDDs. The identification of accurate and reliable biomarkers and imaging techniques can help to diagnose the disease earlier and more accurately, predict disease course and response to treatment, and monitor disease progression.

Targeted therapies represent a promising approach to the management of NDDs. The development of the therapies is guided by the identification of specific biomarkers and disease subtypes, and the use of targeted therapies can improve treatment efficacy and reduce the risk of adverse effects.

The implementation of PPM in the field of NDDs also requires addressing several challenges, including the standardization of diagnostic criteria and treatment guidelines, the development of affordable and accessible technologies and therapies, and the ethical and legal considerations of personalized treatment. PPM represents a promising approach to the management of NDDs, with the potential to improve diagnosis, prognosis, and treatment outcomes. Together, these data-driven insights enable the design of more precise therapeutic interventions in targeted patient populations. And future directions of PPM in the field of NDDs should aim to address these challenges and improve the integration of precision medicine in clinical neurology-related practice.

PPM-guided neurology stands at the threshold of a revolutionary transformation with the advent of PPM. And OMICS-driven and IT-supported potential to advance Personalized Precision Neurology (PPN) hinges on resolving core challenges across four pillars-models, data, feasibility/equity, and regulation/innovation-through concerted pursuit of targeted recommendations. The intricate tapestry of NDDs, 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 NDDs. 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 NDDs. And PPM in neurology holds the promise of advancing our understanding of NDDs and transforming healthcare by tailoring interventions to the unique needs of each patient and even pre-illness person-at-risk.

Stefano Sandrone PhD, MEd

Imperial College London, UK

Neuroscience education: From ‘learning by doing’ in the classroom to technology-enhance learning

In 1906, the Italian biologist and pathologist Camillo Golgi and the Spanish pathologist and histologist Santiago Ramón y Cajal shared the Nobel Prize for Medicine and Physiology ‘in recognition of their work on the structure of the nervous system’. They are considered the founders of neuroscience: For the first time, the world could visualise neurons in the brain. 118 years later, neuroscience has changed the landscape of bioscience research. It is an overall young discipline that is still growing at an incredible rate. From an educational perspective, it has fully embraced the digital revolution, which the pandemic has accelerated, and is attracting students from a high number of scientific backgrounds, from biology to medicine, from psychology to engineering, from philosophy to computing. But what will the future of neuroscience education look like? This talk will guide the audience through some of the most innovative aspects that will shape the future of neuroscience education, from active and distance learning to the metaverse, via mixed reality, gamification and technology-enhance learning.

Biography

Stefano Sandrone is an Italian neuroscientist and educationalist working at Imperial College London, won the Science Educator Award presented by the Society for Neuroscience (SfN) in 2019; the A. B. Baker Teacher Recognition Award (twice) from the American Academy of Neurology (AAN) in 2020 and 2024; the Miriam Friedman Ben-David Award from the Association for Medical Education in Europe (AMEE) in 2024; the Distinguished Neurology Teacher Award from the American Neurological Academy (ANA) in 2024. Stefano Sandrone wrote three books, including Nobel Life, in which he interviewed 24 Nobel Laureates about their life stories, advice for future generations, and what remains to be discovered.

Biography

Torbjörn C. Bäckström currently works at the Clinical Sciences, Umea Neurosteroid Research Center, Norrlands University Hospital. Torbjorn does research in sex and stress steroids effects in the brain, neurosteroids, neurology, mood disorders and appetite/ obesity. Their current project is 'Satiety' induction in polycystic ovarian syndrome and Prader Willi Syndrome.

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GABA-A receptor modulating steroid antagonists decrease GABAergic tone, improve memory impairment, attenuates fatigue, and decrease neuroinflammation

Gamma-Amino Butyric Acid (GABA) is the main inhibitory neurotransmitter in the brain and GABA-ergic transmission is important for the regulation of learning and memory. The progesterone metabolite Allopregnanolone (Allo) is a potent positive allosteric GABA-A Receptor Modulating Steroid (GAMS). In an Alzheimer transgenic mice model, Allo impairs memory and learning when given continuously at doses corresponding to low- grade stress. In humans, Allo impairs episodic memory and four years' treatment of postmenopausal women with a GAMS, medroxyprogesterone acetate, doubled the frequency of dementia. CNS manifestations of advanced liver disease such as Hepatic Encephalopathy (HE) and/or primary biliary cholangitis, impaired cognition and increased fatigue are associated with elevated Allo levels. A GABA-A Receptor Modulating Steroid Antagonist (GAMSA; golexanolone-GR3027) can block the adverse effects of GAMS, especially on the GABA-A receptor subtype, alpha5, which is related to memory and located in the hippocampus. In healthy rats, golexanolone mitigates GAMS-induced anesthesia and impaired memory and, in healthy adults, reverses GAMS-induced sedation and impaired saccadic eye velocity. In rat models of HE, golexanolone restores learning and motor coordination and reverses neuroinflammation in the cerebellum and hippocampus. In humans with advanced liver disease and evidence of covert HE, golexanolone improves vigilance, cognition and pathological EEG patterns. These findings suggest that golexanolone shows promise as treatment for impaired cognition.

Professor W S El Masri FRCS Ed, FRCP, PHF

Hon. Clinical Professor of Spinal Injuries (SI), Keele University

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Predictors spontaneous neurological recovery in patients with traumatic spinal cord injuries

The incidence of Traumatic Spinal Cord Injuries (TSCI) is small and ranges between 10-50/million population/ year. Prior to the second WW the great majority of patients died within two years of injury. Since the 2nd WW, due to the efforts of the pioneers who dedicated their professional lives to the field of TSCI, most well managed patients have been able to lead healthy, enjoyable, dignified, fulfilling, productive and often competitive lives. Moreover many exhibit significant degrees of neurological and functional recovery locally or below the level of their injury depending on the extent of sensory sparing vicinity and/or below the level of injury. To achieve this however requires in depth understanding of the systemic effects of cord damage on the neurological and functional outcomes as well as expert simultaneous management of the injury together with the potentially devastating medical and life-changing physical, psychological, social, financial, vocational, environmental & matrimonial consequences of the injury. These not only affect the patient but also the family members and close friends.

Biography

Prof W S El Masri FRCS Ed; FRCP is a Clinical Professor of Spinal Injuries-Keele University, Consultant Spinal Injuries–Hunters Moor Neurorehabilitation Centre Birmingham, Emeritus Consultant Surgeon in Spinal Injuries Robert Jones and Agnes Hunt Orth. Hospital Trained in the speciality of spinal injuries at Stoke Mandeville, Oxford, Guys Hospitals & the USA between 1971 and 1983. To date personally treated 10,000 patients with traumatic Spinal and spinal cord Injuries. Developed, and led the Midland Centre for Spinal Injuries (MCSI) between 1983 & 2014. Took responsibility for the management of the injured spine, the multisystem malfunction as well as the range of medical, non-medical and physical effects of cord injury in the acute, subacute, rehabilitation phases as well as in the long term. Lectured worldwide in developed and developing countries. Contributed to the literature with 140 publications. Documented the prognostic indicators of neurological recovery following Traumatic Spinal Cord Injuries. Demonstrated that with simultaneous Active Physiological Conservative Management of

TSCIs cause a multi-system physiological impairment and malfunction. This impairment is dynamic and affects the functioning of the various system of the body during the transitional stage between spinal areflexia and return of autonomic and spinal reflexes. During this transition the management of the various systems of the body requires modulation. Following the return of reflex activity the function of the various systems affected remains at risk of being unstable and erratic. This is due to the effects of the various inter-system autonomic and spinal reflex activity caused by the loss of inhibitory and coordinating influence of the higher centres. The combination of an unstable neuro-physiological impairment and sensory impairment/ loss can in inexperienced hands result in the development a wide range of potential complications and increase in disability. Some complications can further damage the injured and physiologically unstable spinal cord, cause neurological deterioration, delays or absence of recovery imposing further challenges to patients and clinicians. Fortunately with adequate Active Physio Conservative Management (APCM) of the injury and its medical effects almost all complications following TSCI can be prevented or diagnosed early and treated before further damage develops.

This necessitates a period of treatment in recumbence until the full return of the autonomic and spinal reflexes. This period ranges between four to eight weeks.

Neurological Recovery can be predicted early in the presence of spared sensory tracts and depending on the extent of the sparing when complications are prevented or diagnosed and treated early. This recovery has been repeatedly documented by various groups to occur irrespective of the radiological presentation on X-rays, CT & MRI since 1969. Unfortunately it has been rarely referred to in the literature in the last three decades.

The last three decades have witnessed increasing claims of benefits of a mechanical interventional approach focusing on the injured spine often at the expense of the adequacy of management of the medical and non-medical effects

the injury and all its medical effects neurological recovery occurs irrespective of the degree of Biomechanical Instability, Canal encroachment or Cord Compression. Introduced the concept of “Physiological Instability of the Injured Spinal Cord” Peer reviewer and on the Editorial Boards of a number of Journals Held the offices of: President of the International Spinal Cord Society, Chairman of the British Association of Spinal Cord Injury Specialists, Executive Member of the British Society of Rehabilitation Medicine. Founder Member and trustee of a number of charities that support Health Care professionals and patients. Raised about six million pounds from charity to rebuild and furnish the MCSI. Advisor to WHO ‘s International Perspectives on Spinal Cord Injury which was published in 2013 Member of the NICE Guideline Developing Group in spinal injuries. Obtained a number of awards including: The Medal of the International Spinal Cord Society, National Hospital Doctor Team Award for Innovation, 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 and the prestigious Paul Harris Fellowship of the Rotary Club.

of the cord injury. Claims that early interventions expedite the mobilisation, rehabilitation and discharge of patients; improve neurological outcomes or achieve both are currently influencing practice in both well-resourced and under-resourced countries. The risk of further mechanical and non-mechanical damage to neural during or after an intervention and during some of the related practices can be potentially detrimental to neurological and functional outcomes.

I will in this presentation discuss the extent of anticipated neurological recovery, the factors that influence its achievement, the role of clinical and radiological findings, and the role of surgery on the short, medium and long term.

Knowledge and experience in the provision of a fit for purpose and skilful management of patients with TSCI, thorough familiarity of the short, medium and long term outcomes of a holistic simultaneous Active Physiological Conservative Management of the spinal injury and the effects of neural tissue damage are paramount for a sound evaluation of outcomes of various surgical and non-surgical interventions. This necessitates training in dedicated centres that treat all aspects of TSCIs in large numbers and under one roof including the ability to offer both conservative and and surgical management of the injured spine.

Biography

Dr. Yong-Xiao Wang has been a Full Professor in Department of Molecular and Cellular Physiology at Albany Medical College since 2006. Dr. Wang obtained MD at Wannan Medical University, PhD at Fourth Military Medical University, and postdoctoral training at Technology University of Munich and University of Pennsylvania. Has made many important findings using complementary molecular, biochemical, physiological, and genetic approaches at the molecular, organelle, cellular, tissue and organism levels in animals and human samples, had numerous publications in Nature Commun (impact factor: 14.290), Antioxid Redox Signal (8.209), Proc Natl Acad Sci USA (9.432), Nature (34.480), Circ Res (9.214), and other highly peer-reviewed journals and academic books, and served as the editorial board member and/or section editor as well as the executive committee member and/or subcommittee chair for professional societies.

Yong-Xiao Wang

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Essential roles, mechanisms and consequences of vascular dementia

Vascular Contributions to Cognitive Impairment and Dementia (VCID) is a common neurodegenerative disease. This dementia includes all types of vascular dementia. It is caused by brain cerebral blood vessel dysfunctions. VCID has high morbidity and mortality. Diabetes is a leading factor in VCID. However, the essential roles, mechanisms and consequences of VCID are still largely unknown. Moreover, the current treatments for VCID are neither very specific nor effective. Dysfunctions of Cerebral Arteries (CAs) may cause blood hypoperfusion to the brain and then makes an important contribution in the initiation and progress of VCID. Perfusion of CAs is predominantly generated and controlled by contraction and relaxation of Smooth Muscle Cells (SMCs). These two cellular processes are fundamentally produced and regulated by cell calcium signaling. The cell calcium signaling is primarily determined by ion channels on the plasma membrane and Sarcoplasmic Reticulum (SR) membrane. Therefore, we have started to explore whether and which ion channels might be essential for diabetes-evoked VCID. Consistent with previous reports by us and other investigators, we have found that intraperitoneal injection of streptozotocin caused a large increase in blood glucose, leading to diabetes in mice. A series of our studies have also discovered that the diabetic mice had declined cognition, impaired memory, and increased anxiety, thereby exhibiting significant VCID. This diabetic vascular dementia might occur due to cerebral vasoconstriction and subsequent blood hypoperfusion, as revealed by Laser Speckle Imaging System. Diabetic cerebral vasoconstriction could result from increased

intracellular calcium concentration ([Ca2+]i) in CASMCs. Increased [Ca2+]i was attributed to the augmented Ca2+ release from the SR, the major intracellular Ca2+ store, which followed the hyperfunctional activity of type-2 ryanodine receptor (RyR2), the calcium release channel on the SR in CASMCs. Biochemical and genetic experiments indicated that the hyperfunction of RyR2 channel was a result of dissociation of FK506 binding protein 12.6 (FKBP12.6), an endogenous channel stabilizer (or inhibitor). In conclusion, our findings provide the first evidence that RyR2/FKBP12.6 dissociation exerts a novel essential role in the development of diabetes-caused VCID; presumably, specific pharmacological and genetic inhibition of RyR2 and/or stabilization of FKBP12.6 in vascular SMCs may become specific and effective treatment options for diabetic VCID.

Zhenhuan LIU

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

Neuroimaging by evaluation nerverenovate and europlasticity of acupuncture 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

Biography

Zhenhuan LIU professor of pediatrics, Pediatric acupuncturist Ph.D. tutor. 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 Profassor of Chinese University of Hong Kong in recent 10 years. Zhenhuan LIU is most famous pediatric neurological and rehabilitation specialists in integrated traditional Chinese and Western medicine in China. Zhenhuan LIU has edited 10 books and published 268 papers in international and Chinese medical journals.

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 1 year 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.

BOOK OF ABSTRACTS

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ORAL PRESENTATIONS

Veronicah Akankunda
Golden Age Elderly Homes Kampala, Uganda

Personalised nutrition plans and recreational activities in helping patients with neurological disorders in care homes/assisted living facilities

A natural nutrition plan for elderly people with neurological disorders should focus on whole, nutrient-dense foods that support brain health and address specific needs.

Unprocessed foods that promote overall health and address age-related concerns. Remember to consult with a healthcare professional or registered dietitian to create a personalized nutrition plan in case you are doing it on an individual basis.

A Natural Nutrition Plan For People With Neurological Disorders

- **Omega-3 Rich Foods:** Fatty fish, nuts, and seeds to reduce inflammation.
- **Antioxidant-Rich Foods:** Berries, leafy greens, and other fruits and vegetables to combat oxidative stress.
- **Magnesium and Potassium-Rich Foods:** Dark leafy greens, nuts, and whole grains to support neuronal function.
- **Vitamin B-Rich Foods:** Whole grains, lean meats, and fish to support neurotransmitter production.
- **Probiotic-Rich Foods:** Fermented foods like yogurt, kefir, and kimchi to support gut-brain health.
- **Healthy Fats:** Avocados, nuts, and seeds to support brain function.
- **Stay Hydrated:** Adequate water intake to maintain cognitive function. Here are some guidelines:
- Emphasize whole grains, fruits, vegetables, lean proteins, and healthy fats.
- Include calcium and vitamin D-rich foods for bone health.
- Choose omega-3 fatty acid-rich foods for heart health.
- Select fibre-rich foods for digestive health.
- Limit processed, sugary, and high-sodium foods.
- Stay hydrated with plenty of water.
- Consider supplements like vitamin B12, vitamin D, and probiotics.

Some Beneficial Foods For Elderly People Include:

- Leafy greens (spinach, kale)

- Berries (blueberries, strawberries)
- Nuts and seeds (almonds, chia seeds)
- Fatty fish (salmon, tuna)
- Sweet potatoes
- Legumes (lentils, chickpeas)
- Whole grains (brown rice, quinoa)
- Avocados
- Fermented foods (yogurt, kimchi)
- Herbal teas (green tea, peppermint)

Specific Considerations For Common Neurological Disorders:

- **Alzheimer's Disease:** Emphasize omega-3s, antioxidants, and B vitamins.
- **Parkinson's Disease:** Focus on magnesium, potassium, and vitamin B6.
- **Multiple Sclerosis:** Include omega-3s, vitamin D, and probiotics.
- **Stroke and Cerebral Vasculature Disorders:** Emphasize omega-3s, antioxidants, and potassium-rich foods.

Remember to consult with a healthcare professional or registered dietitian to create a personalized nutrition plan.

Recreational activities can greatly benefit patients with neurological disorders, promoting physical, emotional, and cognitive well-being. Here are some suitable activities:

- Art therapy (painting, drawing, colouring)
- Music therapy (listening, singing, playing instruments)
- Gardening
- Yoga or tai chi (modified for accessibility)
- Walking or wheelchair-friendly exercise
- Board games and puzzles (cognitive stimulation)
- Reading (adapted formats like audiobooks or large print)
- Creative writing or journaling
- Photography
- Cooking or baking (simple recipes)
- Animal-assisted therapy (interacting with pets)
- Virtual reality experiences
- Sensory stimulation (e.g., aromatherapy, texture exploration)
- Group activities (socialization, support groups)
- Adaptive sports (e.g., wheelchair basketball, adaptive tennis)

Considerations:

- Consult healthcare professionals before starting new activities.
- Tailor activities to individual abilities and interests.

- Ensure accessibility and safety.
- Provide necessary assistance and support.
- Encourage social interaction and community engagement.
- Monitor progress and adjust activities as needed.
- Incorporate cognitive stimulation and memory exercises.
- Offer relaxation techniques (e.g., meditation, deep breathing).

Benefits:

- Improved mood and reduced stress
- Enhanced cognitive function
- Increased mobility and flexibility
- Boosted self-esteem and confidence
- Socialization and community connection
- Emotional expression and creativity
- Pain management and relaxation
- Overall quality of life improvement

Remember to prioritize patient comfort, safety, and enjoyment when selecting recreational activities.

Biography

Veronicah Akankunda is a Ugandan Gerontologist, Neuro Researcher, social entrepreneur and advocate for elderly care. Also the Founder and CEO of Golden Age Elderly Homes (GAEH), a pioneering organization providing holistic care to seniors in Uganda as well as the Geriatric Academy that equips students with Nursing skills in Elderly care. Veronicah is a passionate geriatric care specialist, visionary leader with expertise in Gerontology, healthcare management, and social work for over 10 years. Her dedication to elderly care is inspiring. The engaging presentations, public lectures and compassionate care to seniors inspire audiences to action. Apart from Gerontology consultancy she has innovated age-friendly living spaces. Golden Age Elderly Homes being the first care home in the country is a beacon of hope for Uganda's seniors offering a comprehensive range of services designed to cater to the diverse needs of the elderly population. From the Geriatric Training Academy that equips students with Nursing skills in Elderly care, to Mobility Aides, Personal Care, Elderly Nutrition, and Rehabilitation, the organization stands as a one-stop destination for elderly care support. The unique blend of home services, including Physiotherapy and Massage, Adult day care centre sets Golden Age Elderly Homes apart, providing a holistic approach to caregiving as seniors age gracefully with Dignity in the comfort of their homes. Veronicah's work focuses on addressing the Psychosocial, emotional, and healthcare needs of the elderly, promoting dignity, and challenging age-related discrimination. Has gained recognition for her efforts to improve elderly care in Uganda and Africa. Veronicah’s dedication to enhancing the lives of older adults has earned respect and admiration internationally. Veronicah work continues to inspire positive change and promote a culture of care and inclusivity for all ages. And projects have Improved lives of countless elderly individuals and their families, Raised awareness about Geriatric care and age-related issues. Inspired a new generation of social entrepreneurs and caregivers, Contributed to policy changes and advocacy for elderly rights in Uganda. Veronicah’s selflessness, compassion, and innovative spirit makes a true champion for the elderly and a role model for social entrepreneurship. Has won numerous Awards for Excellency in Palliative care, Health entrepreneur, Innovation and Entrepreneurship, Research in Geriatric Care, Neurology and Neurological disorders has been internationally published and continues to impact society. Golden Age Elderly Homes has left an indelible mark on Ugandan communities. The organization has provided geriatric care to over 1962 elderly individuals, conducted more than 134 community health camps, and trained over 350 home care-based carers. The impact extends beyond physical care, touching on community health and general well-being.

Alberto Cliquet Junior

Department of Orthopedics, Rheumatology and Traumatology/UNICAMP, Campinas, SP., Brazil

Scientific-technological integration towards the clinical rehabilitation of spinal cord injured individuals

Individuals with spinal cord injury rarely have access to innovative technology. The work presented here has allowed sensorimotor recovery to paraplegics and tetraplegics, in addition to the production of cutting-edge technology for control of upper and lower limbs. Neuromuscular Electrical Stimulation provides a unique opportunity to recover bipedal walking. Scientific findings and clinical interventions, such as voluntary restoration of patients' locomotion, showed that technology associated with the gait task (nervous system learns through repetition) makes neuroplasticity possible. For upper limbs of tetraplegics, our protocols allow functional gains through a wearable system, including virtual reality. We have also implemented protocols towards improving clinical outcomes. Absence of the sympathetic nervous system causes autonomic dysreflexia, as tetraplegics, generally young adults, are on the path to atherosclerosis, where the carotid is thickens by hypertensive peaks generated by the comorbidity. Gait and physical activity can reduce that thickness. Our protocols for spasticity assessment do show improvements with electrical stimulation. Rehabilitation protocols with innovative technology can allow greater longevity for these patients, minimizing the clinical complications inherent to the pathology.

Biography

Alberto Cliquet Jr., PhD (Univ. of Strathclyde/Glasgow). Full Professor, Dept. of Orthopedics, Rheumatology and Traumatology. Coordinator of The Postgraduate Course in Surgical Sciences, FCM-UNICAMP. Coordinator of the Spinal Cord Injury Outpatient Clinic (University Hospital). Fellow, The Royal Society of Medicine, London. Full Professor, Dept. of Electrical and Computer Engineering, EESC-USP.

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Transcranial pulse stimulation for treatment-resistant depression: A case series

Depression, a prevalent condition, has reached unprecedented levels in certain countries, such as Germany, as indicated by the most recent data. Depending on the severity of the condition and the patient's individual preferences, different treatment options are available. However, treatment-resistant depression can pose significant challenges for both patients and healthcare providers. Transcranial Pulse Stimulation (TPS), an alternative Non-Invasive Brain Stimulation (NIBS) method based on shock waves, has been available for several years and is CE-certified for the treatment of Alzheimer's dementia. This innovation facilitates precise and non-invasive modulation of subcortical brain regions, previously requiring surgical intervention. The objective of this case series is to examine the safety and effectiveness of TPS in patients diagnosed with treatment-resistant depression. A total of five patients (gender ratio female to male 3:2) who met the criteria for treatment-resistant depression underwent a total of six treatments within a 14-day period. Each session consisted of 6,000 pulses at an energy level of 0.25 millijoules per millimeter and a frequency of 4 Hertz. The application of pulses in the frontal, parietal, and precuneus regions was facilitated by neuro-navigation, based on individual MRI scans. Furthermore, the shell region of the nucleus accumbens was targeted with 300 pulses on both sides due to its involvement in the pathophysiology of depression. The Beck Depression Inventory (BDI-II) was utilized to evaluate the severity of depressive symptoms prior to the initial and subsequent treatment sessions. Apart from a temporary feeling of pressure in the area of the temples in one patient, none of the patients experienced any side effects during and after the treatment. A reduction in the BDI-II total score was observed in all patients in the pre-post comparison.

Biography

Dr. Günes studied Medicine at the University of Dusseldorf, Cologne, Zurich and Sydney and graduated as MD in 2012. Received Board certification for neurology after completing fellowship at Alfried Krupp Hospital, Essen, Germany in 2018 and received Doctoral degree in the same institution researching on vasculary disease. After completing psychiatric fellowship at University Hospital Dusseldorf, received Board certification for Psychiatry and Psychotherapy in 2022. Dr. Günes published research articles, wrote chapters for neurological textbooks (SOPs in Neurology) and held oral speeches during national (DGN, DGPPN)/international neurological (EAN) and psychiatric (EPA) Congresses.

Carlos Gutierrez Merino

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A critical appraisal of strategies to afford protection against neurotoxic amyloid β peptides-induced brain degeneration

The 2022 world Alzheimer's report pointed out the importance of early detection and diagnosis of Alzheimer's Disease (AD), as nearly 75% of individuals with dementia are not diagnosed globally. The histopathological hallmarks of AD are the extracellular amyloid-β (Aβ) plaques and intracellular neurofibrillary tangles. The neurotoxic Aβ peptide Aβ (1-42), which is found in higher concentrations in the brain of AD patients and associated with Aβ plaques, is produced from the Amyloid Precursor Protein (APP) by the called Amyloidogenic Pathway through the sequential activity of β-site amyloid precursor protein cleaving enzyme 1 (BACE1) and γ-secretase. An enhanced activity of BACE1 and shift towards the amyloidogenic pathway of APP processing has been reported to be linked to several factors known to foster the neurodegeneration in AD-affected brains, like iron dyshomeostasis, brain oxidative stress, hypercholesterolemia, and brain hypoxia. Nevertheless, nearly all BACE1 inhibitors used as candidate therapeutic agents in AD have failed in later phases of clinical trials, due to safety and/or efficacy issues, and others were discontinued early. Thus, exploration of alternate approaches to reduce Aβ toxicity seems a timely issue. Indeed, it has been noted recently that some phytochemicals that inhibit Aβ-induced neurotoxicity, Aβ self-aggregation, and acetylcholinesterase enzyme activity show anti-AD effects. Another novel potential therapeutic target for AD is the attenuation of signaling pathways leading to Aβ overproduction. The shortest Aβ (1-42)-derived peptide that retains the toxicity of the full-length peptide is Aβ (25-35), and this experimental observation is of particular relevance for the identification of peptides that can antagonize the actions of neurotoxic Aβ peptides. Cumulative experimental evidences show that intracellular Aβ oligomers are linked to AD pathogenesis and are the cause of neuronal damage, moreover, the metabolic and neurotoxic effects of Aβ (1-42) have been linked with neuronal uptake of Aβ oligomers and the subsequent rise of their intracellular concentration. As anti-Aβ antibodies are expected to trap only extracellular Aβ, this could, at least in part, account for the limited and partial protection reported for aducanumab treatment in AD. The studies performed in vitro, ex-vivo with cellular cultures and in vivo with animal models of AD open new perspectives for the clinical management of AD, and these are the main focus of this presentation. Also, the current limitations and need of further research studies for the translational application of these findings will be briefly discussed.

Biography

Dr. C. Gutierrez-Merino received the PhD degree in 1977 at the Universidad Complutense of Madrid. Professor of Biochemistry and Molecular Biology of the Universidad de Extremadura (1989-2022) and from 2023 Honorary Investigator of the Instituto de Biomarcadores de Patologías Moleculares of the Universidad de Extremadura, Badajoz, Spain. Member of the executive committee of the Spanish societies of Biochemistry (1988-1992) and Biophysics (1994-1998). Author of around 200 scientific publications, largely published in JCR-indexed journals. Present research interests: molecular mechanisms of neurodegeneration induced by amyloid-β peptides and neurotoxins, and neuroprotection agents.

Changqing Zhao[1*], Xiaojia Zhu[1,2], Hongying Duan[1,2], Yanjie Wang[1,2], Xiaoke Li[2,3], Fengli Cheng[1], Luyao Wang[1,2], Haoxiang Zhang[1,2], Xueping Qi[1], Sirui Fu[1,2], Danni Xv[1,2], Yan Liu[1,2]

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Neuromedin U induces airway goblet cell hypersecretion through eosinophil-derived interleukin-1α in allergic rhinitis

Background: Neuropeptide Neuromedin U (NMU) is elevated in Allergic Rhinitis (AR) and has been linked to eosinophil activation, but its role in Goblet Cell (GC) secretion during AR remains unclear. This study aimed to investigate whether NMU induces GC hypersecretion.

Methods: Nasal mucosal tissue samples were collected from healthy controls, AR patients, and those with Non-Allergic Rhinitis with Eosinophilia Syndrome (NARES). The correlation between NMU expression and eosinophil peroxidase and MUC5AC was assessed. In vivo, eosinophil proportions in nasal and bronchoalveolar lavage fluids were quantified by flow cytometry, and GC numbers were measured using Alcian blue-periodic acid Schiff staining. Sensory nerve ablation in AR models was performed to assess mucus expression. A co-culture model of eosinophils and airway epithelial cells was used to examine MUC5AC and MUC5B secretion. After NMU induction in eosinophils, transcriptome sequencing was performed, and CellChat was used to analyze intercellular communication.

Results: NMU expression was higher in NARES and AR samples than in controls, correlating with an increased GC proportion in the inferior turbinate. In vivo, NMU stimulation increased eosinophil and GC proportions, while NMU inhibition attenuated these effects. Sensory nerve ablation reduced mucin and Nmu mRNA levels in nasal mucosa. In vitro, NMU increased MUC5AC and MUC5B secretion in co-cultured eosinophils and airway epithelial cells, but not in epithelial cells only. Transcriptomic analysis and CellChat revealed eosinophil-derived-IL-1α and IL-1R modulation on GCs, contributing to GC hypersecretion.

Conclusions: NMU increased MUC5AC secretion from GC through the activation of eosinophil-induced IL-1α. Trigeminal nerve ablation affected NMU expression and MUC5AC hypersecretion, highlighting a neuroimmune axis in AR pathogenesis.

Keywords: Allergic Rhinitis, Neuroimmune, Eosinophil, Goblet Cell Hypersecretion, Mucin, Neuromedin U.

Biography

Prof. Changqing Zhao received his Ph.D. from Xiangya School of Medicine, Hunan Medical University in 1995. As a leading expert in otorhinolaryngology, serves as a Ph.D. supervisor, NSFC grant review expert, and former National Committee member of the Chinese Society of Otorhinolaryngology-Head and Neck Surgery. Prof. Changqing pioneered the concept of the “nose-brain axis” to elucidate neuroimmune mechanisms in upper airway hyperresponsiveness, gaining international recognition. Changqing Zhao work includes 300+ publications, with 46 high-impact SCI papers (e.g., JACI, Allergy), and two translated medical monographs (1 million words). And continues to shape therapeutic strategies for allergic respiratory diseases through translational neuroimmunology research.

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Systemic inflammation and neuroinflammation in alzheimer's disease: An unexplored relationship

Growing evidence suggests a link between systemic inflammation and cognitive decline. Studies in cognitively healthy individuals have demonstrated an association between low-grade systemic inflammation, and accelerated cognitive decline. Furthermore, some research indicates a potential role for systemic inflammation in the pathogenesis of Alzheimer's Disease (AD). Preclinical models exploited for the investigation of this link have shown how acute systemic inflammation can exacerbate neurodegenerative processes in the context of existing neurodegeneration, leading to accelerated disease progression. While the precise mechanisms remain under investigation, it is now clear that systemic inflammation, both acute and chronic, can trigger a cascade of events, which, in turn, can influence brain microenvironment. Given the mounting evidence linking various systemic inflammatory conditions to AD risk, we hypothesized that acute systemic inflammation in the pre-symptomatic phase would influence in the long term the timing and the degree of cognitive decline. One of the risk factors associated to a higher prevalence of AD is the diagnosis of inflammatory bowel disease as described by different epidemiological and preclinical studies, but the mechanisms at the basis of this link are still unclear. To help bridge this gap, we induced acute colitis by Dextran Sulfate Sodium (DSS) administration in young, presymptomatic/preplaque Tg2576 mice (carrying the Swesidish APP mutation) and in C57BL6 wild type mice, to model human IBD and evaluate the effects of this acute peripheral inflammation on the age of cognitive symptoms onset and/or on its worsening. Results indicated that gut inflammation in 3 months old Tg2576 mice anticipated the learning and memory impairment, which usually appears in Tg2576 mice starting from 6 months of age. Gut microbiota/microbiome dysbiosis, as well as peripheral inflammation, is thought to have an active role in the natural course of AD, acting way before the onset of the first clinical symptoms. Following DSS-induced gut inflammation, we observed an altered firmicutes/bacterioides ratio in Tg2576 but not in WT, suggesting an anticipated aging of the microbiota profile in Tg2576 recovered from gut inflammation, in line with what observed in IBD patients. Finally, we focused on hippocampal tissue as it is the main player in the symptomatology observed in behavioral tests and we found significantly altered inflammation- and neuroinflammation-related parameters in 3-months old Tg2576, with a rapid progression towards a worsening of the scenario at the age of 5.5 months. Our results points towards an astroglial dysfunction as a possible contributor for AD onset, with a functional impairment in astrocyte population as a consequence to the gut inflammation. This approach can be a valuable tool to dissect the impact of known or possible risk factors for Alzheimer's disease, helping in the understanding of the intricate interplay

between peripheral inflammation, neuroinflammation and disease onset, which is crucial for the unveiling of the heterogeneity of AD with the aim to develop effective therapeutic strategies.

Biography

Dr. Quadalti graduated in Biotechnology in 2013 at the University of Bologna and received her Ph.D. in 2017 in the same institution with a project on the generation of animal models for human neurodegenerative diseases. As a postdoctoral researcher, Dr. Quadalti worked focused on the identification of novel clinical biomarkers for the early diagnosis of neurodegenerative diseases, such as Alzheimer's and Parkinson's. Dr. Quadalti is currently engaged in a research project funded by the PNRR, focusing on the study of systems biology of pre-clinical and clinical models of neuro-functional phenotypes, towards the identification of new multidimensional biomarkers.

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Determination of blood biomarkers for dynamic monitoring-A promising method for evaluating the effectiveness of alzheimer's disease treatment

Belarusian researchers have developed an algorithm for the clinical and laboratory diagnosis of dementia of various origins, based on the use of clinical indicators and laboratory studies of the concentration of biomarkers of the neurodegenerative process in the blood: β-amyloid (Aß40, Aß42), phosphorylated tau protein (f-tau protein). [Astashonok A. et al., 2023]. The levels of these biomarkers in cerebrospinal fluid and blood were compared, correlations have been identified, the normative values in blood were determined. The minimally invasive method of tau protein and amyloid protein analysis can be widely used both in clinical practice and in research activities. Taking into account the clinical symptoms, standard laboratory data and the level of biomarkers, the patient is prescribed personalized therapy and dynamic monitoring is carried out. The effectiveness of the method is illustrated by a clinical example.

The patient is a male, 74 years old, with decreased memory, household skills, lethargy, and sleep disorders for 2 years. The patient's older brother suffered from dementia.

In neurological status, without focal symptoms. Testing showed: MMSE: 16 points–moderate dementia, FAB: 6 points–frontal type dementia.

QEEG: Diffuse disturbances of cortical rhythm, low-amplitude beta activity prevails, peak alpha frequency is reduced to 8.9 Hz.

MRI–grade 1 leukoareosis on the Fasekas scale, 4 points. According to the scale of atrophy of the medial sections of the temporal lobes.

The content of neurodegenerative markers in the blood: Aß42–18 pg/ml, Aß40–14 pg/ml, f-tau protein-32 pg/ml.

According to the results of the examination, dementia in Alzheimer's disease was diagnosed, moderate. A course of cerebrolysin 20 ml intravenously daily No. 20 is prescribed.

After the course of treatment: Improvement in dressing, toilet use; MMSE score increased to 17 points; serum Aß42 content is 18 pg/ml, Aß40-15 pg/ml, f-tau protein-29 pg/ml.

Changes in the values of neurodegeneration markers in the blood serum during a dynamic examination of the patient showed a complete correlation with a decrease in clinical symptoms. In the future, a minimally invasive method for monitoring the content of f-tau protein and amyloid proteins in the blood can be used not only as a prognostic sign of the development of the pathological process, but also possibly for large-scale screening studies.

Biography

Dakukina Tatsiana completed a PhD at the age of 35 years from State Medical University, Belarus and Professor of the Department of Neurology at the State Medical University, Minsk, Belarus. Dakukina Tatsiana has over 200 publications in different fields of neurology: Epileptology, neurophysiology, pharmacogenetic testing, neurodegenerative diseases, pediatric autism and been serving as an editorial board member of several reputed journals.

Nurul Sulimai, Jason Brown, David Lominadze*

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Vascular effects during neuroinflammation

Many neuroinflammatory diseases are accompanied by neurodegeneration and memory deficits. In a substantial number of these diseases, deleterious causes originating in vasculature play a significant role in the development and/or progression of morbidity. Traumatic Brain Injury (TBI) can be considered an example of such a disease. Inflammation that is associated with TBI, results in increased blood content of Fibrinogen (Fg), called Hyperfibrinogenemia (HFg). During mild-to-moderate TBI, despite no noticeable microvascular ruptures about seven days after injury insult, Fg extravasation was still detectable 14 days after injury. This vascular effect of Fg extravasation resulted in its deposition in the extravascular space in the vicinity of astrocytes and neurons. Similar to our finding of a direct association of Fg with astrocytes and neurons, we discovered that the resultant activation of astrocytes, in conjunction with its direct interaction with neurons resulted in neurodegeneration and a reduction of short-term memory. In this study, we will present data indicating mechanisms involved in the interaction of Fg with astrocytes and neurons, we will show the role of Fg in the generation of reactive oxygen species which contributes to neurodegeneration, and we will document the specific role of Fg in the reduction of short-term memory. Overall, we will show that the HFg that accompanies neuroinflammation leads to increased cerebrovascular permeability via caveolar transcytosis principally, enhances deposition of Fg in extravascular space, and results in neurodegeneration. All these effects directly indicate a significant role of vascular-cognitive impairment during neuroinflammatory diseases such as TBI, Alzheimer's disease, and others that are accompanied by HFg.

Biography

Dr. David Lominadze studied biophysics at the Tbilisi State University (Georgia) graduating in 1979 and joined the Laboratory of Physiology and Pathology of Cerebral Circulation at the Iv.Beritashvili Institute of Physiology, Georgian Academy of Sciences. Dr. David received Ph.D. degree in 1990 at the same institution. In 1992-1999, was an Adjunct Assistant Professor at the Center for Applied Microcirculatory Research, University of Louisville (UofL), USA. Since 2000 Dr. David was a faculty at the Department of Physiology, UofL, and was tenured in 2011. Since 2019, been a professor of surgery at the University of South Florida.

David Sperbeck
Private Practice, United States

Can specific personality traits serve as protective factors against age-related cognitive decline? A longitudinal study of experiential openness and executive functions in healthy older adults

Background: General studies of neurocognitive processing across the lifespan have demonstrated gradual declines among healthy adults in the domains of memory, problem-solving, executive functioning, and processing speed. However, a number of specific factors have been identified which account for significant differences between individuals in their capacity to compensate for and/or otherwise decrease the magnitude of this neurocognitive decline. Understanding and recognizing these individual differences in critical areas of cognitive processing is likely to be essential to improving the functional abilities and quality of life for older persons.

Experiential Openness (EO), first proposed by McCrae and Costa (1978, 1987) in their five-factor model of personality, has been found to be positively related to enhanced autobiographical memory recall and reminiscing activity (Sperbeck and Whitbourne, 1982, 1985). Additionally, Ihle, Zuber, Gouveia et. al. (2019) found that EO adults engaged in more leisure time activities which served to mediate smaller cognitive declines in executive functioning relative to their Experientially Closed (EC) counterparts.

Participants: The current study was designed to test volunteer participants' executive functioning, short-term memory, and incidental memory every five years for 25 years. The average age at the onset of the study was 55.57 years for the initial 175 participants who all completed the Experience Inventory. Over 25 years, participant attrition resulted in a total of 70 (32 Experientially Open and 38 Experientially Closed) participants completing all six testing sessions scheduled five years apart (circa 1995, 2000, 2005, 2010, 2015, 2020). Study participants were well-educated (mean years of education=14.75 years) and screened for physical health. Participants with medical evidence of (prodromal) neurological disease were excluded from the study.

Method: Participants agreed to complete a brief (1 hour) battery of neurocognitive tests once every five years for 25 years. The test battery consisted of the Halstead Category Test, Stroop Test, Wechsler Digit Span Test, and the Bender Visual Motor Gestalt Recall Test.

Results: Experientially Open participants displayed statistically significant (serial Mann-Whitney U Tests) preservation of neurocognitive test performance in all areas of executive and memory functioning between ages 55-75 years relative to their Experientially Closed counterparts. Even after age 75, declines were observable with EO participants but significantly less severe and precipitous than with EC

participants. The most significant skill set preserved was noted in incidental memory (BVMGT) and the Interference Trial of the Stroop Test.

Discussion: Experiential Openness may serve a protective role in the preservation of neurocognition in the healthy aging population. The tests administered in this study likely reflected neuroanatomical correlates in the prefrontal cortex and hippocampal regions of the brain. As such, increased physical and ideational activities of a novel nature characteristic of Experiential Open adults may contribute to and enhance the neurogenesis process and lead to protection and preservation of neurocognitive capacities, and ultimately the postponement of debilitating cognitive decline.

Biography

David J. Sperbeck earned his Ph.D. from the University of Rochester in 1982 after completing neuropsychological internships at Monroe Community Hospital (Rochester, NY) and the Veterans' Administration Medical Center (Bath, NY). Dr. Sperbeck thereafter was appointed to the position of forensic psychologist and neuropsychologist at the Alaska Psychiatric Institute in Anchorage, Alaska where he conducted more than 2000 forensic psychological examinations between 1982-2005. Dr. Sperbeck held the position of Clinical Professor of Psychiatry & Behavioral Sciences at the University of Washington School of Medicine from 1985-2020 and served at the Director of Psychological Services at North Star Behavioral Health Hospital from 2005-2019. David J. Sperbeck is a Fellow of the National Academy of Neuropsychology and has authored more than 150 papers and/or journal articles over the past 45 years in the areas of forensic psychology, suicide prevention, and neurocognition across the lifespan.

Di Brina Carlo[1*] MD, PhD, Child Neuropsychiatrist; Cerni Tania[2]

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The absolute beginner. Right-and left hand process-based measures in handwriting

Fine motor performance across ages is traditionally assessed through handwriting and drawing, focusing on both the final product and the kinematic aspects of the movement (Blank et al., 2019; Smits-Engelsman et al., 2001; van Drempt et al., 2011). In kinematic analysis, tablet-based dynamic parameters, such as the number of velocity inversions and movement fluency (e.g., speed, frequency), have revealed important characteristics of handwriting development and maturation, particularly in early writers (Rueckriegel et al., 2008). When it comes to product quality, it is often assessed through human judgment (Hamstra-Bletz, De Bie, & Den Brinker, 1987). A notable attempt to digitize this evaluation is the use of the Dynamic Time Warping (DTW) method to assess the consistency of letter formation (Di Brina et al., 2008). Building on these insights, handwriting, whether evaluated for quality or kinematics, is also significantly influenced by cognitive factors such as spelling and motor sequence learning, which shape kinematic performance and overall writing efficiency (e.g., Kandel et al., 2006; Fitjar et al., 2021; Meulenbroek & van Galen, 1989).

In this study, we aimed to assess fine motor skills by analyzing computerized kinematic and quality variables, isolating the impact of cognitive factors. To achieve this, we compared the Dominant Hand (DH) and Non-Dominant Hand (NDH) in a group of 26 adults (4 males, mean age 28.8, range: 22-35). The rationale was that the DH serves as an expert in fine motor movements (Annett, 1992), while the NDH represents a non-expert hand, although still operating with a memorized motor pattern. Participants performed three letter formation tasks (normal velocity, fast, and accurate) and a path tracing task on a digitized tablet, with kinematics and DTW variables recorded via CSWin software (Marquardt et al., 2021). Linear mixed model analyses compared the two hands considering each collected measure as dependent variables.

Results showed significant kinematic differences between the DH and NDH, with the DH displaying faster, more fluent movements, though no differences in global letterform coherence were found. It confirms executional outperformance of the DH over the NDH noted in previous literature (Dexheimer et al., 2007; Heuer et al., 2007) moreover DH and NDH seem to show a common level of representation in motor programming (Grosskopf et al., 2006).

These findings, compared with prior research (Di Brina et al., 2008), suggest that letterform consistency might be not a powerful indicator of fine motor skills, but a robust indicator of motor sequence learning.

Biography

Carlo Di Brina obtained his MD, Child and Adolescence Neuropsychiatrist Specialization at University of Rome "Sapienza"and PhD at Human Neuroscience Department at the same University. And, now actually working in the Policlinico Umberto I° Hospital, Child and Adolescence Unit, sited in Via dei Sabelli, 108, Rome. Carlo Di Brina research interests have been primarily dedicated to neuropsychology and kinematical analysis of Movement Disorders such as Cerebral Palsy and Developmental Coordination Disorders. And, actually dedicated to handwriting analysis, manual dexterity and motor learning. Serving as investigator in multicenter studies with the Teachers Education University of Lucern and Bern, Swizerland. First author and key contributor, has had numerous publications in highly peer-reviewed journals. Translated, adapted and validated for Italy one of the most popular tool used in the diagnosis of Developmental Dysgraphia: The BHK Scale. Developed an innovative method for the use of Dynamic time warping software based analysis, in the study of letter shape consistency.

Dixie J Woolston
Mayo Clinic Arizona/Scottsdale, United States

Brain health factors in aging executives: Insights from Mayo Clinic Arizona's THRIVE pilot study

Background: Brain health is essential for cognitive, emotional, and behavioral functioning throughout life. The WHO's 2022 position paper emphasized the critical role of modifiable factors—such as physical activity, sleep, stress, and substance use—in maintaining brain health. Executives, a high-functioning group with unique cognitive demands, face challenges such as cumulative stress, pressure to use substances, including cognitive enhancers, to improve cognitive functioning, irregular schedules, and aging-related vulnerabilities. Despite these demands, research on brain health in executives remains sparse.

Objective: The THRIVE pilot study investigated correlations between lifestyle-driven Brain Care Scores, subjective cognitive concerns, and objective cognitive performance using CNS Vital Signs (CNSVS) and the Neuropsychology Questionnaire Short-Form (NPQ).

Methods: Thirty executives underwent a 2-day intensive health assessment, including 12–18 specialty evaluations on brain fitness, sleep, exercise, integrated medicine, cardiovascular health, and overall well-being. Brain Care Scores were calculated, and their relationship with CNSVS cognitive performance and NPQ-reported concerns was analyzed. Lifestyle factors such as poor sleep, alcohol use, low exercise, and high stress were examined for their impact on cognition. Group-level CNSVS data were analyzed for a potential cognitive profile specific to executives.

Expected Results:

- Higher Brain Care Scores will correlate with higher CNSVS cognitive scores.
- Subjective cognitive concerns will not align consistently with objective findings.
- Poor sleep, high stress, low exercise, emotional distress, and substance/alcohol use will correlate with lower CNSVS and Brain Care Scores.

Implications: Findings will inform THRIVE 2.0, developing tailored assessments and interventions for executive brain health. This research addresses gaps in understanding how lifestyle factors influence aging executives' cognitive profiles and provides a model for precision health care in high-functioning populations.

Conclusion: By integrating subjective, objective, and lifestyle data, this study highlights the importance of personalized strategies for optimizing brain health in aging executives, advancing knowledge in neurology and preventive medicine.

Biography

Dr. Dixie Woolston obtained a PhD in 2006 from University of Texas Southwestern Medical Center. Dr. Dixie completed a 2-year fellowship in Clinical Neuropsychology also at UT Southwestern. 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.

Prof. Esther Shohami PhD
The Hebrew University of Jerusalem, Israel

Targeting the endocannabinoid system: A novel approach to the treatment of traumatic brain injury and stroke

Cannabis is one of the most widely used plant drugs in the world and was used for millennia for its medicinal and mind-altering effects. The latter are known to be mediated via the activation of the endocannabinoid (eCB) receptor CB1. Developments in medicinal chemistry of novel non-psychoactive synthetic cannabinoids have indicated that it is possible to separate some of the therapeutic effects from the undesirable psychoactivity. Indeed, Cannabidiol (CBD) the major non-psychotropic cannabinoid found in cannabis sativa, has low affinity to the cannabinoid receptor types and has no "mind altering" properties. Nevertheless, it exerts properties affording neuroprotection in animal models of brain disorders, similar to those reported for the eCB system.

The eCB system includes ligands, such as anandamide and 2-Arachidonoyl Glycerol (2-AG), which bind to receptors (CB1, CB2, TRPV1), and a group of cannabionoid-like lipids, identified as fatty acid amides of ethanol amines and amino acids [N-acyl aminoacids e.g. arachidonoyl-serine, AraS), which do not bind to the eCB receptors yet exert pharmacological activities similar to these of the "classical" cannabinoids. The pharmacological profile of the eCB includes modulation of neurotransmitter systems and of the BBB along with antioxidants and anti-inflammatory activities. Our earlier studies, using mouse model of Traumatic Brain Injury (TBI), revealed a 10-fold increase in 2-AG in the injured hemisphere 4h after TBI. Importantly, treatment with synthetic 2-AG attenuated edema formation, infarct volume, BBB permeability, neuronal cell loss at the CA3 hippocampal region, and neuroinflammation. Moreover, AraS exerted eCB-mediated neuroprotection after TBI via the induction of a pro-survival and antiapoptotic cascade, and involves Akt and ERK phosphorylation in its downstream signaling. These effects were partly blocked by CB1, CB2 and TRPV1 antagonists, despite the lack of directing eCB binding to them. The properties of the newly described eCB system provide a mechanistic basis to support its role in reducing the effects of the harmful mechanisms of the primary events post-TBI, and to offer a basis for the long-term neuro-recovery effects on the secondary injury.

Biography

Prof. Esther Shohami has received doctorate in Physiology from the Hebrew University (HU), Jerusalem, Israel, joined the HU faculty of medicine in 1981, and is currently a Professor Emerita. Prof. Esther was a member of the executive board of the International Neurotrauma Society and was awarded (2022) by the Society Lifetime Achievement Award. And, served (2012-2014) as the president of the Israel Society for Neuroscience. Has published more than 250 articles, reviews and book chapters. Using animal models, she studied for more than 2 decades the involvement of the endocannabinoid system in neuro-recovery after traumatic brain injury.

Ewelina Jalonicka[1,2*], Konstantin Rusakov[3], Grzegorz Szwachta[1], Piotr Hanczyc[1,4]

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Mucin detection based on Thioflavin T and lasing effect–A proposal for diagnostics of brain tumors from human tears

Brain tumors, especially glioblastoma, are highly malignant and lethal to humans. There is no rapid, non-invasive, early diagnostic method in the world. Recent reports indicate a major role of mucins in the pathogenesis of glioblastoma. Unfortunately, there is no method for rapid examination of the mucin layer. Current methods focus on specific mucins and are time-consuming, e.g. ELISA, and Western Blot. Moreover, due to the anatomical proximity of the eye and the brain, human tears are an interesting option as material for research. We propose a new diagnostic method based on the study of mucins in human tears.

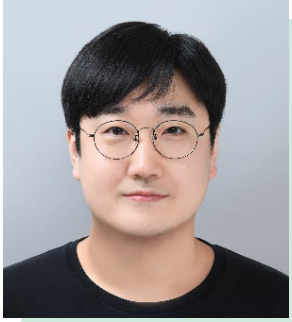
The method is based on the sensitive detection of mucins based on their staining with Thioflavin T (ThT) and subsequent laser measurements. ThT is regarded as the gold standard for detecting subtle molecular changes in the structure of biomolecules, including proteins and DNA. It is broadly used in the diagnosis of neurodegenerative diseases. We have implemented this technology for mucins.

In this paper, we present the results of lasing effects obtained in experiments with Fabry-Pérot cavities. Our study demonstrates that ThT selectively binds to mucins (modeled by MUC3) in DEMI water, artificial tears, and simulated tear environments. The application of Fabry-Pérot cavity lasing spectroscopy enabled the resolution of distinct spectral signatures of the ThT-mucin complex, including the emergence of dual lasing peaks and an increased lasing threshold in mucin-rich samples compared to controls.

The results indicate the possibility of detecting mucins from human tears and in the longer term, a proposal for fast, minimally invasive diagnosis of brain tumors from human tears.

Biography

Ewelina Jalonicka is a sixth-year medical student that combines the clinical research with basic science. The main research direction she undertakes are neurodegenerative disorders, brain tumors with particular emphasis on highly malignant glioblastoma, and opportunistic infections. In her glioblastoma research, she is looking for both new therapies and a modern diagnostic method. She was involved in three grants: the JPND grant (EU Joint Programme-Neurodegenerative Disease Research), SONATA NCN at the University of Warsaw (Faculty of Physics), and also in MAESTRO NCN at the Institute of Physical Chemistry (Polish Academy of Sciences in Warsaw).



Hakjoo Kim, Ph.D.

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Measuring and modulating neuronal excitability with transcranial magnetic stimulation: Approaches and mechanisms

Transcranial Magnetic Stimulation (TMS) has emerged as a non-invasive neuromodulation technique capable of inducing neuroplasticity, with significant potential in both basic research and clinical applications. This presentation will explore the application of various TMS protocols aimed at assessing and modulating neuronal excitability. First, Dr. Kim will outline approaches for measuring corticospinal and corticocortical excitability. A key focus will be on dual-coil paired-pulse TMS, a method used to investigate corticocortical connectivity between distinct brain regions, including the primary motor cortex (M1). Particular attention will be given to pathways such as cerebellum-M1 and supplementary motor complex-M1, highlighting their functional roles and modulatory effects. Next, the presentation will cover advanced techniques for evaluating cortical excitability in brain regions beyond M1. Using TMS-EEG integration, Dr. Kim will illustrate how neuronal excitability can be studied in areas like the Dorsolateral Prefrontal Cortex (DLPFC), expanding our understanding of TMS applications in diverse cortical areas. Additionally, the effects of repetitive TMS (rTMS) on neuronal excitability will be explored, with insights drawn from both preclinical models and human studies. Dr. Kim will discuss the underlying mechanisms driving these neuromodulatory effects, with a specific focus on post-synaptic plasticity, and will elaborate on the potential for mechanism-based pharmacological augmentation to enhance these outcomes. Finally, the presentation will conclude by discussing the broader implications of these findings, emphasizing their contributions to advancing future research and clinical applications of TMS.

Biography

Dr. Kim obtained his Master's and Ph.D. degrees from Texas A&M University in College Station, Texas. Currently a postdoctoral research fellow at McLean Hospital, affiliated with Harvard Medical School. Dr. Kim’s research has focused on neuroplasticity and non-invasive brain stimulation techniques, such as Transcranial Magnetic Stimulation (TMS). And, current projects involve investigating the mechanisms of synaptic plasticity through accelerated-TMS protocols and pharmacological augmentation.

Hyelim Chun[1*], Hee Won Lee[2], Sang Soo Ha[3], Seung Bong Hong[4], Kang-Jun Yoon[3]

[1]Clinical Trial Center, St. Peter's General Hospital, Seoul, Republic of Korea

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Efficacy of transcranial photobiomodulation in mild cognitive impairment and early alzheimer's disease: A randomized controlled study

Transcranial Photobiomodulation (t-PBM) is a novel, non-invasive, neuromodulation technique. It has a wide range of indications in neurodegenerative diseases including Mild Cognitive Impairment (MCI), which serves as a potential preclinical stage of early Alzheimer's Disease (AD). Infrared light of wavelength 808 nm emitted from t-PBM device penetrates robustly into the cortex, stimulating neuronal activity via increasing mitochondrial ATP production and regional cerebral blood flow. Based on such molecular background, we investigate the efficacy and safety of t-PBM in cognitive improvement in MCI patients.

MCI patients who meet all the core clinical criteria for MCI due to AD based on the diagnostic guidelines provided by the National Institute on Aging and Alzheimer's Association (NIA-AA) were recruited. Participants scoring MMSE 23~27 and CDR 0.5~1.0 with possible causes of cognitive decline other than AD ruled out were enrolled. Subjects underwent t-PBM treatment 6 times per week for 12 weeks total at home, with light directed at dorsolateral prefrontal cortex. Subjects were evaluated at weeks 7 and 13 with Korean version of Montreal Cognitive Assessment (K-MoCA), Korean version of Mini-Mental State Examination (K-MMSE2), Korean version of Consortium to Establish a Registry for Alzheimer's Disease (CERAD-K), and Geriatric Depression Scale.
Mean changes in total scores of each exam obtained at weeks 7 and 13 were compared to the effect of sham with two-sample t-test.

A total of 26 subjects (70.0 ± 6.97 years; 73% female) were investigated. K-MoCA score improvement of experimental group was statistically significant ($p<0.05$) compared to sham at week 13. Subjects presented no serious adverse events.

Findings suggest that t-PBM is effective and safe for home-use in MCI population. t-PBM could become a promising treatment candidate for MCI, serving as a protective as well as preventative measure against progression into AD. t-PBM's therapeutic application in normal aging group warrants further evaluation.

Biography

Dr. Hyelim Chun received Bachelor of Science from University of California, Los Angeles (UCLA), majoring in Molecular, Cell, and Developmental Biology with a minor in Biomedical Research and was part of the Karen Lyons lab at UCLA, studying the role of ALK5 in BMP signaling pathway in mammalian cartilage development. Dr. Hyelim attended Inha University School of Medicine where received MD. Now works as a clinical researcher at St. Peter's General Hospital, studying photobiomodulation technique with a special interest in its application in neurodegenerative diseases.

Jin Woo Shin M.D., Ph.D.

The president of the Korean Pain Society Professor, Department of Anesthesiology and Pain Medicine, SEOUL ASAN Medical Center University of Ulsan College of Medicine 86, Asanbyeongwon-gil, Songpa-gu, Seoul 138-736, Korea

Novel procedure: Spinal epidural balloon decompression and adhesiolysis

Nonsurgical treatments, such as nerve block, in chronic pain patients with severe adhesions are reported to have a relatively low effect and a high risk for relapse. This may be attributed to the fact that epidural adhesions themselves are difficult to remove through such methods, and also that they interfere with the effective spread of a therapeutic agent to the lesion.

If a simple nerve block does not have a sufficient effect in a patient with pain caused by adhesions or stenosis, it is important to confirm whether pain is associated with adhesion and where the adhesion, suspected of being a lesion, is positioned. Once adhesions or stenosis are confirmed as a cause of pain, neuroplasty may be performed to relieve them.

Conventional neuroplasty may be divided into either chemical adhesiolysis using hypertonic saline or mechanical adhesiolysis using a catheter that can be moved laterally.

Conventional neruoplasty has shown good short-term analgesic effects; however, functional improvement has not been enough.

We hypothesized that the balloon dilatation used for the relief of vascular stenosis may be applied to the epidural space to enable relief of spinal stenosis by more extensive epidural adhesiolysis and by expansion of the marginal space around the nerve as much as possible in the stenotic intervertebral foramen. We also hypothesized that balloon dilatation enables minimization of the nerve damage during adhesiolysis. Based on the aforementioned premises, we conducted the following clinical study. The subjects included patients with only intractable neural foraminal stenosis who were nonresponsive to conventional transforaminal epidural block or whose alleviation of pain did not continue for 1 month or more. We compared two procedures: in one group, transforaminal balloon catheter was inserted to the intervertebral foramen, balloon dilatation was performed, and the asteroid was injected; in the control group, the same procedure was performed excluding the balloon dilatation. When conventional transforaminal block is performed in patients with severe neural foraminal stenosis or having epidural adhesions, a contrast agent is sometimes not introduced into the epidural space through the intervertebral foramen. In that case, even the trial to insert and withdraw a thin catheter may introduce the contrast agent into the epidural space. The reason may be that the action of inserting and withdrawing the catheter partially relieves stenosis and eliminates epidural adhesions. The control group procedure without balloon dilatation might be more

effective than conventional transforaminal block in the sense that the catheter was inserted into and withdrawn. Nevertheless, the pain relief and functional improvements were much greater in the group in which the balloon dilatation was performed, with the effects lasting for 3 to 4 months after the procedures. In addition, the ratio of patients whose pain decreased by more than 50% for 1 year or more was 18.8% in the group with balloon dilatation and 0% in the group without balloon dilatation.

In cases of spinal stenosis, most conventional nonsurgical procedures have shown good short-term analgesic effects; however, functional improvement has not been enough. The procedure used in our study has great clinical significance in the sense that it greatly improved not only pain but also functions such as neurogenic claudication in even cases of intractable spinal stenosis. In the control group, where balloon dilatation was not performed, the catheter was inserted into and withdrawn from the intervertebral foramen in a manner similar to mechanical adhesiolysis in conventional neuroplasty. The fact that such a powerful procedure was less effective than the procedure with balloon dilatation indicated that balloon dilatation is likely more effective than pre-existing procedures.

To test whether the positive outcome was the result of marginal space expansion in the intervertebral foramen by balloon dilatation, as well as to examine how the marginal space around nerve may expand, the spread of contrast agent in the intervertebral foramen before and after balloon dilatation were reconstructed three-dimensionally in four patients. Comparing the degree of expansion of the marginal space showed that the diameter of the region where the contrast agent spread was increased by 28% and the diffusion volume was increased by 98% after the procedure.

Based on the finding that balloon dilatation in the epidural space can relieve not only adhesions but also stenosis, a novel catheter (Ziazag-Inflatable Neuroplasty: ZiNeu, JUVENUI, Korea) was developed by adding the balloon dilatation function to the same type of video-guide catheter used for conventional epiduroscopy. As this catheter has the additional function of balloon dilatation, it may eliminate an adhesion more effectively, causing less neural damage or dural injury. It may even be used to relieve the neural foraminal stenosis. Moreover, as this catheter has the function of leaving a thin epidural catheter for precise drug injection at the target lesion, it may be used to perform chemical adhesiolysis at once.

Also, the ZiNeuF catheter, which can be accessed directly to the intervertebral foramen, has been developed and used extensively in Korea.

The ZiNeu catheter has 18 SCI(S) articles and 3 Korean papers have been published since it was marketed in 2013 in Korea. Recently, the ZiNeu catheter has obtained CE certification and started to be used in Italy and Singapore.

Biography

Jin Woo Shin was born in Seoul in 1968. After graduating from Chung-Ang University Medical School, completed the resident course, master's, and doctoral degrees in the Department of Anesthesiology and Pain Medicine at Seoul ASAN medical center. Jin Woo Shin worked as a clinical fellow, assistant professor, and associate professor and have been a professor since 2013 at Seoul ASAN Medical center. During his period as an assistant professor, conducted animal experiments on neuropathic pain at Children's Hospital Boston and Brigham and Women's Hospital which is a related hospital to Harvard Medical School for one year in the USA. Currently serve as a director of executive management at the Korean Pain Society, the Korean Spinal Pain Society, and the Korean neuromodulation Society. Jin Woo Shin have been working as a professor at Asan Hospital Pain Clinic in Seoul for 18 years, mainly treating patients with spinal pain and neuropathic pain, and have published many research papers. As his representative achievement, developed the world's first interventional procedure called ‘Spinal epidural balloon decompression and adhesiolysis’ and published books about it in Korean and English. Jin Woo Shin also systematized the procedure so-called ‘genicular nerve radiofrequency ablation therapy' for patients with chronic knee pain and published it for the first time in a high-profile journal called Pain, and this procedure is currently being performed by many doctors around the world.

Akankunda Veronicah Karuhanga, Anedibo Joan*
Golden Age Elderly Homes Uganda

Benefits of quail eggs for seniors with neurological/brain disorders quail eggs

Have been studied for their potential benefits in neurology and brain disorders, particularly in seniors

Nutritional Benefits: Quail Eggs are Rich in:

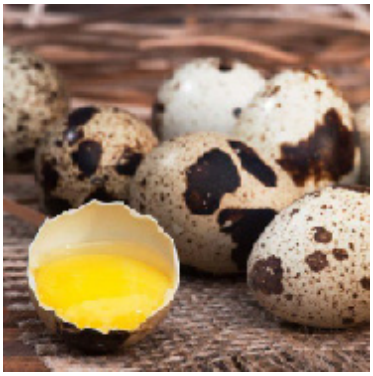
1. **Choline:** Essential for brain health, cognitive function, and memory.
2. **Vitamin B_{12}:** Crucial for nerve function, brain health, and red blood cell formation.
3. **Omega-3 Fatty Acids:** Anti-inflammatory effects, supporting brain health.
4. **Antioxidants:** Protecting against oxidative stress, inflammation, and cell damage.

Potential Benefits in Neurology and Brain Disorders: Research Suggests that Quail Eggs May:

1. **Improve Cognitive Function:** Enhance memory, attention, and processing speed in seniors.
2. **Support Neuroprotection:** Protect against neurodegenerative diseases, such as Alzheimer's and Parkinson's.
3. **Reduce Inflammation:** Quail egg antioxidants and omega-3 fatty acids may help reduce
4. inflammation in the brain.
5. **Enhance Neuronal Health:** Promote healthy neuronal function, potentially slowing down
6. age-related cognitive decline.

Studies and Findings: Some Studies Have Investigated The Effects of Quail Eggs on Brain Health:

1. **Cognitive Function in Seniors:** A study published in the Journal of Alzheimer's Disease found that quail egg supplementation improved cognitive function in seniors with mild cognitive impairment.
2. **Neuroprotection:** Research published in the Journal of Neuroscience Research suggested that quail egg extracts exhibited neuroprotective effects against oxidative stress and inflammation.



In summary, quail eggs may offer nutritional benefits and potential advantages for brain health, particularly in seniors. However, further research is necessary to fully understand their effects

Biography

Joan Anedibo 26, is a Neuro Researcher and a continuing Geriatric student at Golden Age Geriatric Academy, research focusses more on new treatments for onset of symptoms of Alzheimer's disease, familial trends in neurological diseases, new therapies and treatments for epilepsy, multiple sclerosis, Parkinson's disease, and other conditions. Joan Anedibo is currently doing research on advantages of proper Nutrition among seniors with Neurological and brain disorders focussing on the use of Quail eggs.

Physiotherapist Joao Rafael de Oliveira Rocha da Silva[1*], Mariana de Oliveira Rocha da Silva[2]

[1]Postgraduate in interdisciplinary assessment and treatment of Pain at the Hospital das Clinicas of the Faculty of Medicine of the University of Sao Paulo HC-FMUSP, Postgraduate in rehabilitation applied to sport Department of Orthopedics and Traumatology, Escola Paulista de Medicina, Universidade Federal de Sao Paulo CETE-UNIFESP, Brazil

[2]Postgraduate in Clinical Exercise Physiology from Universidade Federal Sao Carlos UFSCAR, Postgraduate student in strength training sciences at the Federal University of Sao Carlos UFSCAR, Brazil

Dysfunctional changes presented by individuals with chronic pain, which impact evidence-based practice for treating pain: Systematic review

Chronic pain is defined as persistent pain for more than three months, resulting in changes in the functional synaptic network of the brain and its gray matter dimensions, causing hypersensitivity to a nociceptive stimulus and increased excitability of the nodal stress mechanism, causing the individual to remain in a state of alert (hyperactivation of the sympathetic nervous system) in normal daily activities, increasing their level 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 high relevance of studies that address the importance of exercising in these individuals, their understanding of the 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 suitable; however, we observed a lack of information on recurrent neurofunctional and biomechanical changes in this population, which we can classify as a pathological pattern that should not be neglected.

In previous studies, we observed that it directly impacts cardiac rehabilitation and adherence to physical exercise, significantly increasing disability and mortality in the population.

We also observed that individuals with chronic pain present patterns of changes in motor control and kinesiophobia, with chronic low back pain and knee osteoarthritis being the most frequent causes of disability, directly impacting cardiac rehabilitation due to the difficulty in obtaining adequate adherence to physical exercise.

Both pathologies mentioned are very relevant in literature and clinical practice. However, any type of chronic musculoskeletal pain can lead the individual to functional disability since musculoskeletal pain in the lower and upper limbs directly impacts gait, work activities, and

physical activities in the practice of exercises and activities of daily living.

In previous studies, it was possible to observe the pathophysiology of chronic pain being responsible for altering the neuromuscular reflex, causing changes in motor control due to several factors such as muscular inhibition, muscular rigidity, body perception deficit, and sensorimotor system changes.

Biography

Pt. Joao 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 Sao Paulo HC-FMUSP. Pt. Joao Rafael Rocha da 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.

Jun Pan*, Shengyi Xu

Department of Translation, Interpreting and Intercultural Studies, Academy of Language and Culture, Hong Kong Baptist University

Neurobiological foundations of language mediation: A systematic review and meta-analysis of fMRI studies in translation and interpreting

Language mediation, ranging from written translation to interpreting, represents one of the most complex cognitive tasks performed by the human brain which involves an intricate interplay of language processing, executive control, and working memory systems. The neural architecture supporting these processes has become increasingly accessible to scientific inquiry through functional Magnetic Resonance Imaging (fMRI), offering unprecedented insights into the neurobiological foundations of language mediation. The past two decades have witnessed a substantial, albeit still small, number of neuroimaging studies examining the neural correlates of translation and interpreting. These investigations have evolved from early studies focusing on single-word translation to more sophisticated paradigms capturing the complexity of interpreting. While individual studies have illuminated specific aspects of the neural mechanisms involved, a comprehensive understanding of the field's development, methodological advances, and emerging research trajectories remains to be synthesised.

This study thus aims to map the landscape of fMRI research in translation and interpreting studies through a mixed methodology of systematic review and meta-analysis. Following the PRISMA 2020 reporting guideline (Page et al. 2021), authors of the study identified and selected a total of 13 journal articles from an initial pool of 2,700 entries collected through a comprehensive search of EBSCOhost, ProQuest, PubMed, and Web of Science databases. All of the entries employed fMRI to investigate brain activity and function in bilingual and multilingual individuals performing language-switching and/or interpreting- related tasks. Through close reading of the selected entries, main variables including bilingualism, expertise/proficiency, brain structure, and brain function were identified and their relationship closely examined. The study concludes with a map indicating the intricate relationship among the variables as reflected in the fMRI experiment results of all 13 studies, showcasing how bilingualism, mediated by expertise/proficiency, correlates with the status and changes in brain structure and function, in particular in aspects of language and cognitive control.

This systematic review pinpoints the evolution of theoretical frameworks in neuroscience approaches to language mediation, as well as methodological innovations in fMRI paradigm design for translation and interpreting tasks. Through examining the convergence of evidence regarding neural substrates specific to language mediation, the study helps to identify emerging research fronts and potential gaps for future research.

Biography

Jun Pan is Professor of the Department of Translation, Interpreting and Intercultural Studies and Director of the Academy of Language and Culture at Hong Kong Baptist University. Prof. Pan's research interests lie in peak performance, professionalism in interpreting, and corpus-based interpreting/translation studies. Jun Pan (co-) authored over 100 journal articles, book chapters and conference papers, a full list of which can be found at Prof. Pan's recent work focuses on Enter-Link, a digital platform that aims to integrate human-machine collaboration to break down language barriers, especially in legal and medical fields, including rare disease communication.

Katarzyna Stachowicz

Department of Neurobiology, Maj Institute of Pharmacology, Polish Academy of Sciences, Smętna 12, 31-343 Krakow, Poland

Interactions between COX-2 and glutamatergic receptors with CB1 as a novel target in mood and cognitive studies

Glutamate, the brain's primary excitatory neurotransmitter, maintains neuronal communication and functional stability. Together with Gamma-Aminobutyric Acid (GABA), the primary inhibitory neurotransmitter, it establishes a balance essential for cognitive processes, emotional regulation, and neural plasticity. Dysregulated glutamatergic signaling has been implicated in various psychiatric and neurological disorders, including depression, anxiety, and cognitive impairments.

Glutamate exerts its effects through two receptor types: Ionotropic, mediating rapid synaptic transmission, and metabotropic (mGluRs), which modulate neuronal activity over longer timescales. Among these, mGluRs are gaining attention for their role in synaptic plasticity and their therapeutic potential in neuropsychiatric conditions.

Cyclooxygenase-2 (COX-2), an enzyme involved in the inflammatory response, plays a significant role in glutamatergic pathways. Its activity is linked to the production of prostanoids, lipid mediators derived from Arachidonic Acid (AA), a polyunsaturated omega-6 fatty acid released by Phospholipase A2 (PLA2). While inflammation mediated by COX-2 is a natural response to injury, excessive activity can contribute to neuroinflammation, cognitive decline, and mood disorders, making COX-2 an important therapeutic target.

Recent findings reveal a novel interaction between metabotropic Glutamate Receptor 7 (mGluR7) and Cannabinoid Receptor 1 (CB1), highlighting their synergistic role in anxiety regulation. This discovery underscores the importance of receptor crosstalk in complex emotional states and suggests potential avenues for developing innovative treatments targeting mGluR7 and CB1.

By bridging the fields of neurotransmission, lipid signaling, and receptor dynamics, these insights deepen our understanding of brain function and its dysregulation in mental health conditions.

Biography

Dr. Katarzyna Stachowicz's research interests focus on the molecular mechanisms of depression, cognition and anxiety. Dr. Katarzyna doctoral dissertation at the Department of Neurobiology, Institute of Pharmacology, Polish Academy of Sciences, Krakow, Poland, focused on molecular mechanisms of anxiety involving mGluRs. Dr. Katarzyna was a postdoctoral fellow at the Center for Neuroscience Drug Discovery, Vanderbilt University Medical Center, Nashville, TN, USA and an assistant professor, postdoctoral dissertation focused on COX-2/mGluRs interactions in depression. Dr. Katarzyna is also a pop scientist who seeks to combine neuroscience and physics to talk about the underlying molecular mechanisms in the brain. Katarzyna Stachowicz published more than 60 research articles in SCI journals and fourteen popular science articles.

Martin D Kafina* MD, Fabio O Danisi MD

Department of Neurology, Westchester Medical Center, Valhalla, NY, USA

Heterotopic ossification of skeletal muscle: A complication of stiff person syndrome

This case report highlights how Stiff Person Syndrome (SPS) can be associated with severe complications, including Heterotopic Ossification (HO). HO of skeletal muscle has not been described in the pathophysiology of SPS and has been described in only one other case report of SPS in which the hip and knee joints were affected. We present this phenomenon to alert clinicians to a potential complication of SPS that can lead to unnecessary testing and delays in patient care.

Background and Objectives: Stiff Person Syndrome (SPS) is a heterogeneous neurological condition, which is thought to be autoimmune in nature. SPS is characterized by a combination of muscle hypertonia, reflex and action-induced spasms, and continuous motor activity at rest. SPS is often associated with the presence of antibodies to GAD 65.

Methods: We review the clinical manifestations of SPS and the entities associated with GAD 65 antibodies and describe the case of a previously healthy 62 year old man who presented to the hospital for evaluation of acute on chronic truncal and lower extremity spasms

Results: Our patient was found to have elevated GAD 65 antibody titers and radiographic evidence of heterotopic ossification of the psoas muscles.

Discussion: Heterotopic Ossification (HO) affecting skeletal muscle in patients with SPS has not been previously described. We present this phenomenon to alert clinicians to a potential complication of SPS that can lead to unnecessary testing and delays in patient care.

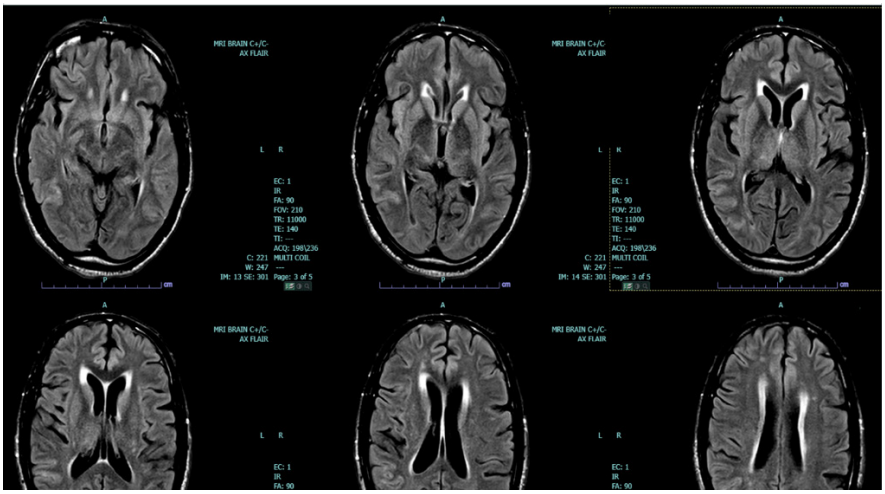
Case Report: We describe the case of a 62 year old African American gentleman who presented to the emergency department for subacute onset of truncal and lower extremity spasms, which progressively worsened. The patient had a past medical history only significant for hypertension and had been in his usual state of health until approximately 3 years before his presentation. He had been athletically active and exercising with running up to 4 miles a day. The patient progressively subsequently developed episodes of sudden and painful tightening of his abdominal muscles. Over time the symptoms of cramping and spasms spread throughout the entire body and severely affected the right lower extremity. Four months prior to presentation, he required the use of a walker due to persistent tightness in his trunk and lower

extremities. The patient was advised by a gastroenterologist and rheumatologist to be seen by a local neurologist. The neurologists tarted the patient on carbidopa/levodopa followed by gabapentin without improvement in symptoms. Outpatient workup included MRI of the brain showing a T2 flair hyperintensity in the left frontal lobe measuring 3.2 cm and a wedge shaped T2 flair hyperintensity in the right parietal lobe measuring 1.4 cm. Additionally, an autoimmune panel including ANA was unremarkable.

The patient presented to the emergency department of our institution due to severe worsening of spasms including severe pain in the right leg Physical examination was notable for stiff muscles of the lower extremities and severely reduced passive range of motion. Reflexes, sensation, and mental status were normal.

MRI of the brain was repeated and showed gyriform parenchymal signal abnormalities within the right parietal and left frontal lobes with superimposed and surrounding punctate foci of susceptibility artifacts without abnormal enhancement [fig 1]. MRI C-spine showed multilevel degenerative changes with acquired spinal and for aminal stenoses most severe at C6-7 and 1 to 2 mm retrolisthesis of C5 on C6 and C6 on C7 [fig 2]. MRI thoracic and lumbosacral spine showed mild lumbar spinal stenosis and bilateral psoas muscle enlargement [fig 3 and 4]. MRI pelvis showed heterotopic ossification within the left iliopsoas muscle suggestive of myositis ossificans and a similar process, likely at a more mature stage, was seen within the right iliopsoas. There was enlargement of bilateral ilopsoas muscles (left appears larger than the right iliopsoas muscle) [fig 5]. CT pelvis revealed calcification in the iliopsoas consistent with heterotopic ossification [fig 6].

Fig 1. MRI brain: Gyriform parenchymal signal abnormalities within the right parietal and left frontal lobes with superimposed and surrounding punctate foci of susceptibility artifacts without abnormal enhancement



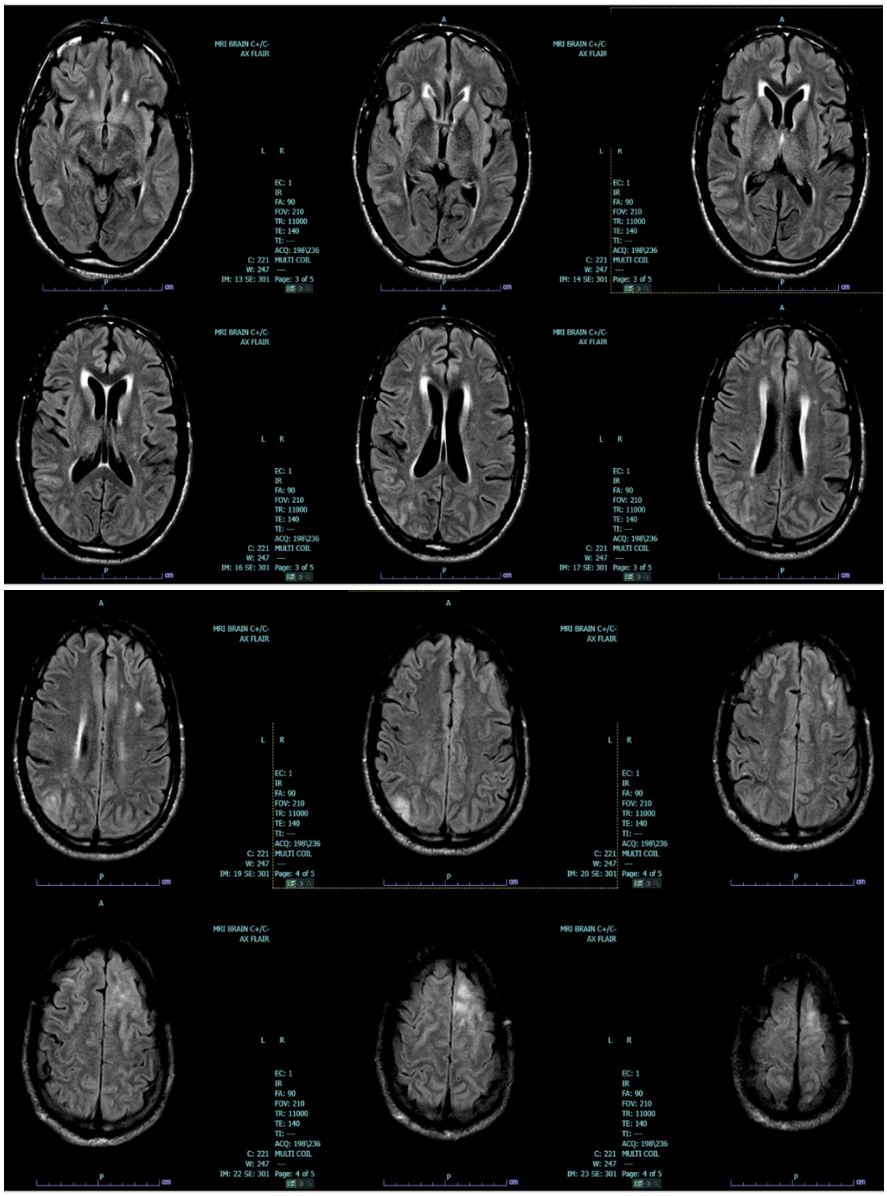


Fig 2. MRI C-spine: Multilevel degenerative changes with acquired spinal and foraminal stenoses most severe at C6-7 and 1 to 2 mm retrolisthesis of C5 on C6 and C6 on C7.

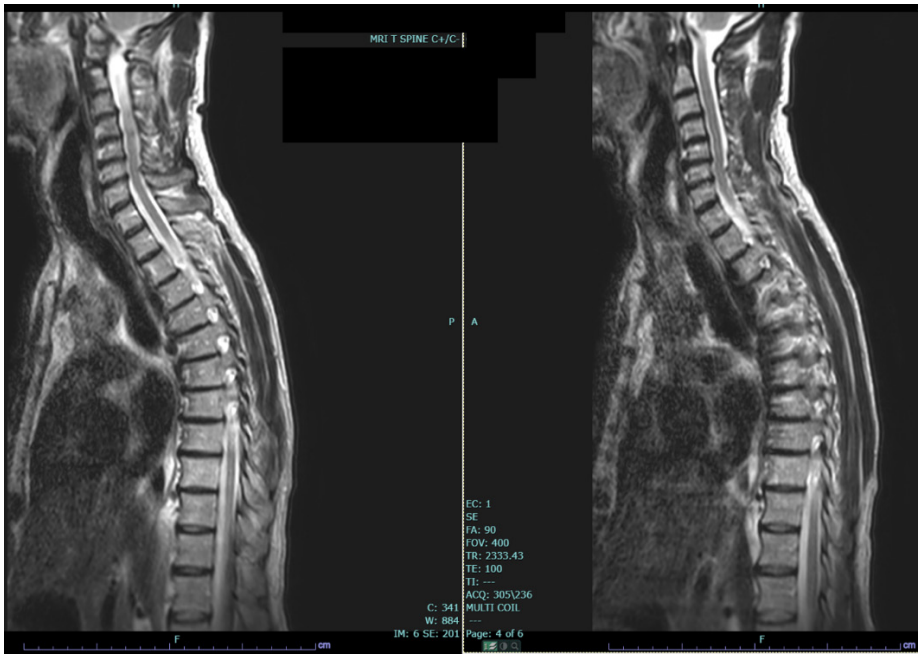


Fig 3. MRI T-Spine.

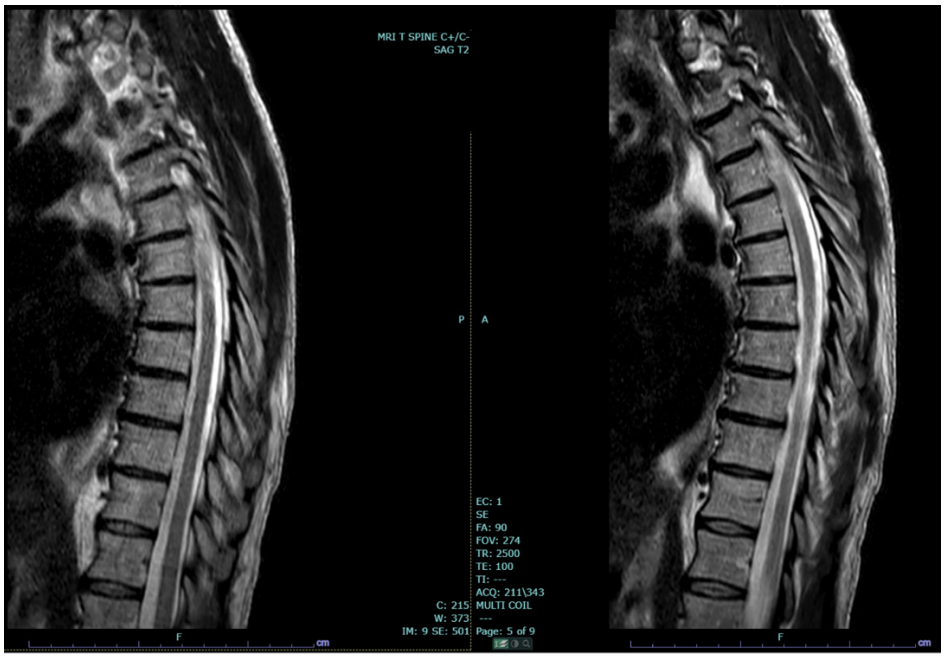


Fig 4. MRI LS-Spine: Mild lumbar spinal stenosis and bilateral psoas muscle enlargement.

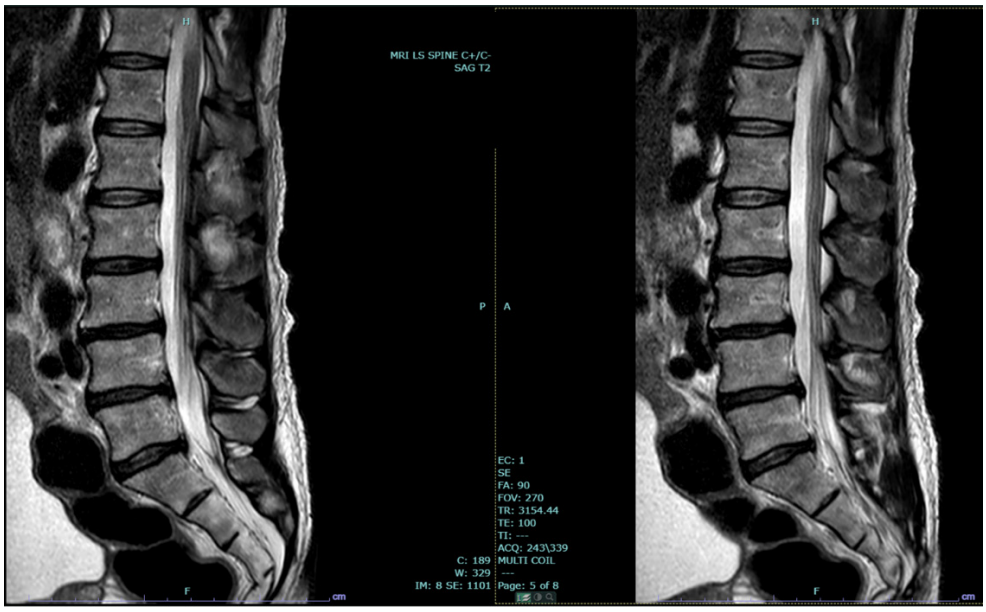


Fig 5. MRI pelvis: Heterotopic ossification within the left iliopsoas; ossification at at a more mature stage seen within the right iliopsoas. Enlargement of L>R ilopsoas muscles.

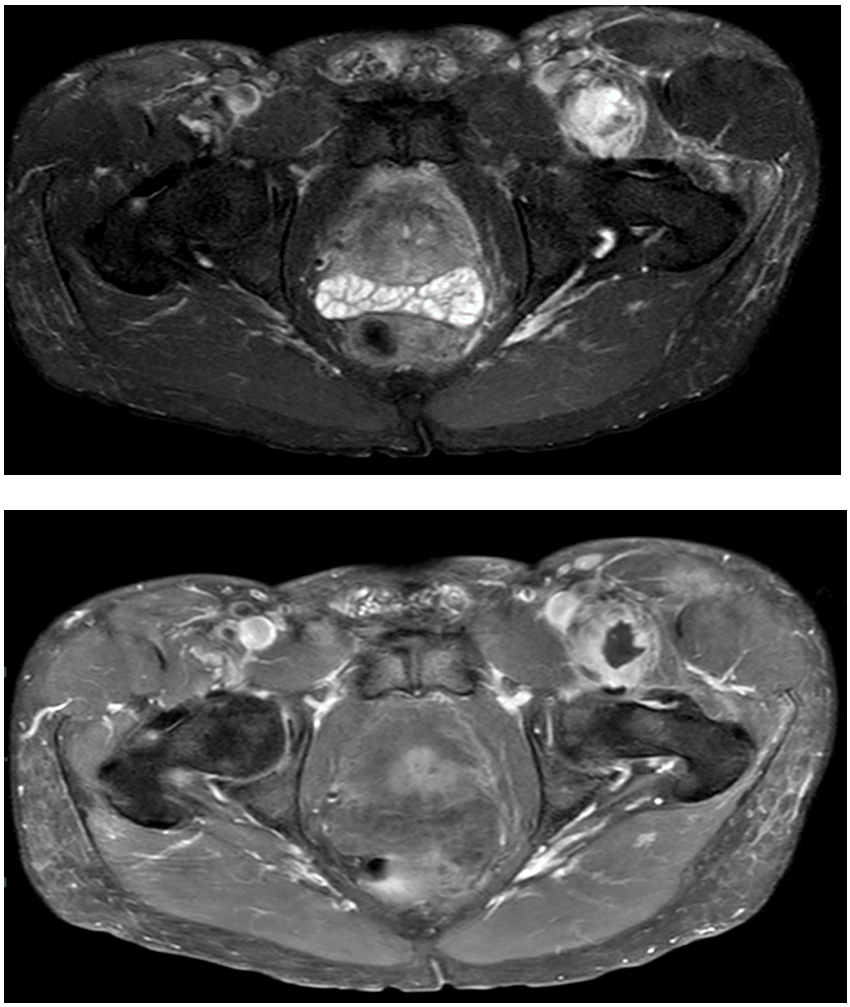
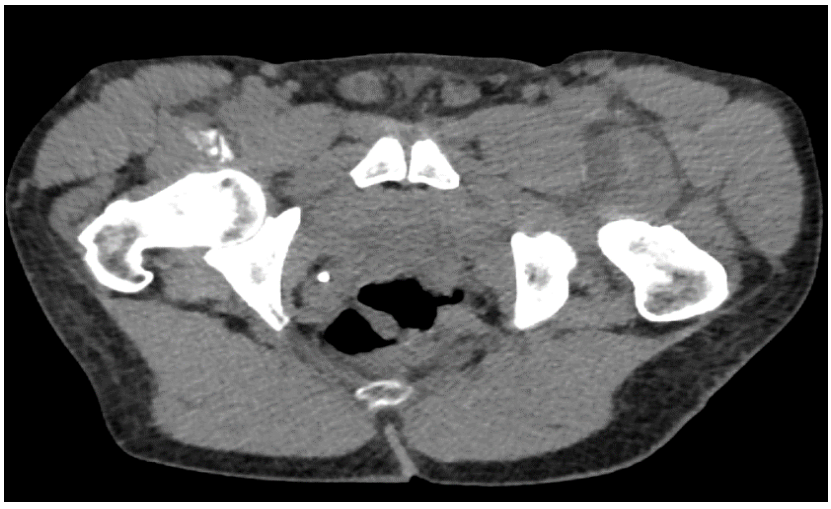
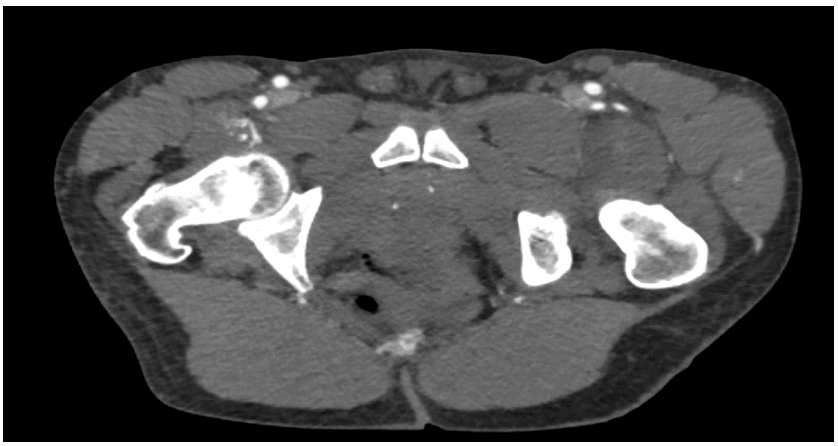


Fig 6. CT pelvis pre (top image) and post IV contrast (bottom image): Calcification in the iliopsoas without contrast enhancement.





Due to the heterotopic ossification of the psoas muscles, consultation with rheumatology, general surgery, and vascular surgery services was obtained.

Laboratory testing revealed elevated CPK at 901 (30-200) with normal CBC and renal function. HgbA1c was 5.5%. Paraneoplastic Ab panel was negative. The GAD65 antibody titer was >250 IU/mL (0-5).

Stiff Person Syndrome: SPS is a heterogeneous neurological condition, thought to be autoimmune in nature, and clinically characterized by symptoms of muscle hypertonia and painful spasms. Patients can be observed to have reflex and action-induced spasms and continuous motor activity at rest.

The most commonly observed phenotype is classical SPS manifesting with spasms in the muscles of the neck, trunk, and proximal lower limbs. Stiff limb syndrome manifests assustained limb spasms, which are often localized distally. Progressive Encephalomyelitis with Rigidity and Myoclonus (PERM) is a more aggressive condition with bulbar involvement and symptoms of dysautonomia.

SPS complications include fractures, joint dislocations, rhabdomyolysis, dysautonomia, hypoxemia, and behavioral disorders.

SPS comorbidities include DM in 35% of cases, autoimmune thyroid disease, pernicious anemia, vitiligo, myasthenia gravis, epilepsy, cerebellar ataxia, thymoma, and other malignancies.

GAD65 Abs are found in 60-80% of classical SPS. Additionally, about 4% of patients have antibodies against amphiphysin, glycine receptor, and gephyrin. A third of cases are seronegative. GAD65 autoantibodies are also seen in other conditions such as cerebellar ataxia, limbic and extralimbic encephalitis, epilepsy, oculomotor dysfunction, and myoclonus. In these cases there is often presence of anti-thyroid peroxidase, anti-thyroglobulin, anti-parietal cell, and anti-gliadin antibodies.

Heterotopic Ossification (HO): Heterotopic ossification, also known as myositis ossificans, is a self limiting, benign, ossifying lesion that can affect any soft tissue and is most commonly found in muscle. In 75% of cases there is a history of direct trauma or repetitive injury. HO is due to inappropriate fibroblast differentiation into osteogenic cells, typically as the result of tissue injury and inflammation.

In the early stage of HO (0-4 weeks) an inflammatory cascade is present. However, calcifications are often not visible and only become radio graphically apparent in the intermediate stage (4-8 weeks). Finally, in the mature stage (>8 weeks) there is bone formation followed by lesion maturation, consolidation, and regression.

HO typically affects young active males after sustaining blunt injuries. Common presentations include “rider’s bone” with ossification of the thigh adductors of horseback riders and “shooter’s bone” with involvement of the deltoid. Non-traumatic HO is uncommon and psoas involvement is rare.

Discussion: We aim to highlight how SPS can lead to severe and potentially persistent musculoskeletal pathology. In our case the primary site of heterotopic ossification was in the psoas muscles, which is an unusual location and was not associated with blunt trauma. Multiple consultants were involved and other reasonable causes of HO were ruled out in our case. The history and clinical presentation of this patient, notably the progressive decline in functional status, suggests an underlying pathology from severe and recurrent spasms leading to debilitating muscle injury.

There has been only one other case report of HO in ananti-GAD65 positive SPS patient. This other case report discussed a HLA-B27 and anti-GAD65 positive patient with SPS and Hodgkin’s Lymphoma that was intubated and admitted to an ICU. Following a period of prolonged immobilization and critical illness the patient developed HO within the joints of the knee and hip. Thus, our case report can be considered an original and unusual presentation of SPS given HO affecting the skeletal muscle proper.

Recognizing that HO can be a complication of SPS will assist the clinician in streamlining workup and patient care. This case uncovers new pathophysiology of SPS and highlights areas for research investigation and therapeutic intervention.

Biography

Dr. Kafina completed a bachelor’s degree in neuroscience & behavior at Wesleyan University and obtained a masters degree in neuroscience at Brandeis University. Following graduate school, Dr. Kafina researched Hematology at Harvard Medical school affiliated with Brigham and Women’s hospital. Dr. Kafina published studies in journals including Science and Blood on treatments for iron deficiency anemia and novel mechanisms of porphyria as well as first author review articles on hematopoiesis. Also, utilized genetic engineering of zebrafish and cellular experiments to achieve the publications. Dr. Kafina studied medicine at St. George’s University School of Medicine and is currently a Neurology resident at Westchester Medical Center.

Michelle Melgarejo da Rosa[1,2*], Barbara Raíssa Ferreira de Lima[3], Leydianne Leite de Siqueira Patriota[3], Thiago Henrique Napoleao[1,3], Patricia Maria Guedes Paiva[1,3]

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Schinus Terebinthifolia Raddi Leaf Lectin (STeLL) modulates subacute depressive and anxious behaviors in stress-exposed mice

Anxiety and depression are major causes of disability worldwide, often worsened by chronic stress. Schinus Terebinthifolia Raddi (SteLL) has been traditionally used for various medicinal purposes, including treating depression with bark-and-leaf tea or leaf decoction. Previous studies suggest that SteLL leaf lectin can alleviate anxiety and depression symptoms in mice.

Aim: To evaluate SteLL's potential in reducing depression and anxiety symptoms in a chronic stress model.

Methods: Mice underwent four weeks of Unpredictable Chronic Mild Stress (UCMS), followed by 21-day treatment with SteLL (2 or 4 mg/kg, i.p.) or fluoxetine (10 mg/kg, i.p., as a positive control). Behavioral tests assessed anxiety and depression-like behavior. Additionally, serum corticosterone, inflammatory markers (cytokines), oxidative stress indicators, and brain monoamine levels were analyzed.

Results: SteLL significantly reduced stress-induced anxiety and depression in behavioral tests. It increased brain levels of serotonin and noradrenaline, reduced oxidative stress, and enhanced antioxidant defenses. SteLL also raised anti-inflammatory cytokine IL-4 while lowering pro-inflammatory cytokines. However, it did not reduce serum corticosterone levels.

Conclusion: SteLL effectively ameliorates anxiety and depression symptoms in stress-exposed mice, likely by modulating monoamine levels, oxidative stress, and inflammation. These findings support its potential as a natural therapeutic candidate for mood disorders, though further studies are needed to elucidate its mechanisms and clinical applicability.

Biography

Michelle Melgarejo da Rosa, an assistant professor at the University of Pernambuco, Brazil, earned Ph.D. in Neurobiology from the Leibniz Institute for Neurobiology in Magdeburg, Germany. The research specialized in molecular mechanisms involved in synaptic plasticity. In 2023, conducted postdoctoral research at UTHealth in Houston, USA, focusing on reward omission responses. Michelle also served as a visiting professor at the DKFZ in Heidelberg, Germany (2023) and in the laboratory of Dr. Monje at Stanford University (2025). Michelle's research focuses on alternatives for anxiety, depression, cognitive disabilities and neurological tumors and has authored over 50 papers.

Milton C. R. Medeiros[1*], Milton Takeshi Medeiros[2]

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Fibromyalgia treated with Transcranial Direct Current Stimulation (tDCS) and physical exercise

Introdution: Fibromyalgia is a chronic and heterogeneous condition, characterized by diffuse musculoskeletal pain and associated changes, such as anxiety disorder, sleep disorder, depression, cognitive impairment and fatigue. Fibromyalgia is a frequent cause of pain and affects about 2.5 to 3% of the world's population. It predominates in women and is an impacting factor in the worsening of quality of life, causing physical, emotional and financial damage. The pain associated with fibromyalgia often is described as a constant dull ache that has lasted for at least three months. To be considered widespread, the pain must occur on both sides of body and above and below the waist. Other condictions often co-exist with fibromyalgia, such as irritable bowel syndrome, migraine and other types of headaches, intersticial cystitis or painful blader, postural tachycardia syndrome and temporomandibular joint disorders syndrome. While there is no cure for fibromyalgia, a variety of treatment is avaliable. Fibromyalgia treatment involves various methods, such as physical activity, cognitive behavioral therapy, and medications. Unfortunately, these approaches do not offer satisfactory results for most patients. Medications, for example, have low efficacy and significant risks of side effects. The most commonly used drugs are tricyclic antidepressants such as amitriptyline, dual antidepressants such as duloxetine, and antiepileptics such as gabapentin and pregabalin. Although there is no clear understanding of the disease’s etiology, the consensus on its pathogenesis is dysfunction in the central processing of pain perception and control systems that result in a state of increased sensitization to pain and Other stimuli. Neuromodulation, especially transcranial Direct Current Stimulation (tDCS), has been shown to have a significant impact on pain relief and functional improvement of fibromyalgia patients in many studies. The tDCS is a neuromodulation method based on cortical modulation, producing facilitating or inhibitory effects in several brain areas. Popularity of the technique has grown over the past decade, as exemplified in a PubMed search, returning 1,500 published articles containing the phrase “tDCS” between 2011 and 2015, in comparison to just 65 articles published between 2000 and 2005. The tDCS involves the emission of a weak electrical current, traditionally via the placement of two electrodes, one anodal that facilitates neuronal depolarization and another cathodal that hinders neuronal depolarization. The tDCS may offer a new line of treatment for fibromyalgia. Another important modality for treating fibromyalgia is physical activity. International treatment recommendations for fibromyalgia highlight the importance of physical activity combined with patient education. In this research, we will demonstrate the results of tDCS alone and tDCS plus physical activity in the treatment of fibromyalgia.

Materials and Methods: Ten outpatients, all women, with fibromyalgia, participated in the study. All signed the informed consent form after detailed explanations about the method. They used different medications for fibromyalgia (anticonvulsants, dual antidepressants, tricyclic antidepressants and muscle relaxants) alone or in different combinations. All had intense pain, with a Visual Analogue Scale (VAS) of 8 or more. These women were sedentary or did very little physical activity (less than once a week). The medications were maintained, and they were randomized into two groups matched by age. In group 1, five patients, with a mean age of 44.4 years old, received tDCS two milliamps for 20 minutes from Monday to Friday, for four weeks, with the anodal pole in the left motor area (M1) and the supraorbital cathodal pole on the right, without practicing physical activity. In group 2, Five patients, with a mean age of 43.2 years old, received the same tDCS stimulus, but with physical activity (walking) of 150 minutes per week divided into 3 or more times. The VAS was reassessed after 4 weeks of treatment.

Results: The patients, all with severe pain before the procedure (average of 8.5 on the VAS), were evaluated before the start of tDCS and after 4 weeks, on the last day of tDCS. All patients completed the study. Patients in group 1 (tDCS without physical activity) had the following VAS values after 4 weeks: 4, 3, 4, 5, and 3. VAS average of 3.8. Therefore, there was a significant reduction in pain intensity just by applying tDCS. In group 2 (tDCS associated with physical activity), the results were even better, with VAS after 4 weeks of 1, 2, 2,1 and 2. VAS average of 1,6. The differences between group 1 (tDCS without physical activity) and group 2 (tDCS associated with physical activity) were statistically significant, with t (t student) of -4.91 and calculation of p showing very small number. There were no reports of significant side effects. In general, there were discrete paresthetic sensations of short duration at the site of electrical stimulation.

Discussion: In this small randomized sample, it was possible to observe significant pain relief in patients with fibromyalgia. Studies have shown that the anodal stimulus in the primary motor area should be the one of choice for the improvement of pain sensations. The procedure is cheap, safe and very well tolerated by patients, showing itself as a promising therapeutic technique for chronic pain in patients with fibromyalgia. In this study, the symptoms that accompany pain in fibromyalgia, such as sleep disturbances, fatigue, memory disorders, depression, or anxiety, were not evaluated. But, there are studies showing that pain is not the only symptom to improve in fibromyalgia, and there is an improvement in quality of life with the use of tDCS. Regarding physical activity, several studies show its effectiveness for pain relief in fibromyalgia. In our study, it was very evident that the association of tDCS with physical activity was much more effective than tDCS alone.

Conclusion: tDCS is a very promising method for the treatment of fibromyalgia, mainly due to its safety, low cost, and possible good efficacy Our study suggests that the association of physical activity with tDCS intensifies pain improvement, when compared to tDCS alone. Studies with a larger number of patients should be carried out for more robust conclusions.

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 (UEL) in 1994. 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. And, a writer, being the author of 6 books and several scientific articles on neurology, in national and international congresses and journals. Latest book is “Fuja do Azheimer Agora Mesmo”, Publisher Viseu, still an exclusive edition in portugueses.

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Genetic code, neurocode and prospects for developing strong artificial intelligence

Further research into the genetic code and its physical properties is an important resource for decoding the neurocode and developing strong artificial intelligence.

DNA has a hierarchical spiral structure, which still remains outside of the attention of science. Meanwhile, it contains part of the genetic information and is a source of a long-range electromagnetic field. Thanks to it, the DNA ensemble forms a single interference pattern. The hierarchy of code fields is a set of programs embedded in one another and performing semantic autodistillation and evaluation of incoming signals. At the same time, a new quality is born: Code structures for regulating physiological processes and brain neurodynamic systems-codes of mental processes.

Neurocode is an active self-reproducing information carrier that performs the functions of a program for managing and regulating physiological processes and forms the fundamental foundations of the psyche. It is an operating system of consciousness, similar in a functional sense to the genetic code.

Consciousness represents the highest levels of self-organization processes included in the scheme of the genetic code. These levels rely on the underlying ones and use their results. Therefore, when studying neurocode, it is important to study the physical means of encoding and decoding information in the nervous system.

The study and decoding of neurocode is one of the main tasks of modern neuroscience. But how does the encoding of external and internal stimuli in the body occur in the nervous system, and then their decoding, “understanding” of their significance for life and for the response?

Although neurocode research has a long history, only recently a new paradigm in solving this problem has appeared. Its essence is that the neural code is not implemented by discrete, identical spikes, but includes an analog (wave) component (it differs in period, amplitude and shape of the phase portrait). In this sense, the neurocode is not digital, but analog-digital and therefore is capable of encoding very large volumes of information in the shortest periods of time.

The problem of decoding the neurocode also includes the inverse problem: Restoring the content of the signal using a neurocomputer interface based on the activation of brain processes that were defined as the code of this content. This direction of neuroscience "Brain Reading" has achieved significant results.

When they talk about the genome mind and its creative capabilities, they mean numerous information competencies that have been developed in the course of biological evolution and are effective ways of managing complex life processes. But it still remains nothing more than a student of the Genomic Mind.

Turning to the genome as the primary source of our mind is an important condition for further study of the neurocode, development of Artificial Intelligence (AI) systems capable of performing the functions of natural intelligence.

Genomic AI (GAI) is a substrate that includes some forms of the genome and its environment-the environment, independently, as a result of self-organization, establishes its state depending on external circumstances.

For these purposes, you can use: DNA extracted from neurons, whole samples and its fragments. Environment-water compositions, gels, etc.

The physical result of the GAI work is a multi-level state of substances, fields and radiation in terms of scale, time, topology and vibration spectrum. It can manifest itself in the form of stable patterns of matter or oscillating interference of waves of various natures. This is a complex and unpredictable state.

Methods of activation, training and self-development of GAI are considered. The possibility of using this concept to create artificial self-reproducing codes similar to the genome is considered.

Biography

Dr. Badyagin completed studies at the Ryazan Radio Engineering Institute in Russia back in 1984. After that, worked at the Moscow Institute of Electronic Technology. In 1997, earned PhD from the same institute. Currently, Dr. Badyagin is a professor at the Ryazan State Medical University named after Academician I.P. Pavlov in Russia. Over the years, Dr. Badyagin published more than 80 research papers in various journals.

Rahul Hajare

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The clinical features of children with autism spectrum disorder in India

The children's sex and age and the children's parents' age and education level, when a diagnosis was determined. We also studied clinical features, including the child's age at the time of early alert signs and at diagnosis, the core alert sign and symptom(s) at diagnosis, and the presence and severity of ASD comorbidities. Of the 180 children involved in this study, 90 were boys (72.5%) and 90 were girls (27.50%). The mean age (SD) at early alert signs and diagnosis was 1.8 (SD=.78) and 7.83 (SD=3.4) years. According to the children's parents, 54% reported communication disorders as the main alert sign, while 62% noted these disorders were the main symptom of their child's ASD. Behavioral and interaction disorders were the other most common characteristics parents identified. The core symptoms of ASD were correlated with epilepsy (p=.027), cerebral palsy (p=.026), and hearing impairment (p=.045). The main clinical features of all 180 children were as follows: Delays in diagnosis and speech co-occurring with epilepsy and a hearing impairment. Taken together, they indicate the severity of ASD in India.

Biography

Dr. Rahul Hajare attends the Vedanta Institute in Kolkata, graduated from Nagpur University with a M. Pharm passed with distinction. Dr. Rahul works as an academic at Sandip University and attends the Hindu University of America as a scholar. Also, works as a postdoctoral fellow at the Indian Council of Medical Research's Dr. Ramesh S. Paranjape National AIDS Research Institute, which is well-known and highly esteemed worldwide.

Robert B. Slocum
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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. We 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.

Biography

Robert B. Slocum is the Narrative Medicine Program Coordinator at University of Kentucky HealthCare and holds a 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. Has taught undergraduate courses in religious studies and ethics. 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. A member of the Hospital Ethics Committee, and the author, editor, or co-editor of 14 books, including a journal of reflections. His 36 articles have appeared in theological or medical journals and as book chapters, and has made presentations at more than two dozen theological and medical conferences. 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 and sees Narrative Medicine as a bridge between medical humanities and clinical practice.

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Neurophysiological grading tool of ulnar nerve entrapment across wrist and across elbow with case presentation

Ulnar Nerve Entrapment Across the Elbow (UNEAE) and Across Wrist (UNEAW) is the second most common entrapment of the hand after carpal tunnel syndrome. There are few gradings available for UNEAE and lesser in UNEAW.

The aim of this research is;

1. To create a clinically appropriate ulnar nerve entrapment grading tool to covers both area of entrapment in one research paper.
2. To see the relation of sensory nerve involvement across wrist with the entrapment across elbow and to evaluate its effectiveness in terms of compatibility with previous research, without any invasive tests like needle EMG examination.
3. To identify the lesion below and across wrist in terms of to support the Clinical Physiologist (CP) to grade them properly and also help the consultant in deciding to treat with conservative or surgical treatment.
4. To compare the recording from the First Dorsal Interosseous (FDI) muscles with the Abductor Digiti Minimi (ADM) muscle to see which muscle is more sensitive and shows early changes in ulnar nerve entrapment.

The proposed revised grading system is based on more nuanced, descriptive categories, ranging from "normal", "early", "mild", "moderate" and "severe". To create full grading system of UNEAW and UNEAE some additional category of clinical grading is therefore proposed.

Biography

Salim Hirani is working in Neurophysiology field for more than 30 years and did is Neurophysiology course from United Kingdom. Salim Hirani works in different country and can speak 4-5 languages. Three research papers was already published i.e. Refine Grading of Carpal Tunnel syndrome in BMC journal in 2019, Neurophysiological Grading tools of ulnar nerve entrapment across elbow in Journal of Neurology, Neurological Science and Disorders in 2023 and third paper of Neurophysiological Study for Ulnar Entrapment at Wrist (meddocsonline.org) in Journal of Psychiatry and Behavioural Sciences in June 2023.

Sang-Hie Lee[1*] PhD, EdD, MM; Juan Sanchez-Ramos[2] MD, PhD; Nancy Burns[3] PhD, LAT, ATC; Saida Omarova[4] MD; Stephanie Carey, PhD[5]

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Neurophenomenology in retraining musicians with task-specific focal dystonia: Three cases

Introduction: Musicians experience a high rate of playing-related pain and injury due to the rigorous nature of repetitive mind-body training, specifically Musculoskeletal Disorders (PRMD), Performance Anxiety (PA), and Task-Specific Neurological Disorder (TSND). TSND is the least understood by the performing artists. Neurophysiological evidence has demonstrated that extensive training and excessive practice can yield maladaptive changes in certain neural networks, leading to task-specific focal dystonia. Mindfulness training with biomechanical pedagogy has shown promise in modifying the neurological behavior pattern. Purpose: In this presentation, we share retraining coaching session details of three professional task-specific focal dystonic musicians. We use neurophenomenological first- and second-person method to frame our mindfulness & mind-body integrative rehabilitation process.

Approach: We experimented with mindfulness meditation in mind-body coaching with three professional musicians, who developed task-specific focal dystonia at the height of their careers. The sessions consist of deep breathing, in-the-moment focus, mental imagery, anatomical and proprioceptive awareness, and deliberate slow playing.

Results: Musicians showed progress in rehabilitation after a few sessions, however, we did not test long-term effects. Sustained and permanent behavioral change will require an extended period of second-person neurophenomenological retraining to increase awareness and proprioception.

Conclusion: Gradual retraining process of mindfulness mind-body experience-based neurophenomenological process is an appropriate model to teach our non-dystonic pre-professional collegiate musicians in a hurried and quantity-oriented modern environment. A third-person external feedback device and brain image monitoring can add to the efficacy of the neurophenomenology.

Keywords: Maladaptive Sensorimotor Plasticity, Retraining Skilled Musicians with Task-Specific Focal Dystonia, Neurophenomenology, Mindfulness Pedagogy.

Biography

Sang-Hie Lee is a performing artist and music performance science researcher. Lee's research interests include pianists' hand biomechanics, collegiate musicians' health intervention program, and organizational conditions and music faculty vitality. Sang-Hie Lee is the author of Scholarly Research in Music: Shared and Disciplinary-Specific Practices (Routledge 2022) and principal editor of Perspectives in Performing Arts Medicine: A Multidisciplinary Approach (Springer 2020). Sang-Hie Lee has published 74 scholarly articles, presented 85 conference papers, keynotes, and lectures, and hosted eight international conferences and served as Associate Dean of the College of Fine Arts. Currently as the Director of University of South Florida Performing Arts Medicine Collaborative.

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Antibody-proteases as the upgraded translational tools of the next-step generation in personalized and precision practice to monitor multiple sclerosis at clinical and subclinical stages

Among the best-validated predictive biomarkers are autoimmunity-related ones to predict and prognosticate risks of the chronification, complications and thus disabling. The latter is so much valuable and important since chronic autoimmune inflammation course is structured to consist from different stages including subclinical and clinical ones.

Multiple Sclerosis (MS) is just one of the chronic tissue-specific autoimmune diseases resulting in a destruction of myelin by different tools, including autoAbs of very broad specificity. Along with canonical Abs, some of the families proven to occur are Abs possessing with catalytic activity (abzymes), and thus to belong to Abs with functionality!

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

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 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, were shown to be attacked by Ab-proteases in MS patients with moderate (remission-type) courses.

The activity of Ab-proteases was first registered at the subclinical stages 1-2 years prior to the clinical illness. About 24% of the direct MS-related relatives were seropositive for low-active Ab-proteases from which 22% 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 subclinical stages of MS), 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. And the “escalation” illustrating re-orientation of the sequence specificity to focus on the more important targeted sites for proteolysis might be an early prognostic and/or predictive sign to monitor demyelination progressing and thus the clinical illness to come. 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.

Sequence-specific Ab-proteases have proved to be greatly informative and thus valuable biomarkers to monitor MS at both subclinical and clinical stages! And the translational potential of this knowledge is in the rational design of new diagnostic tools and new therapeutics based on principles of artificial biocatalysts and biodesign.

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. Therefore, the proposed predictive value of MBP-targeted Ab-proteases for the development of MS is challenged! So, further studies on Ab-mediated MBP degradation and other targeted Ab-mediated proteolysis may provide biomarkers of newer generations and thus a supplementary tool for assessing the disease pro-gression and predicting disability of the patients and persons-at-risks

Biography

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

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New vision for early detection of alzheimer's disease

Background: Recent research on Alzheimer's Disease (AD) has highlighted that the oxidative damage is the earliest event of disease. These oxidative modifications are closely associated with inflammatory molecules. It is necessary to explore these two pathways with AD pathophysiology and targeted for therapeutic intervention. At present, the most validated AD biomarker is A levels in the Cerebrospinal Fluid (CSF) and tau PET scan. None of the techniques can be used to monitor the disease with the response of treatment. The present study focussed on novel molecules in these two pathways for establishing blood-based biomarker and therapeutic interventions.

Method: Blood samples were collected from 46 AD patients, 37 Mild Cognitive Impairment (MCI) patients and 35 Geriatric-Control (GC). The levels of inflammatory molecules (LOX-5, LOX-12, GSK- 3, p53, NFkb) and antioxidant molecules (FOXO, Sestrin, Sirtuins) were measured by using surface plasmon resonance and verified using Western blot in serum from AD, MCI, and GC. Statistical analysis, including Receiver Operating Characteristic (ROC), was done for further affirmation with all demographic data and clinical assessment score. The Alzheimer's disease model neurotoxic SH-SY5Y cell line was treated with antioxidant plant extract for rescue effect on SH-SY5Y to see the effect on expression level of above mentioned proteins.

Results: The significant alteration of some of these proteins were observed in the blood of AD patients compare to MCI and geriatric control. These proteins also showed positive correlation with the known hallmark Tau, pTau and aβ-amyloid in the study group. A significant ($p<0.0001$) downhill correlation was found between Tau as well as p-Tau181 levels with HMSE and MoCA score. The treatment with antioxidant plant extract showed rescue effect of neurotoxicity by enhancing antioxidant enzymes thereby decreased Reactive Oxygen Species (ROS).

Conclusion: These proteins can serve as potential blood markers for the diagnosis of AD and supplementary antioxidant molecules can regulate and suppress their level by rescuing the neurotoxicity. This work has translational value and clinical utility in the future.

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Role of alzheimer's disease presenilin-1 associated protein in mitochondrial apoptosis

The process of apoptosis, a programmed form of cell death, is a normal occurrence during neurogenesis in the maturation of the central nervous system. However, dysregulated or premature activation of apoptosis can contribute to the pathogenesis of various neurodegenerative disorders. Indeed, studies have implicated that apoptosis plays a pivotal role in the pathogenesis of several neurodegenerative disorders including Alzheimer's Disease (AD), Parkinson's disease, Huntington's disease, and Amyotrophic Lateral Sclerosis (ALS). Early studies have demonstrated the involvement of both Presenilin-1 (PS1) and Presenilin-2 (PS2), proteins associated with Alzheimer's disease, in apoptosis. However, the exact mechanism is unknown. In an attempt to investigate the pathogenic function of PS1, using the yeast-two hybrid system, our study identified a novel PS-1-Associated Protein (PSAP). Subsequently, our studies revealed that PSAP functions as a proapoptotic molecule localized in mitochondri. This discovery establishes a direct molecular connection between PS1 and the apoptotic pathway, thereby paving the way for further exploration into the molecular mechanisms underlying PS-1-mediated apoptosis.

In a subsequent study, utilizing siRNA and knockout cell methodologies, we unveiled that PS1-induced apoptosis is dependent on mitochondria and primarily mediated by PSAP. Furthermore, to further elucidate the mechanism underlying PSAP-mediated apoptosis, our investigations demonstrated that PSAP triggers a distinct mitochondrial apoptotic pathway that operates independently of regulation by Bcl-2 family proteins. The apoptotic cascades leading to PSAP-mediated mitochondrial apoptosis were further investigated through a subsequent round of yeast-two hybrid system screening, revealing an interaction between PSAP and the death receptor DR6. Interestingly, PSAP is absolutely required for DR6-induced apoptosis in mediating DR6-induced Bax translocation of DR6 from cytosol to mitochondria. These results suggest PSAP may function as an anchor of Bax on the mitochondria. These novel observations might be exploited to develop a novel drug approach to manage AD.

Biography

Dr. Xuemin Xu earned PhD in molecular biology from Tokyo Institute of Technology in Japan. Following a four-year postdoctoral fellowship at Scripps Research Institute, Dr. Xu joined Case Western Reserve University as an assistant professor in 1994 and was recruited to the University of Tennessee as an Associate Professor in 1999 and promoted to full professorship in 2007. In 2018, Dr. Xu was appointed as the Dr. John Doran Endowed Professor in Neurobiology at The University of Texas Permian Basin. Throughout his career, Dr. Xu has published over 70 research articles in SCI (E) journals.

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Neuroimmune mechanisms in nasal hyperreactivity: Unraveling the nose-brain axis

Nasal Hyperreactivity (NHR), including conditions such as allergic rhinitis, represents a group of common disorders characterized by complex neuroimmune interactions, with underlying mechanisms that are not fully understood. Communication between the nervous and immune systems plays a pivotal role in host-protective immunity at the nasal mucosa. In NHR, external stimuli such as allergens, thermal variations, mechanical, chemical, and biological factors directly impact nociceptive afferents and adjacent tissues, triggering the release of neuropeptides such as CGRP and substance P, as well as pro-inflammatory cytokines including IL-4, IL-5, and IL-33. These interactions regulate both innate and adaptive immune responses through neuroimmune pathways. This response not only exacerbates local neuroimmune inflammation in the nasal mucosa but also transmits external signals to the central nervous system via afferent nerves. Furthermore, recent studies highlight the role of the body-brain axis in mediating communication between the central and peripheral systems, with specific brain regions exerting significant regulatory effects on peripheral immune activity. Central activation resulting from nasal inflammation may, through efferent neural pathways, further aggravate nasal hypersensitivity, creating a bidirectional amplification of neuroimmune loops. This central-peripheral feedback loop may represent a key driving force behind the persistent pathophysiological changes in NHR. This review aims to elucidate the neuroimmune mechanisms underlying NHR, focusing particularly on the "nose-brain axis," a bidirectional communication pathway linking the peripheral immune system with the Central Nervous System (CNS). We propose that further exploration of how neuroimmune interactions shape NHR via the nose-brain axis in experimental models is essential. These findings underscore the potential for innovative therapeutic strategies targeting neuroimmune interactions as an effective approach for treating NHR.

Biography

Mr. Yanjie Wang is currently a Ph.D. candidate in Otorhinolaryngology at Shanxi Medical University, obtained M.S. degree from Shanxi Medical University in 2020 and subsequently served as a Lecturer and Physician at Second Hospital. Since 2022, has been conducting doctoral research under Prof. Changqing Zhao's mentorship, focusing on neuroimmune mechanisms of airway hyperresponsiveness, particularly the interplay between peripheral inflammation and central neural regulation. Mr. Yanjie has participated in 3 National Natural Science Foundation projects and led 2 provincial grants. And, Yanjie's work has resulted in 10+ SCI and core journal publications, with presentations at multiple national conferences.

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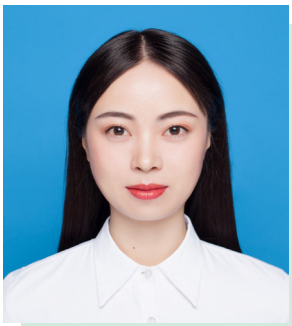
The loss of PINK1 function alters molecular pathways in human fibroblasts

Parkinson's Disease (PD) is the most common neurodegenerative movement disorder. Mutations in the PINK1 and PRKN genes have been linked to early-onset PD and are among one of the most frequent causes for inherited, recessive PD. PINK1 and PRKN together eliminate damaged mitochondria through a mitophagy pathway and the failure of this stress-dependent protective pathway might be linked to pathogenesis of PD. In fact, several studies have shown increased mitochondrial damage and the involvement of mitochondrial quality control in PD. Therefore, it is likely that failure of the PINK1/PRKN pathway also plays a role for idiopathic disease. However, a specific signature of mitophagy failure is not known yet. Here, we used transcriptomic and metabolomics analysis of human fibroblast samples with PINK1 complete loss of function, partial loss of function, and wild type to find such molecular fingerprint. We identified genes differentially expressed by PINK1 loss of function and found that they were enriched in pathways including complement system, STAT3 signaling, axonal guidance signaling and serotonin degradation. Additionally, using weighted gene co-expression network analysis, we found that PINK1 loss of function was associated with upregulated immune response and completement activation. Integrative analysis between transcriptomics and metabolomics revealed enriched purine metabolism. We validated the top genes and pathways other independent RNAseq experiments and in the wet lab. Our study nominates several molecular pathways that can be used as potential surrogates for loss of PINK1 function. These pathways can now be further exploited as biomarkers for mitophagy function and might be important for patient stratification in the future.

Biography

Dr. Ren is an Assistant Professor of Biomedical Informatics in the Department of Quantitative Health Sciences at Mayo Clinic. Dr. Ren's research focuses on developing and applying novel bioinformatics approaches to translational science to help identify novel disease risk factors and therapeutic targets, specifically for neurodegenerative diseases. Dr. Ren expertise is omics analysis of disease models, including whole exome and genome sequencing, bulk and single cell transcriptomics, cell type deconvolution, proteomics, lipidomics, metabolomics, epigenomics, as well as multiomics integration. Currently, Dr. Ren is co-PI or co-I on 9 NIH or foundation grants, and has authored 48 publications.



Yang Fan, Xiaodong Zhang and Yufan He*

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Mature B-cell lymphoma with acute myelitis as the first presentation: A case report and literature review

Background: Lymphomas are malignant neoplasms of the immune system, originating in lymphoid organs and having the potential to affect the central nervous system. Notably, lymphomas presenting initially as acute myelitis are extremely rare. Most of these cases exhibit symptoms of spinal-cord impairment, mainly due to the lack of specificity in their clinical manifestations. This leads to a very high rate of early misdiagnosis and a poor prognosis. Herein, we present a case of mature B-cell lymphoma with acute myelitis as the initial manifestation and conduct a review of the relevant literature.

Case Presentation: In this study, we report on a 70-year-old male patient who presented with bilateral lower-extremity weakness, bowel and bladder dysfunction, and recurrent fever. Imaging revealed a paraureteral mass beneath the right kidney. The final pathological biopsy results indicated CD20 (+), a mature B-cell tumor. However, the patient declined further tests to determine the specific type of lymphoma and subsequent treatment, and requested discharge. Unfortunately, the patient passed away in mid - November 2020.

Conclusions: This case report highlights that lymphoma patients may present with acute myelitis as the first symptom, especially when accompanied by recurrent fever. Conventional myelitis treatment may be ineffective in such cases. Once other causes of myelitis have been excluded, tumors should be considered. Moreover, a targeted search for tumor-related evidence, along with early detection and treatment, may potentially improve patient survival.

Keywords: Acute Myelitis, Mature B-Cell Lymphoma, Secondary Central Nervous System Lymphoma, Case Report.

Biography

Yufan He is an attending physician with 6 years of clinical experience, having worked at Sichuan Provincial People's Hospital and Sichuan Xindu District Traditional Chinese Medicine Hospital. He is currently pursuing postgraduate studies at North Sichuan Medical College. His research focuses on the molecular and neural circuit mechanisms of neuropathic pain, under the supervision of Professor Xiaodong Zhang.

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Guidelines application for the treatment of TBI in children a multidisciplinary approach

Introduction: The main aim of management of pediatric severe Brain Injury (TBI) is to hold normal ranges for optimizing the most favorable outcomes.Clinical and Research Institute of Emergency Pediatric Surgery and Trauma. Annually, more than 10000 children with head injury are consulted in the institute's admission department. The number of children with severe brain injury is not large, but these patients are very difficult for treatment (3%)

The aim of the study is to demonstrate the effectiveness of using clinical recommendations and a multidisciplinary approach to the treatment of pediatric TBI. Since 2004, in our Institute basic principles of sTBI curative strategies have been developed in compliance with contemporary guidelines. Additionally, we have developed the prognosis of outcomes which we utilize as a tool for treatment control. So, the philosophy of treating severe TBI includes some important tasks: Control of intracranial and extracranial complications; ICP control, early surgical treatment of sTBI complication, including cranioplasty, acute and early rehabilitation, multidisciplinary approach.

Results: 311 patients (GCS 3-8) were admitted to our hospital from 2004 till 2018. 58% of them had combined trauma. All patients had ICP control. 97 p-ts had decompressive craniectomy. Early rehabilitation begins after stabilization of vital functions. 44% of patients had reconstructive surgeries because of extensive defects of the scull. The average time for defect closure was 58±12 days. 12 % of patients were operated on for posttraumatic hydrocephaly. A month after the injury, good outcomes (GOS 4-5) were in 42%; 33.5% had GOS 3-4; 24.5%-GOS 1-2. 6 months later, 69% of survivors had good results.

Conclusion: Prognosis and outcomes are largely determined by a timely and fully provided specialized urgent medical aid. More than 30 different specialists may take part in the treatment- from paramedic to social worke. From this point of view, successive and continuous multidisciplinary approach is very important. The timely recovery and support of vital functions, “physiological corridor”, early surgical treatment of complications, early rehabilitation allow to optimize outcomes in children with severe traumatic brain injury.

Biography

Dr. Zhanna Semenova is Pediatric Neurosurgeon, Head of Neurosurgery and Neurotrauma at the Research Institute of Emergency Pediatric Surgery and Trauma and Chief of Pediatric Neurosurgeons in Moscow (2015) and the Central Federal District (2016). Since 2018, they have been a professor at the Russian Medical Academy of Postgraduate Education. Dr. Zhanna Semenova is President of the Society for Pediatric Neurosurgery of Russia and a member of ISPN, EANS, and the IPBIS. Main scientific topics-severe TBI, ICP control, decompressive craniectomy, prognosis and outcomes of TBI, syndrome of trephined, non-invasive ICP.

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11th Edition of International Conference on

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JUNE

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POSTER PRESENTATIONS

Bartłomiej Stańczykiewicz[1*], Józefa Macała[2], Krzysztof Gołąb[3], Błażej Misiak[1], Agnieszka Zabłocka[2]

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Impact of egg-derived proteins, yokan polypeptide complex and ovocystatin, on blood-brain barrier integrity and function

Introduction: The Blood-Brain Barrier (BBB) is a highly selective and protective membrane that separates the brain from the circulating blood, effectively shielding it from harmful substances, toxins and pathogens. Its primary function is to maintain brain homeostasis, regulate the movement of molecules and prevent the onset of neuroinflammation. The endothelial cells of the BBB play a critical role in this process. They have the ability to secrete cytokines in response to stress or inflammation, which helps to regulate the immune response and maintain the integrity of the barrier. When the integrity of the BBB is compromised or dysregulated, it can lead to a range of neurological disorders, highlighting the importance of maintaining its stability for brain health.

Aim: Egg-derived proteins, such as yolkin polypetide complex and ovocystatin, may serve as potential nutraceuticals that support BBB integrity and function.

Material and Methods: The effects of yolkin polypeptide complex and ovocystatin on BBB function/integrity were investigated. Human cerebral microvascular endothelial cells HBEC5i (ATCC) were used as an experimental model of the BBB. Cell viability was assessed using the MTT assay. Changes in Tight Junction (TJ) protein expression were determined by Western blotting. HBEC5i Permeability (PE) measurements to the FITC-dextran molecule were assessed. Cytokine levels: TNFα, IL-1β, IL-6, IL-8 and IL-10 were determined by ELISA.

Results: Our study showed that yolkin and ovocystatin did not affect the viability of HBEC-5i cells. However, yolkin was found to modulate BBB permeability under inflammatory conditions as evidenced by changes in FITC-dextran permeability measurements. Western blot analysis showed no significant changes in TJ proteins, including ZO-1, E-cadherin and occludins. On the other hand, ELISA results showed that yolkin and ovocystatin selectively stimulated the secretion of IL-1β and IL-8 to the luminal side of the BBB, whereas no significant changes in the secretion of TNF-α, IL-6 or IL-10 were observed.

Conclusion: These findings suggest that yolkin and ovocystatin are non-toxic to the brain endothelial cells and affect BBB function by modulating permeability and cytokine production. By selectively stimulating the release of cytokines, such as proinflammatory IL-1β and chemotactic/angiogenic IL-8, they play a critical role in protecting and restoring BBB integrity during immune challenge.

Biography

Prof. Bartłomiej Stańczykiewicz works at the Division of Consultation Psychiatry and Neuroscience, Department of Psychiatry, Wroclaw Medical University in Poland. Prof. Bartłomiej's research integrates biological and psychological approaches in studies regarding mental health issues. Prof. Bartłomiej Stańczykiewicz primary focus is to identify the possible factors that inhibit the development of pathology in neurodegenerative disorders. In addition, Prof. Bartłomiej interested in inflammation in schizophrenia spectrum and neurodegenerative disorders, supportive biological and psychological approaches in mood and anxiety disorders, as well as in age-related cognitive impairment. Also interested in studies investigating factors associated with minority stress in the context of mental health.

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Deepthickness: A novel deep learning method for estimating cortical thickness trajectories in healthy and alzheimer's disease populations

Alzheimer's Disease (AD) is a neurodegenerative disease that presents with critical challenges in diagnosis and treatment. Emerging research indicates that AD-related cortical changes, such as cortical thickness, can appear up to a decade before cognitive symptoms. Accurately measuring cortical thickness can therefore offer a significant avenue for early AD diagnosis and monitoring of clinical progression. Automatic techniques, such as FreeSurfer and CAT12 Toolbox, offer out-of-the-box cortical thickness estimates, but with an excessively long computational time (up to 10 hours per volume), systematic differences between approaches and significant errors when applied to clinical data. We propose Deep thickness; the first Deep Learning-based approach for estimating cortical thickness from structural MRI in just a few seconds. Our method utilises recent advances in deep learning to generate white matter and pial surface mesh reconstructions with cortical thickness estimates as an overlay. We report promising preliminary findings, highlighting our method's similarity to freesurfer in mesh generation and cortical thickness estimations while accounting the software's identified limitations. Leveraging comprehensive clinical datasets, we also showcase our method's use for mapping cortical thickness, cognition and other clinically relevant trajectories over time for healthy, MCI and AD populations.

Biography

Connor Dalby is a 2nd year PhD student studying at the University of Glasgow, UK. Connor Dalby previously graduated with a Bsc (Hons) Psychology and MSc Molecular Neuroscience and has since spent several years working in clinical trials research for neurodegenerative diseases. Connor is now combining newly developed skills in artificial intelligence with neuroscience and clinical expertise to find novel innovations for healthcare.

Cristian Ravariu

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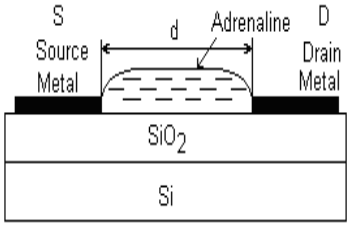
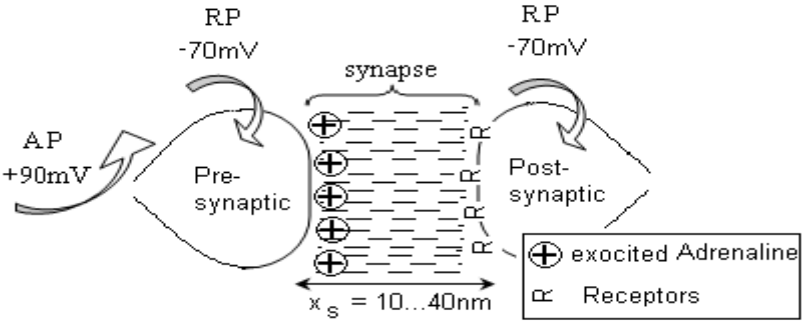
Biomimetic electronic devices for adrenergic synapsis modeling

The importance of the adrenergic transport mechanisms comprehension in the synapses is useful in clinics diagnosis and pharmacology. For this aim, the paper proposes a parallel biodevice composed by Adrenaline Bioliquid on Insulator. Between the source and drain of the device is developing an electric field to drive the neurotransmitter molecules in DC regime. From the polarographic curves the diffusion coefficient of adrenaline was extracted. The switch regime estimated the variations of the current versus time when the external voltage was modified, so that the biodevice overvoltage reaches to the action potential. In this way, a similar model with the real synapses is taken into account. In the case of the pre-synaptic button and post-synaptic cell membrane, some calculations were included and intend to support the proposed model. Figure 1 presents the synapse model. Here the Resting Potential (RP) is -70mV and the Action Potential (AP) is +90mV. Hence, the maximum potential drop over synapse is 160mV, when the pre-synaptic button is charged at +90mV and the post-synaptic cell membrane is still in resting state at -70mV.



(a)



(b)

(a) A cross-section through structure; (b) The Action Potential arrival, followed by the adrenaline releasing into synapse.

In conclusion, besides to the diffusion current still exists the drift component, the j_{diff}/j_{drift} ratio ranging between 11/0.0049 and 11/56, depending on the admitted electric field onto synapse.

Biography

Prof. C. Ravariu studied Microelectronics at the Polytechnic University of Bucharest, Romania and graduated as MS in 1993. Prof. C. Ravariu worked as scientific researcher first 5 years at Institute of Microtechnology, Bucharest, then joined the Polytechnic University of Bucharest. After multiple foreign stages in Bioelectronics (Patras, Greece), Nano-devices (EPFL, Switzerland), Organic Electronics (LAAS, France), received PostDoc degree in 2012 in Romania. Since 2013, obtained the position of Full Professor at the Polytechnic University of Bucharest, Faculty of Electronics, Romania. Prof. C. Ravariu has published more than 250 research articles. Since 2014, Prof. C. Ravariu is Chairman of the Romanian IEEE Electron Devices Chapter and main interest is in nano-bio-devices for electronics and biomedical research.

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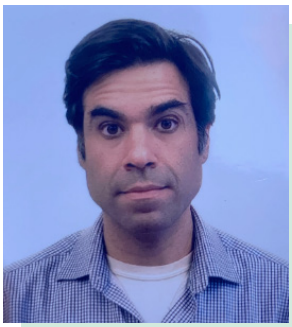
Improvement of brain function by wasabi component “Hexaraphane”

Hexaraphane (6-Methylsulfinylhexyl Isothiocyanate; 6-MSITC) is an isothiocyanate present in the rhizomes and roots of Wasabi (Eutrema japonicum (Miq.) Kiudz.). Here we present the results of two clinical trials conducted on healthy subjects and patients with chronic fatigue syndrome using an extract powder "Wasabi SulfinylTM (WS)" containing 0.8% hexaraphane. In randomized controlled trial conducted on healthy middle-aged and elderly subjects improved memory for three months intaking of 100mg WS. In a clinical trial of fifteen patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) treatment with 1.2g WS for three months improved brain fog and other symptoms.

Biography

Isao Okunishi studied Molecular Biology at Nagoya University, Japan, and graduated as MS in 1995. And, then worked on functional research of Wasabi at Kinjirushi Co., Ltd. and received Ph.D. (Agriculture) from Kagoshima University in 2022. Isao Okunishi has published several papers and books on the functionality of Wasabi, also obtained nearly 30 patents related to his research.



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Alzheimer's Disease (AD) with preexisting hypofrontality versus frontal AD: Radiologic and neuropsychological data in a case of likely dysexecutive alzheimer's disease in a patient with schizophrenia

Frontal Alzheimer's Disease (fAD) is an atypical AD variant which is difficult to differentiate from and frequently misdiagnosed as behavioural variant Frontotemporal Dementia (bvFTD). Most recent work has identified two subtypes of fAD: a behavioural subtype (bAD) associated with changes in personality, behaviour, which may include impulsivity, agitation, aggression, apathy and social cognition, and a dysexecutive subtype (dAD) associated with issues in planning, problem-solving, and other aspects of executive functioning. bAD and dAD are poorly understood in terms of cognitive and behavioural clinical presentations, as well as in a "typical" radiographic pattern of atrophy and hypometabolism beyond predominantly frontal and anterior temporal lobe involvement. This report describes a case of a 61-year-old male patient with a several-decade history schizophrenia, a primary psychiatric condition associated with structural and functional frontal lobe abnormalities, who presented with insidious onset and gradual worsening over the most recent 6-7 months of short-term memory difficulties, apathy, disinterest and anhedonia, with no reported mood changes. Family collateral report indicated significant decline in instrumental activities of daily living (particularly those requiring planning), and recent difficulties in language comprehension. FDG-PET and MRI scans indicated elements consistent with FTLD (frontal and temporal hypometabolism involving superior medial frontal and inferior frontal gyri, anterior cingulate gyrus, Heschl's gyrus, and caudate nucleus), typical AD (posterior cingulate and precuneus hypometabolism, pronounced bilateral mesial temporal lobe atrophy), mild white matter disease (T_2-FLAIR subcortical/periventricular hyperintensities), and there was additional suggestion that findings may reflect long-term effects of schizophrenia and/or chronic antipsychotic use. Neuropsychological evaluation revealed significant dysexecutive features in working memory, strategy formation, behavioural inhibition, visuoconstructional planning, and verbal generativity, as well as poor confrontation naming and slowed encoding and variable storage and retrieval deficits for nonverbal and verbal non-contextual and contextual information. Deficits were not consistent with an FTLD-related Primary Progressive Aphasia (PPA) or bvFTD alone, nor were they strongly consistent with typical or early onset AD, schizophrenia, or vascularly-derived neurocognitive disorder given the range and severity of deficits observed; while presentation and reported history was not consistent with bAD or bvFTD or any PPA subtype. From these data, a likely dAD diagnosis is offered, which adds to our current, limited understanding of the condition. Additional radiographic workup such as amyloid or tau-PET may be beneficial to confirm diagnosis and elucidate patterns of structural abnormality and/or hypometabolism to improve diagnostic speed and clarity in the dAD and bAD phenotypes of fAD.

Biography

Dr. James S. Maniscalco is a neuropsychologist specializing in aging and dementia and neurodegenerative diseases at Northwell Health-Staten Island University Hospital. He graduated from the Clinical Psychology with Emphasis in Neuropsychology doctoral program of the CUNY Graduate Center and Queens College in 2021 and holds additional M.Phil. and MA degrees in psychology (2017) and mental health counseling (2011). He has conducted research in normal pressure hydrocephalus, stroke rehabilitation (in aphasia and visuospatial neglect), vascular dementia, and schizophrenia, as well as in emotion-cognition relationships in healthy individuals.

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Validation study of the cogniscan computerized test for memory and processing speed in a Brazilian sample

Objective: To evaluate the psychometric properties of the Cogniscan screening test, a self-administered software for assessing episodic and working memory, focusing on validating its accuracy in a Brazilian clinical sample.

Participants and Methods: This study involves 300 volunteers aged 18 years or older with at least four years of schooling. Participants will answer a sociodemographic and quality of life questionnaire, in addition to taking the G-38 intelligence test, the Recognition Memory Test (TEM-R-2), attention tests (AOL) and the computerized Cogniscan, which assesses episodic and working memory and processing speed. The results of the Cogniscan are expected to be equivalent to those of the TEM-R-2, showing impressive reliability and construct validity indices.

Results: Preliminary data from 54 participants show a mean age of 36.7 years (SD=13.4), with a predominance of women (66.7%, n=33) over men (33.3%, n=18). Regarding education, 42.6% of the participants had higher education (n=23), and 37% (n=20) had postgraduate degrees. The results of the cognitive tests were expressed as means and standard deviations, demonstrating the variation in the use of Cogniscan as a screening tool.

Conclusions: Initial results suggest that the Cogniscan test has potential as an effective tool for cognitive monitoring and early detection of declines. Validation of this software may significantly contribute to preventive and therapeutic interventions, promoting a better quality of life in the different stages of aging.

Keywords: Computerized Neuropsycholopgical Testing, Memory Complains, Neurophsychological Assessement.

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The efficiency of using clonidine buccally and dexmedetomidine intranasally as non-invasive procedural sedation during magnetic resonance imaging in patients hospitalized at the neonatology department in Ceske Budejovice

Backround: Procedural sedation does not have clear recommendations in the newborn population. There is an effort to find a balance between the need to minimize motion artifacts during imaging (including MRI) and the use of sedatives with the lowest possible adverse effects on the developing central nervous system.

Aims: The aim of the project was to evaluate the efficiency of using Clonidine and Dexmedetomidine as a non-invasive procedural sedation during MRI. Both drugs belong to the group of central antihypertensives. They stimulate receptors in the central nervous system which leads to decreasing of sympathetic tone without suppressing the respiratory center. There have been only a few studies regarging their use in newborns, specifically in the indication of procedural sedation, in which the administration of drugs required IV access. The primary goal was to successfully perform an imaging examination without the need to interrupt scanning or to use general anesthesia.

Methods: We performed an observational, retrospective study, where all data were obtained from patient records. During 4-year time interval (2020-2023), the effect of Clonidine and Dexmedetomidine was assessed in originally full-term or premature newborns. A total of 90 MRI examinations were performed. 26 examinations were excluded (the drug was not used, parallel antiepileptic therapy). The evaluation included 64 examinations, which were divided into two groups: The Clonidine group included a total of 22 examinations, the Dexmedetomidine group included a total of 42 examinations.

Results: Successful imaging was achieved in all cases. In the Clonidine group, 17 examinations (77%) were performed without the need for general anesthesia, in 5 cases it was necessary to add an additional sedative (23%). In the Dexmedetomidine group, 39 examinations were performed without the need for general anesthesia (92%), in only 3 cases (8%) an additional sedative was required. Overall, the success rate of performing MRI with Clonidine or Dexmedetomidine sedation was 87%.

Conclusion: It was possible to successfully perform MRI imaging without the need to interrupt the examination in 100% of cases, in 87% of cases the use of non-invasive sedation with Clonidine buccally or Dexmedetomidine intranasally was sufficient. The success rate was statistically nonsignificantly higher in the second Dexmedetomidine group, 77% to 92% (statistically non-significant, p 0.07). There was no occurrence of bradycardia or apneic episodes, in one case it was necessary to start oxygenotherapy due to unsatisfactory saturations (1.5%). Therefore both drugs are considered to be a safe possibility for sedation. Then on-invasive administration of the medication reduces the need to secure the child with IV access. There is also a possibility to limit the presence of an anesthesiologist during the examination. Due to a small number of patients in both groups, further studies are needed to achieve statistically significant results.

Biography

Dr. Sivakova studied at the Second Faculty of Medicine at the Charles University in Prague, Czech republic and graduated in 2017. Dr. Sivakova works as a neonatologist at the Neonatology Department in České Budějovice Hospital, which is a level 3 NICU.

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Prosodic changes in patients with idiopathic parkinson's disease: The effect of dysarthria severity

Background & Objective: Patients with Parkinson's disease frequently show hypokinetic dysarthria exhibiting both segmental and suprasegmental features of speech, such as monotonous pitch, reduced intensity, inappropriate pauses, slow or fast speech rate, and harsh voice. Prosody is suprasegmental aspects of speech that we use to communicate, emphasize, or revise the meaning of a speaker's message. The purpose of this study is to investigate prosodic characteristics in patients with Idiopathic Parkinson's Disease (IPD) during oral reading and elucidate changes in speech along with dysarthria severity.

Methods: We collected data from 105 patients with IPD aged from 43 to 82 for this study. The patients were divided into 4 groups of mild, moderate, severe, and profound according to dysarthria severity. Each participant was asked to read the whole passage "autumn" composed of nine sentences. For acoustic analysis, the first 4 declarative sentences of the passage were used (total 4 sentences, 134 syllables). Three acoustic dimensions-temporal, spectral, and intensity dimension-with 16 prosodic characteristics were analyzed using Praat software and the ProsodyPro (Xu, Y., 2013). Multivariate Analysis of Covariance (MANCOVA) was used for statistical analysis.

Results: Duration of utterance, pause, and overall pause in the temporal dimension, and jitter (%) in the spectral dimension significantly increased according to the severity of the groups. Also, mean intensity, and maximum intensity significantly decreased when severity increased. However, 4 groups did not show statistical differences in overall speech rate, articulatory speech rate, inappropriate pause, mean f0, minimum f0, maximum f0, f0 variance, minimum intensity, intensity variance, and shimmer.

Conclusions: This study demonstrated the different acoustic characteristics of connected speech according to their dysarthria severity in patients with IPD. The results may contribute to providing clues for exploring the speech biomarker in patients with IPD.

Biography

Dr. Miseon Kwon received her B.Sc. degree from Yonsei University in Korea and M.Sc. degrees from Miami University in the US. Also finished the College of National Rehabilitation Center in Japan and received Ph.D. from Ewha Womans University in Korea in 2004. Currently, Dr. Miseon is working as a research professor at the Department of Neurology, University of Ulsan College of Medicine, Asan Medical Center in Korea. Dr. Miseon Kwon is certified SLP in Korea, the U.S., and Japan and has published noteworthy international papers. The focus of Dr. Miseon's work is mainly on clinical research of neurogenic speech-language-swallowing disorders.

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Diagnostic challenges in Creutzfeldt-Jakob Disease: A case report of atypical presentation

Background and Aims: Creutzfeldt-Jakob Disease (CJD) is a rare, fatal neurodegenerative prion disorder often mimicking other neurological conditions including stroke. This case report highlights the diagnostic challenges in a patient with CJD initially presenting with non specific neurological symptoms.

Methods: A 70-year-old male with a history of bladder cancer, hypertension, osteoarthritis, and migraines presented with acute confusion, fever, and nonspecific symptoms. Initial evaluation revealed altered mental status, elevated inflammatory markers, and chest consolidation on CT imaging. Despite negative cultures and unremarkable initial lumbar puncture results, the patient's condition deteriorated rapidly. The patient exhibited acute confusion worsening over 2 days, billious vomiting, fever (39.1°C), generalized abdominal pain, persistent nausea, and decreased eating, drinking, and mobility. Initial differentials included stroke, hospital-acquired pneumonia, hepatic involvement, and urinary tract infection.

Results: Blood and urine cultures, CT head, and CTPA were inconclusive. MRI head revealed bilateral temporo-parietal cortical restricted diffusion, prompting neurological evaluation. The final diagnosis was confirmed by a positive RT-QuIC PCR test for CJD. Key diagnostic findings included clear colorless CSF with positive RT-QuIC PCR, elevated CRP (281) and WCC (15.2) with neutrophilia (14.1), and MRI showing bilateral temporo-parietal cortical restricted diffusion.

Conclusion: This case underscores the importance of considering CJD in patients presenting with atypical neurological symptoms, even when initial presentations suggest more common conditions like stroke. Early recognition, appropriate neuroimaging, and specialized tests like RT-QuIC are crucial for timely diagnosis and management of this rare but devastating illness.

Biography

Mohamed Shafei is a clinical fellow in medicine at King's Mill Hospital, gained extensive experience across various specialties, including stroke, respiratory, endocrine, and acute medicine. And, Mohamed Shafei's commitment to advancing medical expertise drives aspiration to continue his career in internal medicine. Mohamed Shafei have been actively involved in medical education and have made significant contributions to developing teaching programs for clinical fellows at the trust, with a strong interest in research, eager to explore new avenues in internal medicine that will allow me to improve patient outcomes and contribute to medical advancements.

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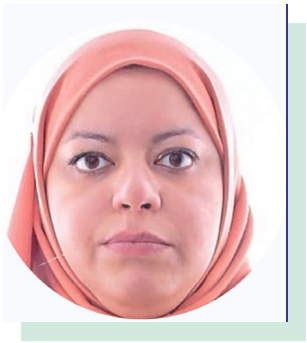
Brain aquaporin expression in development and disease

Aquaporins (AQPs) are channels that participate in water homeostasis in various organs including the kidney and brain. Several classes of these including AQP1, AQP4, and AQP9 are expressed, chiefly by glial cells, in the healthy human brain in a region-specific manner. Cerebral cortical expression of aquaporins has been most widely studied and has been found to be altered in specific diseases. We undertook a comprehensive study of aquaporin expression throughout the brain in postmortem human tissue affected by various age-associated diseases and found most conspicuous alteration in AQP1 expression in most of these. Comparison of these findings with expression throughout the lifespan as determined from studies of neonatal and pediatric brain autopsies showed that expression in adult disease recapitulates patterns found in the brain in infancy and early childhood. The results suggest that one component of astrocyte reaction to the presence of injury or disease may be activation of an earlier developmental phenotype with possible effects on water homeostasis and glymphatic function.

Biography

Dr. Randall Woltjer studied Medicine and Biochemistry at Vanderbilt University where he received his M.D. and Ph.D. degrees. He joined the faculty at the University of Washington and then at Oregon Health and Science University where he serves as Professor of Pathology and Director of the Neuropathology Core of the Alzheimer's Center and the Oregon Brain Bank. His studies focus on changes in the brain that develop in the context of disease and normal aging.



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Epidemiology of cerebral infarction in young adults: Study retrospective at the Annaba CHU, Algeria

Introduction: Cerebral Infarctions (CI) in young people represent a pathology whose incidence is increasing worldwide. The particularity of this young population lies in the diversity of risk factors, as well as in the etiologies incriminated. The main objective of our study was to describe the epidemiological, etiological and evolutionary aspects of cases of cerebral infarction in young adults at the Annaba University Hospital, Algeria.

Methods: Our study is retrospective and descriptive. It covers a period of four years from January 1, 2020 to December 31, 2023. It was carried out in the neurology department, including outpatient consultations and other services at the Annaba University Hospital, Algeria. The inclusion criteria were confirmation of cerebral ischemia by neuroimaging and an age of onset between 16 and 55 years. The etiologies of HF in our patients were determined according to the TOAST (Trial of Org 10172 in Acute Stroke Treatment) classification.

Results: The study involved 180 patients, averaging 42.7 years of age (±9.5) and a male-to-female ratio of 1.2. Hypertension was the primary risk factor, affecting 40.6% of participants, followed by patent foramen ovale in 18.9%. According to the TOAST classification, the most frequent causes were undetermined (41.1%), followed by embolic heart disease (26.7%). COVID-19 infection was the leading cause at 24.4%, followed by homozygous sickle cell disease at 19.5%. Atherosclerosis accounted for 7.8%, and small vessel occlusion for 1.7%. Sequelae occurred in 63% of patients, primarily spastic or non-spastic hemiparesis. The overall mortality rate was 8.3%. Our findings align with existing literature, with many cases having an undetermined origin, underscoring cryptogenic causes. A notable aspect of our study is its timing during the COVID-19 pandemic, which impacted the identified causes that vary significantly across studies.

Conclusion: Cerebral infarction in young individuals poses a diagnostic challenge. Our study emphasizes the need for comprehensive research into risk factors and thorough etiological assessments to develop effective therapeutic strategies for recurrence prevention.

Biography

Dr. Selma Hannachi studied neurology at the Faculty of Medicine in Annaba, Algeria, becoming a specialist in 2014 and became a maitre assistant in 2018 and will hold the position of master of conference B starting July 2024.

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Phosphorylation of Kv7.2 potassium channel's regulatory domain alters binding interactions with calmodulin

Kv7 channels are potassium ion channels expressed in the nervous system. Kv7 channels function to control the excitability of neurons by reducing the rate of action potential firing. The Kv7.2 channel has been shown to play a particularly crucial role in neurodevelopment. Five different *KCNQ* genes encode Kv7 subunits, which form a tetrameric Kv7 channel. Loss of function mutations in the *KCNQ2* gene have been shown to result in neurologic dysfunction, including neonatal epilepsy. Therefore, regulation of Kv7.2 is essential for normal neuronal function. Calmodulin (CaM) is a calcium (Ca^{2+})-binding protein shown to interact with the C-terminus of Kv7.2, particularly with its intracellular regulatory domain. This region contains several hotspots for pathological mutations, suggesting their importance in regulating the function of Kv7.2. The regulatory domain of KCNQ2 is composed of two regions of importance to this study: The A helix (Q2A) and B helix (Q2B). CaM binds to each Kv7 subunit by interacting with Q2A and Q2B. The amino acid sequence of Q2B contains phosphorylation sites at serines S520 and S527 which could potentially contribute to the regulation of Kv7.2 function. This study aims to characterize the Ca^{2+}-dependent binding interactions between CaM and the regulatory domain of Kv7.2. It is also investigating whether phosphorylation of Q2B interferes with CaM interactions and how CaM modifies its binding to the regulatory domain. Binding interactions were studied by electrophoretic mobility shift assay (EMSA) and Isothermal Calorimetry (ITC). Results from the ITC show that CaM has a higher affinity for Q2B (Kd=0.243μM) than Q2A (Kd=5.27μM). Compared to the binding affinity between CaM and an extended Q2B sequence (Kd=0.05μM), the binding affinity is 42x lower when Q2B is phosphorylated at S520 (Kd=2.1μM) and only 5x lower when phosphorylated at S527 (Kd=0.25μM). EMSA shows a degree of CaM binding to Q2B phosphorylated at S527 but demonstrates no binding when phosphorylated at S520. These findings indicate that Q2B phosphorylation at S520 blocks CaM interactions in the presence of Ca^{2+}.

Biography

Sinchana Basoor is a current second-year medical student at Long School of Medicine at UT Health San Antonio. Basoor completed undergraduate education at Baylor University in 2023, where a thesis on mitochondrial morphology in the pathophysiology of hearing loss. Currently, Basoor is involved in biochemical research with Dr. Crystal Archer at UT Health San Antonio. In addition, Basoor is conducting clinical research on orthopedic and plastic surgery outcomes in conjunction with University Hospital and the VA Hospital in San Antonio.

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