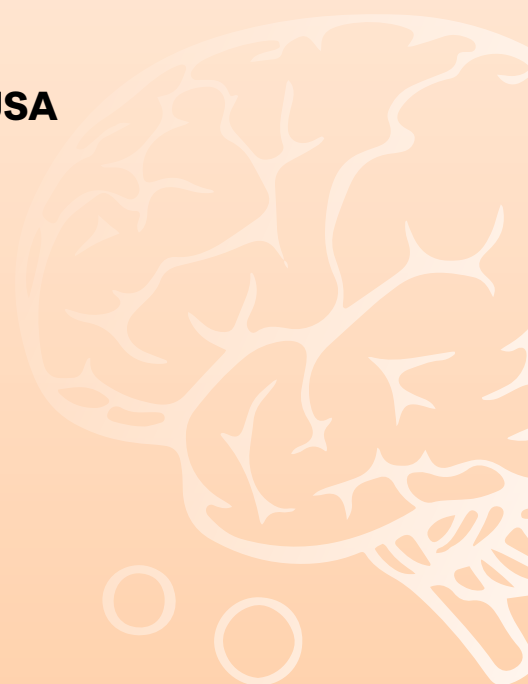




13TH EDITION OF
INTERNATIONAL CONFERENCE ON
**NEUROLOGY AND
BRAIN DISORDERS**

NeuroInnovations: Shaping the
Future of Brain Health

OCTOBER 19-21, 2026
BOSTON, MASSACHUSETTS, USA



13TH EDITION OF

International Conference on

NEUROLOGY AND BRAIN DISORDERS

HYBRID EVENT

19-21
OCTOBER 2026

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Keynote Speakers

Keynote Speakers



Bernd Blobel

University of Regensburg, Germany



Daniel Curry

Texas Children's Hospital, United States



George C Diaz

Memorial Healthcare Systems,
United States



Joe Sam Robinson

Mercer University, United States



Kazuhito Toyooka

Drexel University College of Medicine,
United States



Ken Ware

NeuroPhysics Therapy Institute and
Research Centre, Australia

Keynote Speakers



Robert B Slocum

University of Kentucky HealthCare,
United States



Sergei M Danilov

University of Illinois, United States



Sergey Suchkov

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



Steven Benvenisti

Davis, Saperstein Salomon, PC,
United States



Thomas J Webster

Brown University, United States



W S El Masri

Keele University, United Kingdom



Yong Xiao Wang

Albany Medical College, United States



Zhenhuan Liu

Guangzhou University Chinese Medicine,
China

Welcome Message



Bernd Blobel

University of Regensburg, Germany

Dear participants of the 13th Edition of the International Conference on Neurology and Brain Disorders, which will be held on October 19-21, 2026, in Boston, Massachusetts, USA. It is an honor and pleasure to write a few welcome notes. The central theme of INBC 2026 is NeuroInnovations: Shaping the Future of Brain Health. The healthcare paradigm advanced from empiric and phenomenological through evidence-based and person-centered medicine to personalized, preventive, predictive, participative precision medicine (P5M). Thereby, we must consider and understand the individual health status, conditions and genetic and genomic dispositions in personal social, occupational, environmental and behavioral context. This requires the inclusion of actors from multiple domains with their own methodologies, languages, ontologies, education and skills as well as the subject of care. In this context, communication and cooperation must advance from data level to the knowledge level. Therefore, the highly dynamic, complex, context-aware, multi-disciplinary transformed healthcare ecosystem must be represented as a system of systems, using a system-theoretical, ontology-based, policy-driven approach. I have developed a related model and framework, which has been standardized in ISO 23903 Interoperability and Integration Reference Architecture. Because of its formal and foundational nature, the methodology is not restricted to health and social care, but has been successfully deployed already in many other domains. Welcoming you to Boston, I'm sure that this conference will significantly contribute to your professional establishment and exchange, inspiring collaborative ideas and partnerships. So enjoy a successful event.

Welcome Message



Daniel Curry

Texas Children's Hospital, United States

It's a pleasure to welcome you to this year's conference. As a neurosurgeon, I'm particularly grateful for the opportunity to participate in a setting that values cross-disciplinary dialogue between neurology and neurosurgery. Many of the conditions we manage, epilepsy, hydrocephalus, movement disorders, don't respect the boundaries between our specialties. The best outcomes emerge when we integrate surgical and medical insights, data, and treatment strategies

We now face shared challenges and opportunities: translating physiologic data into actionable decisions, using neuromodulation to treat previously intractable disease, and improving how we monitor brain dynamics in real time. This meeting represents a chance to move those efforts forward together.

Thank you for your commitment to improving patient care through collaboration and innovation.

Welcome Message



Joe Sam Robinson

Georgia Neurosurgical Institute, United States

Dear Conference Attendees,

I have the honorable privilege of welcoming all the participants and guests to the 2026 meeting of the International Neurology and Brain Disorders Conference. Obviously, the world careens along on a difficult path. Happily, however, great progress is taking place in many medical issues. Importantly among them is the extension of life expectancy and, on a worldwide basis, the ability to monitor and control infectious disease outbreaks. Hopefully, another area of progress is the evolution and improved treatment of the vicious cycle that occurs between sleep disorders and cognitive decay. Preservation of higher intellectual capabilities would be the natural partner of increased life expectancy. At this meeting we will do our best to take fulsome steps in that direction, and I am quite pleased to be a part of this discussion.



Welcome Message

Ken Ware

Founder of Neurotricional Sciences Pty Ltd &
NeuroPhysics Therapy, Australia

It is a great honour and privilege to once again extend my warmest welcome to colleagues, researchers, clinicians, and delegates from around the world attending the International Neurology and Brain Disorders Conference 2026.

This conference provides an invaluable opportunity to bring together diverse perspectives from across the neurosciences, clinical medicine, rehabilitation, psychology, physics, and many other disciplines united by a common purpose - to advance our understanding of the human nervous system and improve the lives of those affected by neurological and brain disorders.

Many of the most significant advances in science have emerged not from working within the boundaries of a single discipline, but through the willingness to explore new ideas, challenge established assumptions and engage in open and respectful scientific dialogue. Conferences such as this provide the ideal environment for that process to flourish.

As we share our research, clinical experiences, and emerging discoveries over the coming days, I encourage each of us to remain curious, collaborative, and receptive to new perspectives. By learning from one another and embracing thoughtful innovation, we continue to build the foundations for future breakthroughs that may ultimately benefit patients and communities around the world.

I extend my sincere appreciation to the organising committee for bringing together such an outstanding scientific programme and to every presenter and participant whose contributions make this conference possible.

I wish you all a stimulating, rewarding, and memorable International Neurology and Brain Disorders Conference 2026.



Welcome Message

Robert B. Slocum

University of Kentucky HealthCare, United States

Dear conference participants and visitors, I am honored to offer these brief notes of welcome for you concerning my presentation.

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

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

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

Welcome Message



Sergey Suchkov

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

Dear Colleagues,

I look forward to welcoming you to 13th Edition of International Conference on Neurology and Brain Disorders, to be hold in Boston, MA, USA, offering a rich blend of modern, traditional, and ancient and valuable cultural heritage. As we delve into global neurology-related practices, Boston will remind us of the importance of adaptable and culturally sensitive strategies, exquisite cuisine and warm hospitality of the City, and its resilience and charm will undoubtedly enrich our discussions and collaborations.

This extraordinary gathering serves as an exceptional and unique platform that brings together neuroscience and clinical neurology experts, biodesigners and bioengineers, practitioners and medical insurers, healthcare administrators and students not only from the US but from around the world to share their latest research findings, and foster collaborations in the field of neuroscience and clinical neurology forward. The Conference will provide the ideal forum to stimulate ideas and establish partnerships as well as to initiate intense discussions.

Understanding the activity of a healthy and an altered brain is a vital focus of scientific research. Making progress in the field of **Personalized and Precision Neurology (PPN)**, the cornerstone of modern neuroscience and neurology, is thus one of the most significant global challenges of our time, with a plenty of questions remaining.

Advances in fundamental, translational and clinical research and the availability of highly reliable and validated biomarkers are beginning to transform the clinical neurology to make it personalized and precision, and healthcare landscape as a whole. Validated and highly informative biomarker platforms and targeting principles construe the work that goes into bringing the most promising experimental therapies, diagnostic and monitoring technologies to the PPN.

The core of the Event is its Scientific and Teaching Program. The Scientific and Organizing Committees are working tirelessly to create an engaging Program that includes keynote and oral speakers, eminent faculty members and opinion leaders, esteemed practitioners, biopharma and biotech experts from around the globe. Workshop sessions are aiming to open discussion between participants and lecturers, and immediate application of new knowledge. Beyond professional engagement, we are dedicated to making educational materials accessible to patients, their families, and the public, breaking down barriers to knowledge and understanding. The Conference underscores the significance of fostering collaboration on both regional, national and international levels - which would stimulate the exchange of professional expertise among participants to lead to transformative outcomes and advancements in the field of neuroscience and clinical PPN. Students will participate in faculty-led exercises such as debates.

We do hope that your interaction with the colleagues from many different countries will stimulate a creative exchange of ideas and will be personally rewarding. We firmly believe that this extraordinary learning opportunity will provide you with passionate perspectives that will expand your knowledge and inspire you to elevate the quality of care you provide to your patients and persons-at-risk.

Let's meet, share science together and enjoy the welcoming hospitality!



Welcome Message

Thomas J. Webster

Brown University, United States

Dear Colleagues and Friends,

Welcome to INBC 2026!

Without a doubt, recent advances in neurology and brain disorders are helping millions of patients. But, how did this occur? Was your research part of these advancements? Have we innovated and commercialized enough? Are companies not paying attention to this wonderful research? Are Universities not doing enough to help you? What about federal funding agencies? And, most importantly, are you in the right environment to innovate and commercialize your research?

In my own experience, above all else, it takes a supportive environment. Find the right community. I left the very negative, ultra-competitive Northeastern University in Boston (I have never seen such negative people in my 30 year academic career) over 5 years ago and I have never looked back. I found a truly supportive environment by forming my own companies and yes, finding supportive people who value me and my contributions at other Universities. Only then was I able to start numerous companies and commercialize my research into medical devices now in over 42,000 patients with no failures, only success!

So, I encourage everyone to find that right environment. Make that move. And attend the right conferences: the 13th Edition of the International Conference on Neurology and Brain Disorders 2026 (INBC 2026) is such a conference where you will meet the right people and be inspired!

It will change your life once you make the commitment to surround yourself with positive people. I know, because I lived it.

I look forward to welcoming you to my home town of Boston and sharing my story...which led to my Nobel Prize in Chemistry nomination!

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About Magnus Group

About

Magnus Group, a distinguished scientific event organizer, has been at the forefront of fostering knowledge exchange and collaboration since its inception in 2015. With a steadfast commitment to the ethos of Share, receive, grow, Magnus Group has successfully organized over 200 conferences spanning diverse fields, including Healthcare, Medical, Pharmaceuticals, 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 INBC 2026

About

The **13th International Conference on Neurology and Brain Disorders (INBC 2026)** will take place from **October 19-21, 2026**, at **Boston, Massachusetts, USA**, with an option for virtual participation. The conference will bring together neurologists, neuroscientists, clinicians, researchers, academics, healthcare professionals, and industry leaders from around the world.

Under the central theme **NeuroInnovations: Shaping the Future of Brain Health**, **INBC 2026** provides a global platform for sharing knowledge, presenting innovative research, and exploring emerging technologies shaping the future of clinical neurology and brain health.

The hybrid conference will feature keynote lectures, plenary sessions, oral and poster presentations, panel discussions, and networking opportunities, covering topics including Alzheimer's disease, Parkinson's disease, epilepsy, stroke, cognitive and memory disorders, pediatric neurology, neuro-oncology, neuroimmunology, neuroinflammation, neurocritical care, neurorehabilitation, neuropharmacology, neuroimaging, computational neuroscience, artificial intelligence, neurodegenerative diseases, novel drug development, and traumatic brain injury.

INBC 2026 encourages collaboration between academia and industry, highlighting clinical advancements, practical solutions, translational research, innovative diagnostics, therapies, and patient-centered approaches addressing global challenges in neurological and brain health.

About CPD Accreditation About

INBC 2026 is officially CPD-accredited, ensuring that professionals continue to enhance their knowledge, skills, and competencies through structured learning activities. At INBC 2026, participants can earn 3 CPD credit for every hour of attendance in conference sessions. These credits formally recognize the time spent in professional learning and support ongoing professional development across clinical, academic, research, and industry fields.

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

In addition to educational value, the conference provides opportunities to engage with neurologists, neuroscientists, clinicians, and peers from around the world, encouraging knowledge exchange and professional networking. Attending **INBC 2026**, in person in Boston or online, offers a high-quality scientific learning experience and CPD credits that reflect participants' dedication to continuous professional growth in neurology and patient care.

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NEUROLOGY AND BRAIN DISORDERS

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KEYNOTE PRESENTATIONS





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

Managing healthcare transformation towards personalized, preventive, predictive, participative precision medicine ecosystems

For realizing pervasive and ubiquitous health and social care services, health and social care system have to undergo an organizational, methodological and technological transformation towards personalized, participative, preventive, predictive precision medicine. For designing and managing the resulting highly complex, distributed and dynamic ecosystem, we must consistently and formally represent the system and its components from the perspective of all actors from different domains including the subject of care, using different methodologies, knowledge, language and experiences. The granularity level of the considered components may range from elementary particles up to the society and universe. This must be done, using a system-theoretical, architecture-centered, ontology-based and policy-driven approach. Over the last 30 years, the author developed the necessary model and framework, which is meanwhile standardized as ISO

23903 Interoperability and Integration Reference Architecture. The approach has been defined as mandatory for any specification or project at ISO, CEN, IEEE, etc. addressing more than one domain. The presented approach enables design, implementation and management of intelligent and ethical health and social care systems as well as knowledge-based communication and cooperation of all actors involved. Thereby, it manages also security, privacy and trust in detail. The Keynote introduces necessary standards and methodologies for designing and managing 5P medicine ecosystems as well as practical examples.



Daniel Curry, MD*, Rebecca White, FNP

Texas Children's Hospital/Baylor College of Medicine,
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Biography: Daniel J. Curry, MD is an academic pediatric neurosurgeon and director of the Pediatric Functional Neurosurgery Program at Texas Children's Hospital. He work spans surgical treatment of epilepsy, movement disorders, pain, and hydrocephalus. He lead the nation's largest pediatric SEEG and RNS program, pioneered MR-guided laser ablation for hypothalamic hamartomas, and launched the first intracranial gene therapy at our institution. He advancing real-time, ambulatory ICP monitoring and closed-loop shunt systems for hydrocephalus. His translational efforts also

include DBS in Rett syndrome models and focused ultrasound initiatives. He founded the TCH Neuroinfusion Center for enzyme therapy in Batten disease and NIH-directed intrathecal drug delivery research.

What we don't know about hydrocephalus and its management

Symptomatic hydrocephalus has been treated primarily with Cerebrospinal Fluid (CSF) diversion since the 1950s. Ventriculoperitoneal and related shunting procedures have been highly effective at relieving acute, life-threatening elevations in Intracranial Pressure (ICP). However, despite decades of technical refinement, modern shunt systems remain fundamentally passive devices, offering limited ability to optimize ICP across the wide physiologic spectrum encountered from infancy through adulthood. As a result, many patients live with chronic under- or over-drainage, intermittent symptoms, and recurrent hospitalizations despite functioning shunt systems as evidenced on imaging.

Several foundational questions in hydrocephalus management remain unanswered. These include the definition of optimal ICP targets across developmental stages, the degree to which ICP should be allowed to fluctuate in response to posture, activity, and sleep, and how environmental factors such as barometric pressure and altitude may influence symptom burden. Current clinical decision-making relies heavily on indirect assessments-symptom reports, neuroimaging, and episodic invasive measurements-rather than continuous physiologic data. This gap is particularly consequential for pediatric patients, individuals with communication limitations, and those with complex or atypical symptom profiles.

This abstract will review the current state of hydrocephalus management, including contemporary valve technologies, programmable and gravitational shunt systems, and existing invasive ICP monitoring approaches. While these tools have improved safety and customization, they remain limited by their inability to provide real-time, longitudinal

pressure data in the outpatient setting. As a result, clinicians are often forced to infer ICP dynamics indirectly, leading to delayed interventions and potentially avoidable morbidity.

We will also examine emerging concepts in neurobariatrics and pressure physiology, highlighting the dynamic and context-dependent nature of Intracranial Pressure regulation. Early evidence suggests that ICP is not a static variable but one influenced by development, behavior, and environment—factors that are not adequately captured by current treatment paradigms.

Finally, this discussion will identify critical technological, including the absence of ambulatory ICP monitoring, limited integration of physiologic data into clinical workflows, and the lack of evidence-based pressure targets tailored to individual patients. Addressing these gaps represents a necessary step toward shifting hydrocephalus care from crisis-driven management to proactive, data-informed treatment aimed at improving long-term daily neurologic health and quality of life.



George C Diaz

Memorial Neuroscience Institute, United States

Biography: Dr. George Diaz is a neurologist at Memorial Healthcare Systems in Hollywood, Florida. Dr. Diaz has promoted improved stroke care in South Florida since 1998. In addition to Vascular Neurology, Dr. Diaz is also board certified in Neurology, Brain Injury Medicine, and Neuromuscular Medicine. Dr. Diaz has lectured on multiple neurological issues, such as acute ischemic stroke, neurological emergencies, and neuromuscular diseases. Dr. Diaz earned his bachelor of science degree in chemistry at Florida International University and his medical degree at the University of Miami School

of Medicine. He interned and completed his neurology residency at the University of Miami–Jackson Memorial Hospitals. Dr. Diaz also completed a Neuromuscular Fellowship at the University of Texas Medical Center–MD Anderson and Herman Hospitals. Dr. Diaz and his partners at Memorial Healthcare are dedicated to the advanced treatment of patients with neurological diseases.

Atypical presentation of juvenile myoclonic epilepsy in a 16-year-old female: A case report

Background: Juvenile Myoclonic Epilepsy (JME) is a common idiopathic generalized epilepsy syndrome that typically manifests during adolescence, with onset most often between the ages of 12 and 18 years. It accounts for approximately 5–10% of all epilepsies and up to 26% of idiopathic generalized epilepsies. The condition is characterized by myoclonic jerks, usually occurring shortly after awakening, and may be associated with generalized tonic–clonic seizures and, in some cases, absence seizures. Despite the well-described electro clinical pattern, JME can be under recognized due to the subtlety of myoclonic jerks and the absence of overt convulsive episodes. Early recognition is critical because the disorder responds well to appropriate antiepileptic therapy, and untreated cases can progress to generalized tonic–clonic seizures or psychosocial impairment.

Case Presentation: We report a 16-year-old female with a two-year history of morning, sudden, brief episodes, which she calls glitches, accompanied by a transient sensation of her eyes rolling back and arms extending outwards. There is no myoclonic jerking, loss of consciousness, confusion, or other neurological deficits.

She denies any preceding aura, visual disturbances, or generalized convulsions. Her past medical history is unremarkable, with normal developmental milestones and no family history of epilepsy or neurologic disease.

Neurological examination was entirely normal, with no focal deficits, gait disturbance, or cognitive impairment. Routine laboratory studies, including metabolic and electrolyte panels, were within normal limits. A brain MRI was performed and was unremarkable. Electroencephalogram (EEG) revealed classic findings consistent with juvenile myoclonic epilepsy, characterized by generalized epileptiform consistent with the diagnosis of JME.

These findings, along with the clinical history, confirmed the diagnosis of JME.

Discussion: This case illustrates an atypical presentation of JME in which the patient's self-described glitches corresponded to subtle morning eye rolling back sensation with arms outstretched and without any generalized tonic-clonic or absence seizures. Such subtle presentations may be overlooked or misattributed to near syncope, benign tics, anxiety-related movements, or sleep deprivation. The absence of loss of consciousness and a normal neurological examination can further obscure recognition. Importantly, this case underscores the value of obtaining a detailed patient history. EEG remains the diagnostic cornerstone, as its characteristic generalized spike and polyspike-wave patterns can distinguish JME from other movement disorders or focal epilepsies.

Management of JME typically includes lifelong antiepileptic therapy, with valproate being the most effective agent. However, in adolescent females, alternative medications such as levetiracetam or lamotrigine are often preferred due to teratogenic and metabolic considerations. Early diagnosis and treatment can prevent progression and help patients maintain normal academic and social functioning.

Conclusion: Juvenile myoclonic epilepsy may present with subtle, self-described glitches rather than classical myoclonic jerks or generalized seizures. Clinicians should maintain a high index of suspicion when evaluating adolescents with brief, involuntary morning movements and normal neurological examinations. Prompt EEG evaluation and early treatment are crucial to achieving seizure control and preventing complications. Recognition of atypical presentations enhances diagnostic accuracy and ensures optimal long-term outcomes.

Keywords: Juvenile Myoclonic Epilepsy, Adolescent, Myoclonic Jerks, EEG, Case Report.



Joe Sam Robinson, Jr., M.D.^{1,2*}, Edward Robinson, M.D.³, Harold Groce, MIP^{1,4}

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Biography: Joe Sam Robinson is President of the Georgia Neurosurgical Institute and Chief of Neurosurgery at Atrium Navicent The Medical Center and Mercer University, all in Macon, Georgia. Robinson is an alumnus of Harvard University and the University of Virginia Medical School. Dr. Robinson received his neurosurgical training at Northwestern University with rotations at the National Institute of Health and Sloan-Kettering Hospital, New York City. Robinson has served on numerous government commissions, has widely published, and presented scientific papers in numerous academic locations.

The role of compromised cerebrospinal fluid circulation in sleep apnea

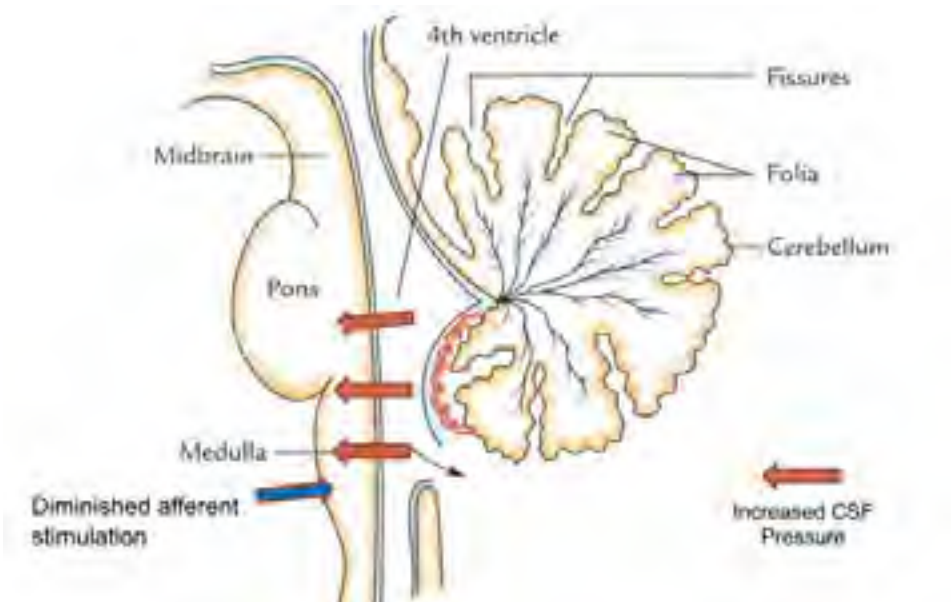
Since its first literary description by Charles Dickens in 1833, followed by its first scientific description in 1949, interest in sleep disturbances has skyrocketed (over 40,000 documented scientific papers have discussed the problem). Sleep apnea, however, remains an incompletely understood affliction. A complicated interconnected causal construct has been fashioned with different treatment modalities proposed and monitored. We suggest that an unappreciated factor may be episodic CSF disturbances often caused by cervical stenosis. On this point much evidence exists that, in an older population, osseous overgrowth may restrict the CSF pathways through which CSF normally flows from the cerebrum into the spinal canal. Such obstruction would logically result in episodic pressure disjunction.

Such pressure disturbances via the fourth ventricle would directly impact the pons and medulla, structures in which the complicated anatomical and neurophysiological arrangements responsible for respiratory regulation reside. CSF pressure alterations generally make their appearance in an aging population at exactly the time that neuronal population and fibrous tract integrity in the brainstem may have been selectively compromised, and, importantly, on the same timeline in which the symptoms of sleep apnea often appear (see graph 1).

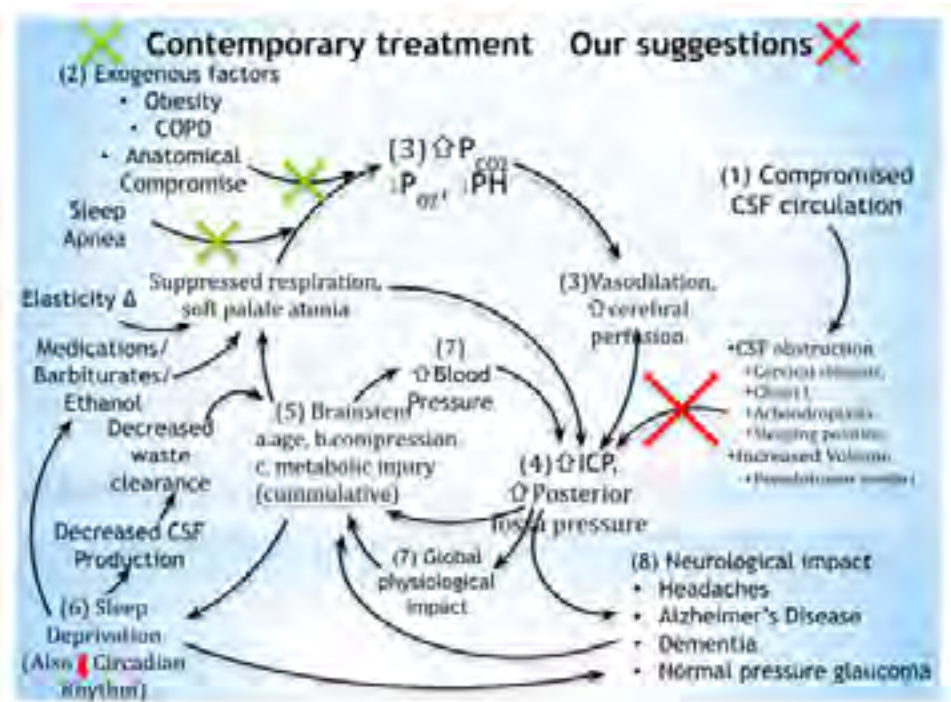
Logically, such CSF circulation disturbances, coupled with the naturally occurring aging process, would diminish robust brainstem function, causing a primary increase in sympathetic neuronal stimulation. Such an event then serves to provoke a vicious cycle, causing a number of physiological shortfalls.

Understanding the vagaries of CSF circulation (which in some ways might be likened to the physiological impact of blood pressure variation) and instituting appropriate therapeutic interventions might effectuate neurological improvement, interrupting the vicious cycle through which sleep apnea interferes with normal physiological balance. The treatment of cervical stenosis as a causal vector in sleep apnea could be conservatively effectuated by posture changes or, in more aggressive circumstances, surgical intervention (see graph 2).

Graph 1:



Graph 2:





Xiaonan Liu, Shiena Watanabe, Sierra Coleman, Vicky Shih, William R. Telfer, Vasu D. Kansagra, Lilit Drak, Laasya Reddy Pesaladinne, Diane Kim, Samridhi Sudan, Anushka Singhal, Kazuhito Toyooka*

Drexel University, United States

Biography: Kazuhito Toyooka, Ph.D., is an Associate Professor in the Department of Neurobiology and Anatomy at Drexel University College of Medicine. Toyooka's research examines the cellular and molecular mechanisms of cortical development and their disruption in autism spectrum disorder and other neurodevelopmental disorders. Toyooka studies ADNP and 14-3-3 proteins, including how 14-3-3 proteins regulate ADNP localization and how ADNP affects neurite formation, dendritic spine development, neuronal connectivity, and neuronal activity. Toyooka's laboratory uses mouse

models, primary neuronal cultures, *in utero* electroporation, CRISPR/Cas9, molecular biology, and functional analyses to determine how disease-associated gene dysfunction alters cortical circuit formation.

ADNP regulates cortical development: Mechanism of ADNP syndrome/autism

Activity-dependent neuroprotective protein (ADNP) is essential for cortical development. Heterozygous pathogenic ADNP variants cause Helsmoortel-Van der Aa syndrome (aka ADNP syndrome), a neurodevelopmental disorder that commonly includes intellectual disability, delayed speech and motor development, and autism spectrum disorder. ADNP acts in distinct cellular compartments. In the nucleus, full-length ADNP participates in chromatin remodeling and transcriptional regulation. In the cytoplasm, ADNP interacts with microtubule-regulatory proteins and helps regulate neurite initiation, axonal growth, dendritic development, and synapse formation. The mechanism coordinating the nuclear and cytoplasmic functions of ADNP during neuronal development remains unclear.

Our studies examine 14-3-3 proteins, particularly 14-3-3 ϵ , as regulators of ADNP nucleocytoplasmic shuttling. 14-3-3 proteins bind phosphorylated target proteins and can regulate protein localization, stability, and molecular interactions. We propose that binding to 14-3-3 promotes the redistribution of ADNP from the nucleus to the cytoplasm or retains ADNP in the cytoplasm, increasing the cytoplasmic pool of ADNP available for cytoskeletal regulation. Biochemical and cellular analyses indicate that ADNP associates with 14-3-3 in developing cortical neurons. Reduced ADNP alters neurite number, axonal growth, and dendritic development, indicating that both ADNP expression level and subcellular localization affect neuronal morphogenesis.

This presentation will integrate molecular, cellular, and developmental findings into a model in which 14-3-3-dependent nuclear-to-cytoplasmic shuttling coordinates ADNP-mediated transcriptional regulation with microtubule-dependent neuronal development. Pathogenic ADNP variants may disrupt 14-3-3 binding, alter ADNP localization, or reduce the cytoplasmic pool of ADNP. These changes could impair cortical development, including neurite formation, spine/synapse formation, and circuit formation, and contribute to the neurological features of ADNP syndrome. Determining how 14-3-3 regulates ADNP localization may identify specific molecular steps for therapeutic investigation.



Ken Ware*

Founder of Neurotricial Sciences Pty Ltd and NeuroPhysics Treatment (NPT), Hervey Bay, Queensland, Australia

Biography: Ken Ware is the founder of Neurotricial Sciences Pty Ltd and NeuroPhysics Therapy and Research and he had been in private practice for almost 30 years, while doing independent and collaborative research. He also presented unique research at more than 35 major International Science Conferences including neuroscience, Physics, Psychology and Life Sciences, which covers a very broad scientific audience. He had published relative publications in 'Frontiers in Clinical Physiology' - 'World Journal of Neuroscience' - 'World Journal of Cardiovascular diseases' - 'Journal

of Behavioural and Brain Sciences' - 'Complexity Science in Human Change'. He is Former Mr. Universe 1994, National powerlifting and Bodybuilding champion and record holder. He is recipient of Her Majesty, Queen Elizabeth's 'Australian Sports Medal' - in 2000, in recognition for personal contributions to the development of the Australian Sporting Culture.

Triple-network dysfunction, ME/CFS, and the NeuroPhysics treatment process: A dynamical systems perspective on psychophysical organization and environmental interaction

Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) and related post-viral conditions such as Long Covid are associated with complex disturbances involving cognition, autonomic regulation, fatigue perception, emotional processing, sensory integration, and motor function. Increasing evidence from network neuroscience suggests that dysfunction involving the Default Mode Network (DMN), Salience Network (SN), and Central Executive Network (CEN) may contribute to impaired adaptive regulation in these conditions. The dynamic interaction between these large-scale brain networks has become known as the Triple Network Model.

This paper presents a systems-neuroscience perspective of NeuroPhysics Treatment (NPT), a psychophysical treatment approach developed through decades of clinical observation involving posture, movement, environmental interaction, sensory-perceptual processing, and autonomic regulation in individuals with complex neurological and psychophysical disorders. Longstanding clinical observations within a NPT environment suggested that many individuals with ME/CFS were not simply physically deconditioned but appeared to exist within maladaptive psychophysical states characterized by excessive self-referential monitoring, altered effort perception, sensory over-association, autonomic hypervigilance, and compromised environmental engagement.

Contemporary triple-network neuroscience provides a framework through which these observations may be better understood. Within this model, excessive DMN dominance relating to symptom monitoring and predictive concern, combined with maladaptive salience attribution and reduced executive adaptability, may contribute to unstable psychophysical organization and impaired environmental interaction. The Anterior Cingulate Cortex/Gyrus (ACC/G), strongly associated with salience regulation, effort appraisal, conflict monitoring, autonomic integration, and behavioral adaptation, may play a particularly important integrative role within these processes.

NPT is discussed as a process that through its purposeful prescription, design and implementation helps enhance network flexibility and psychophysical organization through stable, low-noise, repeatable sensory-motor environments involving controlled movement, postural organization, attentional refinement, reduction of unnecessary muscular tension, and gradual exposure to predictable environmental demands. From this perspective, NPT may assist the individual in progressively reducing maladaptive over-associations between sensory input, threat appraisal, autonomic activation, and defensive behavioral prediction.

The paper further proposes that the interaction between the DMN, SN, and CEN may exhibit properties conceptually analogous to nonlinear dynamical systems, including sensitivity to initial conditions, unstable transitional states, and difficulties maintaining stable long-term synchronization under perturbation. In this regard, the Triple Network Model may bear conceptual similarities to the classical three-body problem in physics, where interacting systems continuously influence one another in highly complex and often unpredictable ways.

This framework is not presented as a definitive mechanistic explanation for ME/CFS, but rather as a systems-level perspective intended to stimulate further discussion regarding the relationship between large-scale brain network dynamics, psychophysical organization, environmental interaction, and adaptive behavioral regulation in complex chronic conditions.

Contextual Note: Since 2014, NeuroPhysics Treatment (NPT) has been utilized in the management of more than 100 individuals who travelled internationally seeking assistance for ME/CFS, Long Covid, and related complex chronic conditions. Many of these cases involved longstanding and highly debilitating symptom presentations and have been documented through international and national media coverage, social media platforms, Vimeo, YouTube, and peer-reviewed scientific publications.

Keywords: NeuroPhysics Treatment (NPT), Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS), Triple Network Model, Default Mode Network (DMN), Salience Network (SN), Central Executive Network (CEN), Dynamical Systems Theory, Three-Body Problem Analogy.



Robert B. Slocum, Ph.D.

Narrative Medicine Program Coordinator, University of Kentucky HealthCare, Lexington, Kentucky, United States

Biography: Robert B. Slocum is the Narrative Medicine Program Coordinator at University of Kentucky HealthCare. Slocum holds doctorates in law (Vanderbilt), ministry (University of the South), and theology (Marquette). Slocum has experience in pastoral ministry as well as academic teaching and administration. Slocum has taught undergraduate courses in religious studies and ethics. Slocum is an Assistant Professor (voluntary faculty, Internal Medicine) at the University of Kentucky College of Medicine (COM). Slocum teaches a fourth-year COM elective on the narrative basis for patient care and

resilient practice. Slocum is a member of the Hospital Ethics Committee. Slocum is the author, editor, or co-editor of 14 books, including a journal of reflections. Slocum's 36 articles have appeared in theological or medical journals and as book chapters, and Slocum has made presentations at more than two dozen theological and medical conferences. Slocum has also published short fiction and poetry. Slocum is interested in the clinical application of narrative and the significance of narrative for identity formation. Slocum sees Narrative Medicine as a bridge between medical humanities and clinical practice.

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

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

Patients with FS are frequently misdiagnosed and mistreated for epileptic seizures. Accurate diagnosis may be delayed for many years. FS may cause severe disruption of the patient's quality of life in terms of employment or schooling as well as relationships, and activities of daily living. Some patients with FS have been accused of faking symptoms or malingering, and stigmatized by health care providers, coworkers, family members, and others in society. Patients with FS may have family histories of poor interpersonal communication and conflict resolution, with inherited codes of silence and shame concerning sensitive or traumatic subjects. Patients with FS may have Post-Traumatic Stress Disorder (PTSD) as a comorbidity. They may have significant dissociation and emotional blindness (alexithymia) with inadequate emotional awareness and expression.

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

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

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

This presentation will help the audience to understand the causes and symptoms of Functional Seizures (FS), and respond appropriately when encountering patients with PNES.

This presentation will help the audience to understand the need for Narrative Medicine (NM) interventions for patients with FS, and to make appropriate referrals for care with NM



Sergei M. Danilov, MD, PhD

Department of Medicine, University of Illinois at Chicago, IL
60612, United States

Biography: Sergei M. Danilov, MD completed his PhD and postdoctoral studies from the National Cardiology Research Center, Moscow, Russia. Danilov is the Principal Investigator and Head of the laboratory of ACE biology in the Division of Pulmonary and Critical Care, Department of Medicine, University of Illinois at Chicago. Danilov's laboratory developed more than 40 mAbs to ACE. Danilov has published more than 150 papers on ACE biology and ACE immunochemistry in highly respected journals and has been serving as an editorial board member of *Biomedicines*.

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).

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 >300 carriers of 50+ different ACE mutations and 200+ controls. 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 A β 42, 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.

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) testing of lead candidates on mouse transgenic mice-model of human ACE mutations; 3) 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 proteasome inhibitors.



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¹¹Clinical Chemistry Lab, SFGH, San Francisco, CA, United States

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¹³Multiple Sclerosis and Autoimmune Neurology Mayo Clinic, Rochester, MN, United States

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, Suchkov was a senior Researcher, Koltzov Inst of Developmental Biology. From 1989 through 1995, Suchkov 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). 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. Suchkov was an Exe Secretary-in-Chief of the Editorial Board, Biomedical Science, an international journal published jointly by the USSR Academy of Sciences and the Royal Society of Chemistry, UK. At present, Suchkov is Professor in Medicine & Immunology and Director for Center for Biodesign of N.D. Zelinskii Institute for Organic Chemistry of the Russian Academy of Sciences, Moscow, Russia; R&D Director, InMedStar, Russia-UAE; Senior Scientific Advisor of China Hong Kong Innovation International Business Association, Hong Kong; Member, New York Academy of Sciences, USA; Member, EPMA (European Association for Predictive, Preventive and Personalized Medicine), Brussels, EU; Member, ISPM (International Society for Personalized Medicine), Japan; Member, PMC (Personalized Medicine Coalition), Washington, USA; Member, AMEE (Association for Medical Education in Europe), Centre for Medical Education, Dundee, Scotland; Member, ACS (American Chemical Society), Washington, DC, USA; Member, AHA (American Heart Association), Dallas, TX, USA; Member, ARVO (The Association in Research in Vision & Ophthalmology), Rockville, MD, USA; Member, ISER (International Society for Eye Research), Anchorage, AK, USA; and Secretary General, United Cultural Convention (UCC), Cambridge, UK.

Personalized and Precision Medicine (PPM) as a unique healthcare model to secure human healthcare and biosafety through the view of clinical neurology practice to secure human healthcare and biosafety

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

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

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

The future of PPM-guided neurology lies in multimodal digital data, enabling the principles of PPM to be applied in neurological disease diagnostics, treatment, and monitoring at scale, expanding the benefits to everyone. This approach offers a highly accessible, cost-efficient,

and non-invasive approach for diagnosing neurological diseases at their clinical and subclinical stages, placing an individual precisely along a disease continuum, and providing the most effective possible canonical and preventive treatment pathways.

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

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

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



Steven Benvenisti

Davis, Saperstein Salomon, United States

Biography: Steven is a Partner at one of the largest personal injury law firms in the United States and is included on the list of the National Trial Lawyers Top 100 Trial Lawyers. He is President of the Brain Injury Alliance and is a Director of Mothers Against Drunk Driving. Steven has received over 30 awards, including a US Congressional Citation, US House of Representatives Certificate of Special Congressional Recognition and Citizen of the Year. Steven authored *Spring Break: A True Story of Hope and Determination* about a famous case he handled involving a severely brain injured college student. *Spring*

Break credits the doctors, nurses and rehabilitation professionals for the wonderful work that they do in improving the lives of patients and their families. He has been a keynote speaker at over 100 conferences and gladly donates 100% of his honoraria to charity.

Spring break: A true story of hope and determination

Traumatic brain injury is the silent epidemic of our time. In spite of over 1.7 million people sustaining a T.B.I. every year in our country, only a very small minority of medical professionals, legal scholars, employers, educators and people understand the reality of what the survivor and family members are going through. The most effective and efficient way to get an understanding of traumatic brain injury and how to deal with it is directly from the perspective of a survivor.

This program begins by presenting a powerful story to the audience about an All American college student on Spring Break with his fraternity brothers. Midway through his vacation, he is walking and struck by a drunk driver's vehicle. His parents are called in the middle of the night and asked to consent to organ donation due to their son's severe traumatic brain injury and other catastrophic injuries. The family declines to consent to organ donation and instead gets a room in the hospital to be with their son 24/7.

After seeing the powerful photographs of the student in a coma, the audience are then delighted to next learn that he awoke after almost two (2) weeks, survived and had a full recovery. They are then astounded by the announcement that the speaker before them is actually the T.B.I. survivor who is the feature of the program.

The remainder of the program educates the audience about T.B.I. from the perspective of the survivor and family, while also providing them with needed tools to help them in their own professional and personal capacities in their dealings with Traumatic Brain Injury.



Thomas J. Webster

School of Health Sciences and Biomedical Engineering,
Hebei University of Technology, Tianjin, China

Division of Pre-College and Undergraduate Studies, Brown
University, Providence, RI, United States

School of Engineering, Saveetha University, Chennai, India

Biography: Thomas J. Webster's (H index: 135) degrees are in chemical engineering from the University of Pittsburgh (B.S., 1995; USA) and in biomedical engineering from RPI (Ph.D., 2000; USA). Webster has formed over a dozen companies who have numerous FDA approved medical products currently improving human health in over 30,000 patients. Webster currently serves as CSO of Novaurum whom is pioneering the use of ALD in medical devices. Webster is also currently serving as a professor at Brown University, Saveetha University, Hebei University of Technology, UFPI, and others. Webster

has numerous awards including: 2020, World Top 2% Scientist by Citations (PLOS); 2020, SCOPUS Highly Cited Research (Top 1% Materials Science and Mixed Fields); 2021, Clarivate Top 0.1% Most Influential Researchers (Pharmacology and Toxicology); 2022, Best Materials Science Scientist by Citations (Research.com); and is a fellow of over 8 societies. Webster is a former President of the U.S. Society for Biomaterials and has over 1,350 publications to his credit with over 55,000 citations. Webster was recently nominated for the Nobel Prize in Chemistry. Webster also recently formed a fund to support Nigerian student research opportunities in the U.S.

Nanoparticles passing the blood brain barrier to treat cancer, infection, and more

Nanotechnology has revolutionized numerous industries from space exploration to medicine. This talk will emphasize how nanoparticles can be synthesized by traditional precipitation techniques or by cells to pass through the blood brain barrier to treat brain cancer, brain infection, stroke, and more. *In vitro* and *in vivo* studies will be presented which show that the size, charge, chemistry, and crystallinity of nanoparticles can be controlled to penetrate the blood brain barrier and without drugs kill unwanted cells and promote the growth of other healthy cells. Specifically, one animal study will be presented in which stem cells with carbon nanoparticles were injected into stroke induced rat brains and were able to fully return motor and reflex functions in as short as 7 days. In this manner, this presentation will provide a comprehensive view of how nanoparticles can be used for a wide range of neurological disorders.



W S El Masri, FRCS Ed, FRCP, PHF

Keele University, United Kingdom

Robert Jones & Agnes Hunt Orthopaedic Hospital, Oswestry, United Kingdom

Biography: Prof W S El Masri FRCS Ed, FRCP currently Hon. Clinical Professor of Spinal Injuries (SI), Keele University has trained between 1971 & 1983 in the Oxford group of hospitals, Guys & Stoke Mandeville hospitals and the USA. He obtained the first accreditation in Spinal Injuries and General Surgery in 1982. Appointed Consultant Surgeon in Spinal Injuries at the Midland Centre for Spinal Injuries in 1983. He personally treated 10,000 patients with. He published 145 manuscripts. He the author of the: Concepts of Physiological Instability of the Spinal Cord, Time related Biomechanical Instability, Micro-instability of the injured spine and published the largest series

of Bladder cancer in SCI patients. He has repeatedly demonstrated and published on the discrepancy between the radiological and neurological presentation of patients in support of the hypothesis that the initial force of the impact and the quality of the management of both the injured spine and the effects of cord injury are the two major determinants of the initial neurological loss and the neurological outcome. He is Past-President of the International Spinal Cord Society; Past Chairman British Association of Spinal Cord Injury Specialists and has lectured world-wide. He won many National and International awards.

Acute traumatic spinal cord injuries - relevance of the model of service delivery and methods of management to outcomes?

Traumatic Spinal Cord Injuries (TSCI) are life-changing events from their medical, physical, psychological, social, financial, vocational, environmental & matrimonial effects. Although the clinical symptoms and signs of the condition was documented in the Edwin Smith papyrus 5000 years ago since which times many attempts have been made to treat the condition by attention given to the injured spine the majority of the patients died from major complications within a year from injury.

During the 2nd World War L. Guttman, a well-trained and experienced Neurosurgeon, directed his attention to the impact of the impaired physiology caused by the neurological injury, the consequences of the impaired physiology on the functioning of the various systems of the body, the complications that caused death and the methods of their prevention. He also gave a great deal of attention to the non-medical effects of spinal cord and cauda equina damage.

Guttman was disappointed with the outcomes of Laminectomy. He developed instead a model of system care to patients that deals with the consequences of generalised physiological impairment, multi-system malfunction, multiple disabilities, wide range of potential complications, sensory impairment/loss as well as the non-medical effects of

paralysis that impose challenges to patients, carers and clinicians. Within only four years from the development of such a service at Stoke Mandeville Hospital (SMH) Guttman demonstrated that patients not only survived but the majority could lead healthy, dignified, productive and often competitive lives. Moreover some patients exhibited various degrees of motor improvement. This system of care has been described by El Masri (Guttman's and Frankel's student) as Active Physiological Management (APCM) of all aspects of the effects of Spinal Cord and Cauda Equina Injury (SCCEI).

In the mid nineteen sixties Frankel and his colleagues at studied the Spontaneous Neurological Recovery (SNR) in 612 patients admitted within 15 days of injury and treated with APCM. He observed that 60% of patients with long sensory tract sparing and over 75% of patients with long tract sensory & poor motor sparing underwent significant motor recovery to move their legs and many could stand and walk. He published these predictable outcomes in a neuro-functional classification in 1969 since which time these outcomes were confirmed by many other groups and until the early 1980s APCM has proved to be the traditional method of management of TSCCEI.

The better visualisation of the injury by CT & MRI, improvement of spinal instrumentation and safety of anaesthesia since the 1980s encouraged the promotion of spinal surgery based on the assumption that surgical decompression of the neural tissue within a window of opportunity of 8, 12 or 24 hours of injury is likely to halt many of the detrimental 2ry cellular and cell membrane disturbances; vascular, chemical, metabolic, inflammatory and enzymatic changes caused by the injury and improve the neurological outcome.

Equally surgical reduction and stabilisation became strongly advocated on the assumption that this would enable to safely mobilise, rehabilitate and discharge the patient within a short period of hospitalisation. Unfortunately the effects of the disruption of the blood brain barrier and the loss of auto-regulatory functions of the injured neural tissue caused by the injury seem to have been ignored while advocating these two methods of management.

To date (40 years since the change of practice in the management of the injured spine of patients with neurological damage) none of these assumptions have been justified by evidence of equality or superiority of outcomes compared with the outcomes of APCM.

Professor El Masri will discuss in some depth the relevance of the model of service delivery to patients and the level of evidence of those assumptions that led to change of practice in the management of these patients.



Yong-Xiao Wang

Department of Molecular and Cellular Physiology, Albany Medical College, Albany, New York, United States

Biography: Dr. Yong-Xiao Wang has been a Full Professor in Department of Molecular and Cellular Physiology at Albany Medical College since 2006. Wang obtained an MD at Wannan Medical University, a PhD at Fourth Military Medical University, and postdoctoral training at Technology University of Munich and University of Pennsylvania. Wang 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.

Promising novel therapeutic strategies for vascular dementia

Vascular Dementia (VD) has high morbidity and mortality. Diabetes is a leading factor for VD. The cellular responses and signaling mechanisms for diabetes-induced VD are largely unknown, and the current treatments for VD are neither very specific nor effective. Dysfunctions of Cerebral Arteries (CAs) to cause blood hypoperfusion to the brain makes an important contribution in VD. 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.

We have started to explore whether and which ion channels might be essential for diabetes-evoked VD. Consistent with the previous reports, we have found that intraperitoneal injection of streptozotocin caused a large increase in blood glucose, leading to diabetes in mice. The diabetic mice had declined cognition, impaired memory, and increased anxiety, thereby exhibiting significant VD. This 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 ($[Ca^{2+}]_i$) in CAsMCs. Increased $[Ca^{2+}]_i$ was attributed to the augmented Ca^{2+} release from the SR, the major intracellular Ca^{2+} 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). Excitingly, we have further found that exposing VSMCs and isolated mitochondria to increased free calcium concentrations resulted in a proportional increase in ROS generation. Equally importantly, ROS generation in isolated cells, mitochondria and mitochondrial complex III from CSMCs were all increased in diabetic mice. DNA damage and Tau phosphorylation in CSMCs were largely increased as well.

In conclusion, our findings provide the first evidence that diabetes causes RyR2/FKBP12.6 dissociation, increases RyR2 activity and calcium release, mitochondrial calcium increase and ROS production, DNA damage, and Chk2 phosphorylation in CSMC, thereby leading to Vascular Dementia; presumably, our results further demonstrate that specific pharmacological and genetic RyR2 inhibition, FKBP12.6 stabilizer, mitochondrial ROS inhibitors and removers in vascular SMCs may become specific and effective treatments for diabetic VD and vascular complications.



Zhenhuan Liu

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

Biography: Zhenhuan Liu is a professor of pediatrics, a pediatric acupuncturist, and a Ph.D. tutor. Liu has been engaged in pediatric clinical and child rehabilitation for 40 years, and 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 26,800 children's deformities returned to school and society and became self-sufficient. The rehabilitation effect ranks the international advanced level. Liu is Vice-Chairman of the

Rehabilitation Professional Committee for Children with Cerebral Palsy, World Federation of Chinese Medicine Societies, and has been a Visiting Professor at the Chinese University of Hong Kong for the past 10 years. Liu is the most famous pediatric neurological and rehabilitation specialist in integrated traditional Chinese and Western medicine in China. Liu has edited 20 books and published 300 papers in international and Chinese medical journals.

Transcranial painless neurorehabilitation scalp acupuncture electrical stimulation for neuroregulation of autism spectrum disorder

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

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

Methods: 198 children with ASD were randomly divided into two groups. One group 89 cases received painless scalp electroacupuncture therapy and the other group 89 cases received Pediatrics rehabilitation care and special education for 6 months. Clinical evaluation methods were ATEC, ABC, CARS and Gesell developmental scales. Pre- and post-treatment assessments were performed. The age of the two groups was 3-7 years old, and the gender, degree of illness, comorbidities, family education and rearing methods, course of disease and other factors were statistically analyzed. There was no significant difference between the two groups, and there was a certain comparability between the two groups. Painless scalp electroacupuncture therapy method, acupuncture and precise scalp surface projection in functional language area of cerebral cortex were selected for scalp acupuncture. Broca and Wernicke area were simultaneously stimulated by acupuncture. Painless scalp acupuncture is performed every other day. After painless scalp acupuncture, electrical acupuncture was given to stimulate the language area for 15 minutes, every 10 times of acupuncture, rest for 15 days. A second clinical evaluation was conducted 6 months after painless scalp acupuncture.

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

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

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

Keywords: Autism Spectrum Disorder, Electroacupuncture, Scalp, Painless.

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ORAL PRESENTATIONS





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Activation of longevity protein SIRT3 as a therapeutic strategy for Alzheimer's disease

Approximately 6.9 million Americans are living with Alzheimer's Disease (AD), and by 2050, this number is projected to reach nearly 13 million. A significant number of people with AD have coexisting pathologies caused by diseases including, diabetes, hypertension, and cardiovascular disease. The precondition for these comorbidities is Metabolic Syndrome (MetS), which is caused by the deficiency of Sirt3, a mitochondrial deacetylase. We have shown that Sirt3 gene deletion in an AD mouse leads to exacerbation of β amyloid plaque deposition, and neuroinflammation. Our hypothesis is that SIRT3 is a potential therapeutic target for the treatment of AD, especially with comorbidities. In the current study, we evaluated the efficacy of Sirt3 activator honokiol and its analogs (DCA & Hexa) in microglial cells and mouse brain. Honokiol hexafluoro (Hexa) showed maximum increase in Sirt3 levels and decrease in lysine acetylation of mitochondrial proteins, a marker for their activation, and improved mitochondrial respiration in mouse microglial cells. Next, feeding of Hexa in C57/BL6 mice showed significant increase in the SIRT3 protein levels as compared to control mice. Interestingly, BACE1, an enzyme that generates A β from APP decreased significantly, whereas mitochondrial fusion proteins (Mfn1 & Mfn2) and transcription factor TFAM increased in Hexa-treated mice. Importantly, Hexa treated Sirt3^{-/-} mice showed no significant changes in BACE1, Mfn1, Mfn2, and TFAM expression, suggesting that Hexa effects are mediated by SIRT3. Finally, Hexa decreased pan acetylation in isolated primary microglia of wild type mice but not in Sirt3^{-/-} mice. We conclude that Hexa actions are through induction of Sirt3 expression. Together, our findings demonstrate the strong potential of Hexa as a novel therapeutic agent against AD.

Biography

Alpna Tyagi is an Assistant Research Professor of Medicine at the University of Colorado Anschutz Medical Campus, supported by a VA Merit award. Tyagi earned a PhD from the All-India Institute of Medical Sciences in New Delhi, India and completed postdoctoral training at Vanderbilt University and University of Colorado Anschutz Medical Campus. Tyagi's research focuses on signalling pathways involved in mitochondrial dysfunction, metabolism, and neuroinflammation in the brain, with the goal of developing metabolic and anti-inflammatory therapeutic strategies for Alzheimer's disease.



Amgad Elwy Ali Hussein*,

Asmaa Osama Sayed

Faculty of Physical Therapy, Cairo University, Egypt

Immediate effect of high-frequency transcutaneous electrical nerve stimulation on muscle tone and range of motion in children with spastic diplegic cerebral palsy: A randomized controlled trial

Background: Spasticity is a major motor impairment in children with Cerebral Palsy (CP), limiting functional mobility and joint Range of Motion. Transcutaneous Electrical Nerve Stimulation (TENS) has been suggested as a non-invasive modality to modulate muscle tone through sensory input and spinal inhibitory mechanisms. However, evidence regarding its immediate clinical effects remains limited.

Objective: This study aimed to investigate the immediate effect of high-frequency TENS on muscle tone and Range of Motion in children with spastic diplegic Cerebral Palsy.

Methods: A randomized controlled trial was conducted on 60 children diagnosed with spastic diplegic Cerebral Palsy, aged 3–10 years. Participants were randomly assigned into two equal groups ($n=30$ each): a study group receiving active TENS and a control group receiving sham stimulation. Outcome measures included muscle tone assessed using the Modified Ashworth Scale (MAS), Range of Motion (ROM) measured by goniometry, and neurophysiological assessment using Electromyography (EMG) and H-reflex (H/M ratio). All measurements were taken before and immediately after the intervention.

Results: The study group demonstrated a statistically significant reduction in spasticity levels compared to the control group ($p<0.01$). Additionally, there was a significant increase in Range of Motion in the study group compared to controls ($p<0.001$). No significant differences were observed in the H/M ratio between groups ($p>0.05$). Within-group analysis revealed

significant improvements in ROM and MAS in the study group, while no significant changes were detected in the control group.

Conclusion: High-frequency TENS appears to be an effective, safe, and non-invasive modality for immediate reduction of spasticity and improvement of joint Range of Motion in children with spastic diplegic Cerebral Palsy. Further research is recommended to investigate long-term effects and optimal dosing parameters in comparison with other spasticity management strategies.

Keywords: Cerebral Palsy, TENS, Spasticity, Pediatric Neurorehabilitation, Randomized Controlled Trial.

Biography

Amgad Elwy Ali Hussein is a physiotherapist specialized in pediatric neurorehabilitation. Hussein holds a Bachelor's and Master's degree in Physical Therapy from Cairo University and current PhD candidate. Hussein's research focuses on neuromodulation techniques, particularly transcutaneous electrical nerve stimulation (TENS), in children with cerebral palsy. Hussein has conducted both pilot and randomized controlled trials investigating the effects of TENS on spasticity and motor function. Hussein's clinical and research interests include neuroplasticity, motor control, and evidence-based pediatric rehabilitation.



Andrew Shin

The Harker School - Upper School Campus, United States

Early network-level signatures of Parkinson's disease in human midbrain organoids

Human midbrain organoids are 3-dimension neural tissues derived from iPSC-stem cells. As they self-assemble into layered neural networks, these models capture key aspects of developing human dopaminergic circuitry in a controlled in-vitro setting, enabling mechanistic studies that are difficult in real patients. One important application is Parkinson's disease, a neurodegenerative disorder marked by dysfunction and eventual loss of dopaminergic neurons. Specifically, by modeling these processes in a human-derived system, midbrain organoids offer a powerful platform to detect early electrophysiological abnormalities and to study how disease-like perturbations reshape neuronal networks. Here, we analyzed midbrain organoid multielectrode-array recordings (MaxTwo; Sharf Lab) from healthy controls and 6-hydroxydopamine (6-OHDA)-treated organoids. Spiking activity was isolated using KiloSort2 and quality filtered to ensure robust neuronal signals. Spike trains were then converted into functional connectivity networks, enabling comparison across complementary axes: network complexity, topology, and directed information flow. Across analyses, PD-induced organoids exhibited reduced coordinated network computation. Higher-order interaction metrics revealed a shift toward greater redundancy and reduced synergistic signaling relative to controls ($p < 0.0001$). Network topology was weakened, consistent with disrupted hub-like structure and reduced balance between integration and segregation, while directed communication measures suggested less efficient signal propagation. To assess early detectability, an autoencoder trained on control recordings learned baseline dynamics and flagged treated samples as deviations within short windows (~120s, 94% AUC). Together, these results indicate that PD-like perturbations produce quantifiable network-level dysfunction detectable from organoid electrophysiology, supporting brain organoids as a scientific resource for further biomarker discovery and research.

Biography

Andrew Shin is a high school junior at The Harker School in San Jose, CA with a strong passion for neuroscience and computational biology. He is a research intern at the University of California, Santa Cruz, where he studies Parkinson's disease using human midbrain organoids and computational analysis of neural connectivity. His work focuses on applying machine learning and mathematics to understand complex biological systems. He has received recognition at the Synopsys Science and Technology Championship and qualified for the California Science & Engineering Fair. He hopes to pursue a future in neuroscience, using computational tools to advance healthcare and patient outcomes.



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Health-related behaviors and autism spectrum disorder among U.S. children aged 3–5 years: National Survey of Children's Health (NSCH) 2023–2024

Background: Health-related behaviors, including diet, outdoor activity, screen time, and sleep, are important determinants of child health and development, yet little is known about the associations between these behaviors and Autism Spectrum Disorder (ASD) among young children.

Objective: To examine associations between health-related behaviors and ASD among U.S. children aged 3–5 years.

Methods: Data were obtained from the 2023–2024 National Survey of Children's Health (NSCH). Health-related behaviors included fruit and vegetable consumption during the past week, daily outdoor time during weekdays and weekend, daily time using devices with screens on most weekdays, and daily age-appropriate sleep hours. Daily fruit/vegetable consumption was calculated by averaging fruit and vegetable consumption. Daily outdoor time was estimated as $(\text{daily weekday outdoor time} \times 5 + \text{daily weekend outdoor time} \times 2) / 7$. Survey-weighted logistic regressions, adjusting for covariates (age, sex, race/ethnicity, family structure, and household poverty level), were used to measure associations between each health-related behavior and ASD status. Full model additionally adjusted for other health-related behaviors. Tests for trend were conducted by modelling health-related behaviors as continuous variables and P-value for trend (P-trend) were reported.

Results: After excluding participants with missing variables, the final sample included 18,386 children aged 3–5 years (719 with ASD and 17,667 without ASD). The final sample included

33% 3-year old children and 33% 4-year old children, 50% boys, 66% non-Hispanic White (NHW), and 41% with income<300% of poverty level, and 76% with family structure of two married parents. Compared to children without ASD, children with ASD were more likely to be 4- or 5-year-old, male, non-white, from lower-income households, and from non-two-parent married households (all $P<0.01$). From the full model, greater fruit/vegetable consumption was associated with significantly lower odds of ASD, with adjusted odds ratios (aORs) ranging from 0.21 (95% confidence interval (CI): 0.10-0.46) to 0.38 (95% CI: 0.19-0.74) across consumption categories compared with no fruit/vegetable intake (P -trend=0.011, Table 1). Increased outdoor time was also associated with lower odds of ASD, with children spending ≥ 2 hours outdoor daily having 29%–63% lower odds of ASD than those spending ≤ 1 hour outdoor daily (P -trend<0.001). Children sleeping 10–13 hours daily had 25% lower odds (95% CI: 0.55-1.03) of ASD than those sleeping <10 hours ($P=0.076$). Compared to children with <1-hour daily screen time, the aOR ranged from 1.44 (95% CI: 0.90, 2.29) for 1 hour to 3.17 (95% CI: 1.74, 5.78) for >4 hours screen time (P -trend=0.002).

Conclusions: Among U.S. children aged 3–5 years, greater fruit/vegetable consumption, increased outdoor time, lower screen time, and adequate sleep were strongly associated with lower odds of ASD. These findings highlight important behavioral differences between children with and without ASD, suggesting that young children with ASD may be more likely to engage in less healthy lifestyle behaviors. Interventions that promote healthy eating, outdoor activity, healthy sleep habits, and reduced screen time may help improve overall health and well-being among children with ASD.

Table 1. The associations of health-related behaviors with ASD.

Health-related behaviors	Model adjusting for covariates		Full model: adjusting for covariates and other health-related behaviors	
	OR (95% CI)	P-Value	OR (95% CI)	P-Value
Fruit/vegetable consumption				
0	Reference	-	Reference	-
1-3 times/week	0.34(0.18,0.66)	0.002	0.38(0.19,0.74)	0.005
4-6 times/week	0.18(0.09,0.35)	0.000	0.21(0.10,0.42)	0.000
1 time/day	0.25(0.12,0.53)	0.000	0.33(0.16,0.69)	0.003
2 times/day	0.14(0.07,0.28)	0.000	0.21(0.10,0.43)	0.000
≥ 3 times/day	0.13(0.06,0.27)	0.000	0.21(0.10,0.46)	0.000
P-trend		0.000		0.011
Daily outdoor time				
≤ 1 hour	Reference		Reference	
2 hours	0.63(0.43,0.93)	0.019	0.71(0.49,1.03)	0.069
3 hours	0.40(0.26,0.63)	0.000	0.50(0.32,0.77)	0.002
≥ 4 hours	0.30(0.16,0.55)	0.000	0.37(0.20,0.69)	0.002
P-trend		0.000		0.000
Daily sleep hours				
<10 hour	Reference		Reference	

10-13 hour	0.67(0.48, 0.94)	0.021	0.75(0.55, 1.03)	0.076
Daily screen time				
<1hour	Reference		Reference	
1 hour	1.49(0.94,2.37)	0.089	1.44(0.90,2.29)	0.129
2 hours	1.71(1.11,2.64)	0.016	1.57(1.01,2.46)	0.047
3 hours	1.87(1.15,3.03)	0.011	1.46(0.87,2.43)	0.152
≥4 hours	4.21(2.40,7.39)	0.000	3.17(1.74,5.78)	0.000
P-trend		0.000		0.002

Survey-weighted logistic regression was used and covariates included age, sex, races, family structure, and percent of poverty level.

OR: Odds Ratio; CI: confidence interval.

Biography

Anna Shen is a high school student with a strong interest in child development and public health. Inspired by her experiences growing up with an autistic brother, Shen is particularly interested in understanding factors that support the health and well-being of children with autism spectrum disorder. Shen's academic interests include healthy nutrition, physical activity, and lifestyle behaviors that promote brain health and development. Through research, Shen hopes to contribute to a better understanding of how healthy habits can improve outcomes and quality of life for children and families affected by autism.



Anuradha Sharma

Faculty of Pharmaceutical Sciences, PCTE Group of Institutes,
Ludhiana, India

Structure-based virtual screening and molecular docking of ALZ-801 analogues targeting A β aggregation for Alzheimer's disease therapy

Alzheimer's disease (AD) is a progressive neurodegenerative disorder in which soluble β -amyloid (A β) oligomers are key upstream drivers of synaptic dysfunction and cognitive decline. ALZ-801 (valiltramiprosate) is an oral valine-conjugated prodrug of tramiprosate that inhibits A β 42 oligomer formation by a multi-ligand enveloping interaction with A β monomers, stabilizing them and preventing misfolding and aggregation. In this context, the present work employs structure-based virtual screening and molecular docking to design and prioritize ALZ-801-inspired small molecules targeting A β 42, with the goal of enhancing anti-oligomer potency and optimizing drug-like properties. Candidate ligands are evaluated for binding to key residues (e.g., Lys16, Lys28, Asp23) that mediate tramiprosate's enveloping mechanism, ranked by predicted affinity and interaction patterns, and filtered using *in silico* ADMET profiling. By integrating clinically validated mechanistic insights from ALZ-801 with rational *in-silico* drug design strategies, this work provides a systematic framework for the development of next-generation oral A β anti-oligomer agents. The findings highlight the role of computational approaches in accelerating early-stage discovery and advancing disease-modifying therapeutic options for early intervention in Alzheimer's disease. This computational pipeline not only refines ALZ-801's mechanism but also paves the way for potent, orally bioavailable therapeutics to halt oligomer-driven pathology.

Biography

Dr. Anuradha Sharma is an accomplished Associate Professor and Coordinator in Pharmaceutical Chemistry and Pharmacognosy at PCTE Group of Institutes, Ludhiana, with

over 10 years in academia and 5 years in research. Holding a Ph.D. from Panjab University, Chandigarh, Sharma specializes in drug discovery, molecular docking, QSAR studies, and acridine derivatives as anti-amnesic agents for Alzheimer's disease, authoring 25+ peer-reviewed publications in journals like Current Topics in Medicinal Chemistry. Sharma has authored key books including A Handbook of Computer Aided Drug Design (2017), Computer Assisted Drug Design, Discovery and Delivery System (2023), Futuristic Trends in Pharmacy and Nursing (2024, editor), and Advances in Protein-Peptide Chemistry (2026, editor), plus chapters in CRC Press volumes. Securing grants like USD 400 from the International Association of Parkinsonism for the Milan World Congress, DBT partial funding, and INSA-CSIR fellowships, Sharma earned TIFAC-DST Women Scientist status and Young Scientist Award 2021. An invited reviewer, editor, and conference organizer, Dr. Sharma excels in computational design and mentoring.



Beka Solomon

Tel Aviv University, Israel

Bone marrow-derived microglia may have a clinical value for treatment of Alzheimer's disease

The CXCR4/CXCL12 axis is a major signal transduction involved in inflammation, cell migration, haematopoiesis and cell homing in the bone marrow. Inhibition of this axis with AMD3100, a reversible antagonist of CXCR4, attenuates the neuroinflammation and mobilizes endogenous Hematopoietic Stem Cells (HSCs) from the bone marrow into the periphery. These cells can self-renew, proliferate, cross the blood-brain barrier, promote brain repair by inducing neurogenesis, releasing anti-inflammatory cytokines and hematopoietic growth factors, and interacting with resident neuroglia and transdifferentiating into microglia-like cells. We chronically treated the 3xTg mice model of Alzheimer's disease (AD) with 5mg AMD3100 for four months. The treatment significantly improved memory deficits and attenuate AD pathology. Another finding from our lab showed that AMD3100 treatment upregulates Monocarboxylate Transport 1 (MCT1), the major L-lactate transporter in the brain. The combined treatment of AMD3100 and exogenous lactate resulted in a significant beneficial effect compared to the untreated groups and each component alone regarding cognitive/memory functions as well as alleviated pathology with no adverse effects. To conclude, the inhibition of the CXCL12/CXCR4 axis result in prevention of neuroinflammation via mobilization of HSCs to the bloodstream, suggesting that microglia-targeted therapy may be an effective strategy for the treatment of Alzheimer's disease.

Biography

Prof. Solomon is Tel Aviv University Professor and former Chair for Biotechnology of Neurodegenerative Diseases. After graduate her PhD at Weizmann Institute of Science, in 1977 in Biochemistry and Biophysics, she joined Tel-Aviv University. A world leader in Alzheimer's research and pioneer of the immunotherapeutic approach Prof. Solomon

has the distinction of being the first investigator to recognize the potential for anti –beta antibodies as a therapy for Alzheimer's disease. She was awarded by Alzheimer's Association with the prestigious Zenith Award and the Alzheimer's Drug Discovery Foundation supported her research in Alzheimer's vaccines. She published more than 200 scientific articles in prestigious journals and have a respectable number of international patents.



Bethany Zanrucha^{1*}, Marc C. Patterson¹, Tatiana Bremova-Ertl², Michael Strupp³, Jorgji Kerthi¹, Janelle Raymond¹, Chidinma Ejim¹, Taylor Fields¹, Kyriakos Martakis^{4,5,6}

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A phase 3, multinational, randomized, placebo-controlled, double-blind, crossover trial (IB1001-304) evaluating levacetylleucine in CACNA1A

Objective: To assess the efficacy, safety, and tolerability of levacetylleucine in people with CACNA1A-related disorders.

Background: CACNA1A-related disorders refer to a range of rare, progressive, neurological and neurodevelopmental disorders with pathogenic mutations in the Calcium Voltage-Gated Channel Subunit Alpha1 A (CACNA1A) gene. The gene is primarily expressed in neurons and plays a critical role in calcium-ion signaling. Levacetylleucine enters enzyme-controlled pathways to correct metabolic dysfunction, improve energy production, and normalize lysosomal calcium levels, thereby stabilizing inter-ion calcium signaling i.e., distribution of calcium ions between the cytoplasm, endoplasmic reticulum, and lysosomes. This leads to neuronal membrane potential normalization and inter-cellular signaling. Clinical trials of levacetylleucine in disorders featuring ataxia and cognitive impairment such as Niemann-Pick disease type C, GM2 gangliosidosis, and ataxia-telangiectasia have demonstrated an improvement in neurological signs and symptoms, and levacetylleucine was shown to be safe and well-tolerated.

Methods: IB1001-304 will enroll participants ≥ 4 years with a genetically confirmed diagnosis of CACNA1A-related disorders. Participants will be assessed during three study periods: a baseline period, after which they will be randomized (1:1) to receive treatment

with levacetylleucine or placebo for 12-weeks during the first intervention period (Period 1). Following Period 1, participants will crossover to receive the opposite treatment for 12-weeks during a second intervention period (Period 2). An Extension Phase will be planned to allow participants who participated in the trial to have further access to levacetylleucine. Treatment will be administered orally, and participants will receive a total daily dose of 2-4 g/day based on weight-tiered doses.

Results: The primary efficacy endpoint will be the Scale for the Assessment and Rating of Ataxia. Secondary endpoints include Spinocerebellar Ataxia Function Index, quality of life (EQ-5D-5L for participants >18 years and EQ-5D-Y for participants <18 years), Neurology Quality of Life-Upper Extremity Function, Clinical Global Impression of Improvement, Scale for Ocular Motor Dysfunction in Ataxia, and others. Safety assessments include adverse event incidence and severity.

Conclusion: CACNA1A-related disorders are a group of neurological disorders with no approved treatments. IB1001-304 will evaluate the efficacy and safety of levacetylleucine in improving neurological signs and symptoms, functioning, and quality of life in people with CACNA1A-related disorders.

Biography

Beth Zanrucha, PharmD, has more than a decade of Medical Affairs experience and over 15 years in pharmacy practice, spanning rare neurogenetic, neuromuscular, and lysosomal disease at Sarepta Therapeutics and IntraBio Inc. During her career she has built Medical Affairs capabilities across medical information, medical managed care, field medical, and global medical strategy, supporting first-in-disease and first-in-class launches in the United States and Europe. She has served as team lead for a gene therapy development program, delivered clinical and health economic evidence to national payers and state Medicaid boards, and established thought leader and advisory board programs globally. Currently, she is serving as Senior Medical Director, Global Medical Affairs at IntraBio Inc., a biopharmaceutical company developing therapies for rare and orphan neurological and neurodegenerative diseases, where she leads global medical strategy across Niemann-Pick disease type C, ataxia-telangiectasia, GM2 gangliosidosis, and CACNA1A-related disorders.



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Results from the global phase 3 trial (IB1001-303) evaluating levacetylleucine in children and adults with ataxia-telangiectasia

Objective: To report efficacy and safety results from the pivotal, Phase 3 trial investigating levacetylleucine in people living with Ataxia-Telangiectasia (A-T).

Background: A-T is a rare, autosomal recessive cerebellar ataxia. Levacetylleucine (N-acetyl-L-leucine) is a modified amino acid that enters enzyme-controlled pathways to correct metabolic dysfunction, improve function of the lysosomal-mitochondrial axis, and restore membrane potential and cellular signaling. The Phase 3, randomized, double-blind, placebo-controlled trial (IB1001-303) evaluated the safety and efficacy of levacetylleucine in pediatric and adult individuals with A-T.

Design/Methods: IB1001-303 enrolled A-T participants aged 4 years or older across 10 multinational trial sites. Primary efficacy endpoint was the Scale for the Assessment and Rating of Ataxia (SARA). Secondary endpoints included Spinocerebellar Ataxia Function Index (SCAFI), International Cooperative Ataxia Rating Scale (ICARS), Neurology Quality of Life-Upper Extremity Function (NeuroQOL-UEF), Quality of Life (EQ-5D), and investigator, caregiver, and patient Clinical Global Impression of Improvement (CGI-I). Safety assessment included adverse event incidence and severity.

Results: Seventy-three participants aged 4 to 50 were enrolled. The primary endpoint was met; mean change in the SARA total score with levacetylleucine was -1.92 (SD=2.81) and -0.14 (SD=2.38) with placebo; Linear Mixed Model (LMM) treatment effect -1.88 (SE=0.41) (95% CI: -2.70, -1.06; $P<0.001$). The trial met key secondary endpoints, including ICARS (95% CI: -4.87, -0.81; $P=0.003$) and Investigators' CGI-I (95% CI -0.7, 0.0; $P=0.02$). No treatment emergent adverse events occurred in more than 10% of participants on levacetylleucine and no serious adverse events or deaths occurred in either group.

Conclusions: Levacetylleucine demonstrated a significant benefit versus placebo, and clinically meaningful improvements in neurological manifestations of A-T, functioning, and quality of life, and a favorable benefit-risk profile for the treatment of A-T. Levacetylleucine was observed to be safe and well-tolerated, consistent with its established safety profile.

Biography

Beth Zanrucha, PharmD, has more than a decade of Medical Affairs experience and over 15 years in pharmacy practice, spanning rare neurogenetic, neuromuscular, and lysosomal disease at Sarepta Therapeutics and IntraBio Inc. During her career she has built Medical Affairs capabilities across medical information, medical managed care, field medical, and global medical strategy, supporting first-in-disease and first-in-class launches in the United States and Europe. She has served as team lead for a gene therapy development program, delivered clinical and health economic evidence to national payers and state Medicaid boards, and established thought leader and advisory board programs globally. Currently, she is serving as Senior Medical Director, Global Medical Affairs at IntraBio Inc., a biopharmaceutical company developing therapies for rare and orphan neurological and neurodegenerative diseases, where she leads global medical strategy across Niemann-Pick disease type C, ataxia-telangiectasia, GM2 gangliosidosis, and CACNA1A-related disorders.



Clairisse Whang

Robert Wood Johnson University Medical School, New Brunswick, NJ, United States

Anti-NMDA receptor encephalitis masquerading as refractory viral meningitis

Anti-N-methyl-D-aspartate receptor (NMDA-R) encephalitis is a rare but increasingly recognized cause of acute encephalopathy, often masquerading as infectious meningoencephalitis. We present a case of a 45-year-old man with prediabetes, psoriasis, and recent-onset seizures, who experienced a rapid two-day decline in mental status. Initial symptoms included headache, aphasia, left facial droop, and confusion, prompting admission and extensive evaluation for acute stroke and infectious etiologies.

Neuroimaging revealed progressive leptomeningeal enhancement and cortical T2 hyperintensities, predominantly in the right cerebral hemisphere. Cerebrospinal Fluid (CSF) analyses repeatedly demonstrated lymphocytic pleocytosis and elevated protein, further supporting an inflammatory or infectious process. Despite broad-spectrum antimicrobials and antivirals, the patient's neurological status deteriorated, requiring intubation and transfer to the medical intensive care unit. Serial imaging continued to show worsening leptomeningeal involvement, and the clinical picture was complicated by recurrent seizures, autonomic instability, and secondary infections.

The diagnostic process was confounded by the overlap of clinical and radiographic features with viral or bacterial meningoencephalitis. Multiple infectious workups—including BioFire, PCR panels for common neurotropic viruses, and cultures—were negative. Initial autoimmune panels were equivocal, with only a weakly positive anti-NMDAR antibody in serum. It was not until a repeat Mayo Clinic panel on day 16 of hospitalization that CSF anti-NMDAR antibodies were definitively detected, confirming the diagnosis of anti-NMDAR encephalitis. Immunomodulatory therapy with high-dose steroids, intravenous immunoglobulin, and plasma exchange was initiated, but neurological improvement remained minimal, with only a gradual rise in Glasgow Coma Scale.

This case underscores the diagnostic challenges posed by anti-NMDAR encephalitis, particularly when neuroimaging and CSF findings mimic infectious etiologies. The delay in diagnosis—attributable to the initial focus on infection and the time required for confirmatory autoimmune testing—may have contributed to the patient's prolonged encephalopathy and poor neurological recovery. Early consideration of autoimmune encephalitis in patients with unexplained encephalopathy and suggestive imaging is essential, as prompt initiation of immunotherapy is associated with improved outcomes.

Long-term prognosis in anti-NMDAR encephalitis is closely linked to the timing of diagnosis and treatment. This case highlights the need for increased awareness and expedited evaluation for autoimmune causes in patients with atypical or refractory meningoencephalitis, to minimize morbidity and optimize recovery.

Biography

Clairisse Whang is a third-year medical student at Rutgers Robert Wood Johnson Medical School and a graduate of Rutgers University, where Whang majored in Cell Biology/ Neuroscience and Cultural Anthropology. Clairisse is actively involved in Whang's local AMA chapter, the Human Rights Association, and community health initiatives – specifically starting a nutritional gardening initiative at the local homeless shelter, doing 1-on-1 refugee mentoring, and working as a student doctor in the RWJMS uninsured patient clinic. Whang is pursuing infectious research and an MD with the long-term goal of addressing health disparities and advancing equity through clinical care and health policy.



Courtney M. Klotz, M.Ed.

Renaissance School of the Eastern Shore, United States

Bridging neurology and education: Translating brain function into effective educational practice

Background: Children with neurological, neurodevelopmental, autoimmune, and functional neurological disorders spend significantly more time in educational settings than in clinical environments. While neurologists diagnose and treat the underlying condition, educators are responsible for implementing daily interventions that influence academic success, emotional well-being, and long-term outcomes. However, many educators receive little formal training in brain function or the neurological basis of learning and behavior.

Objective: To demonstrate how increasing educators' understanding of brain function and neurological diagnoses improves empathy, reduces misinterpretation of behaviors, and enables the development of individualized educational plans that better align with each student's neurological profile.

Discussion: When educators understand the neurological mechanisms underlying executive dysfunction, cognitive fatigue, sensory processing, language retrieval, autonomic dysfunction, and emotional regulation, behaviors previously perceived as defiance, laziness, or lack of motivation become recognizable manifestations of neurological impairment. This shift in perspective promotes more effective accommodations, targeted instructional strategies, improved educator-family collaboration, and stronger communication with healthcare providers. Educational observations also provide valuable longitudinal functional data that can complement neurological evaluations.

Conclusion: Education and neurology should function as complementary disciplines rather than independent systems. By equipping educators with foundational knowledge of brain

function and neurological disorders, schools can move beyond behavioral management toward neurologically informed instruction. This interdisciplinary approach has the potential to improve academic achievement, enhance quality of life, strengthen clinical collaboration, and ultimately produce better outcomes for students with neurological conditions.

Conceptual Framework: Diagnosis explains why the brain functions differently. Brain function explains how the student learns, processes information, regulates emotions, and performs daily tasks. When educators understand these neurological principles, empathy increases, stigma decreases, and instructional planning becomes individualized rather than behavior-driven. The result is improved collaboration between educators, families, and healthcare providers, leading to better academic and functional outcomes.

Biography

Courtney Moore Klotz, M.Ed. is the Executive Director and Co-Founder of Renaissance School of the Eastern Shore (RSES), a nonprofit school Klotz has led for the past 11 years, serving students with diverse learning needs through individualized, brain-based educational practices. Since beginning Klotz's career in 2003, Courtney has worked extensively in the fields of education, behavioral intervention, and social services, dedicating Klotz's career to helping children, families, and educators better understand how the brain learns and develops. Courtney holds a Master of Education and is an IBCCES Board Certified Cognitive Specialist and Student Mental Health Specialist. Klotz has completed advanced training in nonprofit leadership, cognitive science, crisis prevention, and educational leadership, and is the founder of Reawakening Education™, an organization focused on equipping educators with practical, neuroscience-informed tools and innovative educational technology. Recognized for Klotz's engaging and practical presentation style, Courtney speaks nationally on topics including executive functioning, neurodiversity, behavior, trauma-informed education, cognitive science, and creating meaningful supports for students with learning differences. Klotz's mission is to bridge the gap between neuroscience and education, empowering educators with strategies that foster both academic success and lifelong confidence in every learner.



Daniel J. Curry, MD

Director, Functional Neurosurgery and Epilepsy Surgery, Texas Children's Hospital; John S. Dunn Foundation Endowed Chair for Minimally Invasive Epilepsy Surgery; Professor of Neurosurgery, Baylor College of Medicine, Houston, Texas, United States

Gene therapy in pediatric neurosurgery: Delivery strategy and the translational path toward hypothalamic hamartoma

Background: Gene therapy is reshaping the treatment of pediatric neurological disease, offering durable, non-destructive correction of conditions long considered untreatable. This lecture reviews the spectrum of intracerebral gene therapy strategies—gene addition, modification, deletion, and regulation—and positions pediatric neurosurgery as central to their safe and effective delivery. The Adeno-Associated Virus (AAV) remains the dominant vector for central nervous system applications, yet its limited cargo capacity, species-dependent tropism, and modest transduction efficiency in human tissue constrain translation. Equally decisive is the route of delivery: Convection-Enhanced Delivery (CED), intraventricular infusion, and intrathecal administration, along with adjuncts of focused-ultrasound blood–brain–barrier opening each present distinct trade-offs in efficiency, invasiveness, and off-target risk.

Clinical Precedent: From the early Canavan and Batten disease trials to the 2024 FDA approval of eladocogene exuparvovec (Kebilidi) for Aromatic L-Amino Acid Decarboxylase (AADC) deficiency, the first gene therapy delivered directly into the human brain, this precedent demonstrates both the promise and the surgical demands of the field. Pediatric delivery introduces distinct challenges, including skull fragility, cranial fixation risk, and the need for floating-frame, skull-mounted, and robotic stereotactic platforms, alongside vector-related toxicities such as insertional mutagenesis, immune and transgene-mediated adverse events, and dorsal root ganglion toxicity.

Translational Framework: The lecture culminates in a translational framework for Hypothalamic Hamartoma (HH), an intrinsically epileptogenic lesion for which intralesional, physiologically based gene therapy—such as activity-dependent hyperpolarizing constructs—

may offer a non-destructive alternative to ablation. Realizing this potential will require studies on living human HH tissue, networked biopsy and tissue-banking protocols, advances in cell-type-specific tropism, and early, sustained collaboration between neurosurgeons and biologics developers. Early neurosurgical engagement in delivery design is identified as a critical determinant of safe and effective therapy.

Keywords: Gene Therapy, Pediatric Neurosurgery, Adeno-Associated Virus (AAV), Convection-Enhanced Delivery, Hypothalamic Hamartoma, AADC Deficiency, Stereotactic Delivery, Epilepsy.

Biography

Daniel J. Curry, MD, is the John S. Dunn Foundation Endowed Chair for Minimally Invasive Epilepsy Surgery, Director of Functional Neurosurgery and Epilepsy Surgery at Texas Children's Hospital, and Professor of Neurosurgery at Baylor College of Medicine in Houston, Texas. Curry's clinical and research work centers on minimally invasive and stereotactic approaches to pediatric epilepsy, including MR-guided laser interstitial thermal therapy and intracerebral drug and gene delivery. Curry has been an early adopter of direct-to-brain gene therapy in pediatric practice, participating in the clinical program supporting MR-guided, convection-enhanced delivery of AADC deficiency gene therapy. Curry's current research interests include convection-enhanced delivery, AAV tropism in human brain tissue, and novel cell- and gene-therapy strategies for hypothalamic hamartoma.



Daniel Zhang

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Pasadena Bio Collaborative Labs, Pasadena, CA, United States

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

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

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

Results: In Study 1, male rats showed a statistically significant dorsal-to-ventral glial density gradient (ACA: 78.3 vs. pooled ventral: 175.7 objects/100,000 px²; p=0.047). Female rats showed a numerically comparable gradient that narrowly missed significance (p=0.068), with large effect sizes in the ACA (Hedges' g=1.91) and ILA (g=1.18), consistent with insufficient power at n=3. In Study 2, neither GFAP nor AIF1 differed by sex in isolation. However, when patients were stratified by EGFR amplification status, amplification was significantly associated with an elevated GFAP/AIF1 ratio in male patients (amplified: 1.420 vs. non-amplified: 0.319; p=0.010) but not in female patients (0.876 vs. 0.658; p=0.552).

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

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

Biography

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



Dev Sathyan

Bridgeland High School, Cypress, Texas, United States

Neuro-fencing: A therapeutic framework for enhancing neurocognitive and social function in children with autism spectrum disorder

Neuro-Fencing is a therapeutic framework integrating Sport-Based Intervention (SBI) with the technical demands, interpersonal dynamics, and neurocognitive requirements of competitive fencing, designed to enhance executive function, emotional regulation, and social engagement in children with Autism Spectrum Disorder (ASD) and associated developmental disabilities. Despite growing evidence that structured physical activity improves neurocognitive outcomes in this population, no formal therapeutic protocol has previously positioned fencing as its central modality. This paper presents the theoretical foundations of the Neuro-Fencing framework alongside preliminary affective outcome data from a pilot camp study, addressing the question: how does structured fencing participation influence mood, calmness, attentional capacity, and social anxiety in children with developmental disabilities?

The framework is organized around six core therapeutic components — anticipation, spatial awareness, rhythmic sequencing, regulated contact, attentional synchrony, and emotional modulation — each inherent to competitive fencing practice. The fencing bout, framed as a rule-bound, predictable, and non-aggressive physical exchange, provides structured experiences of turn-taking, boundary regulation, and opponent-responsive decision-making that serve as naturalistic organizers of inhibitory control, joint attention, and emotional regulation. Critically, the fencing mask removes the demands of facial expression reading, replacing complex social cues with a simplified, movement-based interaction language well suited to the systematizing cognitive profile frequently associated with ASD, effectively lowering the social-perceptual load of dyadic engagement while preserving its relational structure.

From a neuroscientific standpoint, the intervention is grounded in three converging mechanisms. First, the cognitively demanding physical activity embodied in fencing upregulates Brain-Derived Neurotrophic Factor (BDNF), promoting neuroplasticity in prefrontal and hippocampal circuits that underpin learning, memory, and executive control. Second, fencing's continuous opponent-intention reading activates the mirror neuron system, providing structured and motivating exercise of social-perceptual neural networks that are atypically engaged in ASD. Third, long-term fencing practice has been shown to produce durable functional adaptations in frontal inhibitory control circuits, with experienced fencers demonstrating significantly enhanced neural responses on inhibitory tasks compared to non-fencing peers. Together these mechanisms provide a coherent and mutually reinforcing neurobiological rationale for fencing as a therapeutic modality targeting the executive and social-cognitive profiles characteristic of ASD.

A preliminary pilot sub-study was conducted with 31 children presenting with general developmental disabilities at a structured therapeutic fencing camp. Affective state was assessed using a validated four-item visual emoji scale (1–4) administered before and after the camp, measuring excitement and curiosity, attentional focus, calmness, and social anxiety. With social anxiety items reverse-scored, mean adjusted scores improved from 11.65 to 11.90 out of a possible 16. The most clinically meaningful gains were observed in the primary therapeutic target domains: calmness and relaxation improved by 0.26 points and social anxiety reduced by 0.10 points, both directionally consistent with Neuro-Fencing's core predictions. Focus and attention showed a modest positive trend of 0.10 points. A slight decrease in excitement scores is interpreted as normalization of novelty-driven arousal — a well-documented pattern in sport intervention research reflecting sustained engagement rather than declining motivation. Thirty-five percent of participants demonstrated measurable overall improvement, 29% remained stable, and 35% showed mixed responses, consistent with variability expected in an uncontrolled pilot across a diagnostically heterogeneous population.

These findings provide meaningful proof-of-concept that therapeutic fencing participation produces positive directional trends in the affective domains most central to neurodevelopmental intervention: emotional regulation, calmness, and social comfort. Neuro-Fencing constitutes an innovative, evidence-informed proposal uniting the cognitive demands of combat sport, neuroscientific insights into exercise-induced plasticity, and the principles of embodied developmental therapy. The results support and motivate a larger randomized controlled study with standardized neurocognitive and social outcome measures, positioning Neuro-Fencing as a rigorous, engaging, and participant-endorsed pathway toward neurocognitive well-being in children with ASD and developmental disabilities.

Biography

Dev Sathyan is a high school researcher at Bridgeland High School (Class of 2028) with a strong focus on neuroscience and biomedical research. Sathyan is a published researcher through the Rising Researchers Program, where Sathyan conducted a systematic literature analysis on

the impact of social isolation during COVID-19 on cardiovascular and mental health. Sathyan's current research investigates the behavioural and mood effects of fencing on children and young adults with special needs, exploring brain-behaviour connections through pre- and post-activity assessments. Sathyan is a 2025 National Science Bee qualifier, 2026 National Biology Bee Semi-finalist and Harvard MedScience Clinical Program participant.



Dimitris Santorinaios

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The NERVE framework: AI-personalized virtual reality and closed-loop neuromodulation for trauma-linked symptom states

Psychiatry still relies heavily on retrospective symptom reports and static diagnostic categories, despite the fact that many clinically relevant symptoms emerge as dynamic, context-dependent brain states. This creates a major translational challenge: how can psychiatry move from observing symptoms after they occur toward experimentally evoking, detecting and adaptively regulating them in real time?

Post-Traumatic Stress Disorder (PTSD) provides a compelling translational model for neuroadaptive psychiatry because clinically meaningful symptom states — including hyperarousal, threat reactivity, intrusive memory, affective dysregulation and avoidance — can be ethically and experimentally evoked, measured and potentially modulated. At the same time, advances in AI-driven Virtual Reality Exposure Therapy (VRET), multimodal biomarkers and closed-loop neuromodulation suggest the possibility of more dynamic and personalized circuit-based interventions.

This presentation introduces the NERVE Framework (Neuroadaptive Evoked Regulation via Virtual Environments), a translational neuropsychiatric architecture designed to integrate personalized symptom provocation, multimodal state estimation and adaptive neuromodulatory intervention within a closed-loop framework. The central aim is not to propose a finalized therapeutic protocol, but to define a clinically grounded and experimentally testable framework for transitioning from symptom provocation to real-time adaptive regulation.

The framework was developed through an extensive translational synthesis of recent literature across virtual reality exposure therapy, computational psychiatry, digital phenotyping, behavioral and physiological biomarkers, EEG-informed personalization, inflammatory correlates of stress dysregulation and closed-loop neuromodulation technologies including TMS and DBS, combined with clinically informed observations from trauma-focused psychological practice. The proposed model is organized as a provocation–detection–intervention loop: AI-personalized VR environments evoke trauma-relevant symptom states; multimodal physiological, behavioural and affective signals estimate state dynamics in real time and neuromodulatory parameters are adaptively adjusted according to timing, circuitry and individual neurophysiological profiles.

Current evidence supports the feasibility of several components independently: VR can evoke ecologically valid trauma-related responses, multimodal AI systems can infer affective and physiological state changes, and adaptive neuromodulation research highlights the importance of timing, state-dependence and individualized targeting. However, these advances remain largely siloed across disciplines. The major translational gap is the absence of unified neuroadaptive systems capable of integrating personalized symptom provocation, real-time state detection and adaptive circuit-level intervention within a single closed-loop architecture.

In conclusion, PTSD may provide an ideal translational testbed for neuroadaptive closed-loop psychiatry by enabling experimentally accessible yet clinically meaningful symptom–state modulation. The NERVE Framework reframes precision psychiatry from the right treatment for the right diagnosis toward the right signal, in the right patient, at the right moment, for the right symptom state.

Biography

Dimitris Santorinaios is a Clinical Psychologist and Psychodynamic Psychotherapist with a multidisciplinary background in clinical mental health, trauma-focused psychotherapy and translational psychiatry. Santorinaios holds an MSc in Clinical Mental Health Sciences from University College London (UCL), where Santorinaios also worked as a Research Assistant, and has clinical experience within the South London and Maudsley NHS Foundation Trust. Santorinaios serves as a Supervisor at the Clinical Research Laboratory, Department of Psychology, National and Kapodistrian University of Athens. Santorinaios's work focuses on computational and neuroadaptive models of psychiatry, with particular interest in real-time symptom-state detection and adaptive closed-loop interventions for trauma-related psychopathology.



Elena Ward*, Andrew Hill, Lauren Hepworth

St Helens & Knowsley Foundation Trust Hospitals,
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Exploring retinal artery occlusion pathways to care: Could thrombolysis be implemented with current pathways?

Introduction: Central Retinal Artery Occlusion (CRAO) or ocular stroke, has an incidence between 1-10 per 100,000, causing sudden and painless vision loss. Following CRAO there is an increased risk of cerebral stroke, rendering it a potential sight and life-threatening event. Intravenous thrombolysis has been shown to increase visual recovery rates if given within 4.5 hours of onset, compared to standard therapy and natural history. Therefore, direct therapy and provision of secondary prevention are time critical. The American Heart Association in 2021 recommended systems of care should evolve to prioritise early recognition and triage of CRAO. This study aimed to evaluate the time taken from onset to arrival, time to diagnosis and treatment, with the overall aim to judge whether thrombolysis is viable with current pathways.

Methods: This single-centre retrospective records review identified 41 individuals referred to TIA clinic with a diagnosis of retinal artery occlusion between February 2024 and February 2025. 19 cases were excluded due to insufficient data regarding the onset of symptoms, incidental finding diagnosis or re-referral from a prior diagnosis. Data was extracted from electronic medical records of 22 individuals with a diagnosis of CRAO or Branch Retinal Artery Occlusion (BRAO), including: time from onset to arrival, referrals made, contact points, time between contact points and diagnosis and visual acuity outcome.

Results: Only 13.6% of the 22 individuals in this study presented to hospital within the 4.5-hour timeframe for thrombolysis. The median time from onset to first healthcare contact point was 2.2 days. 86.4% met with two different healthcare providers with no urgent referral to the stroke team. 36.4% sought help from a location where thrombolysis is currently administered. The

most common initial presentation locations were high-street opticians, optometrists or the emergency department. The primary delay was public lack of recognition of the severity of symptoms and seeking help too late.

Conclusion: This study identified inefficiencies in current referral pathways and public education, preventing timely intervention essential for the implementation of thrombolysis. The study results support findings from other studies which found thrombolysis rates for CRAO are much lower compared to cerebral stroke. Improvements including enhanced rapid referral protocols and campaigns to educate the public on CRAO/BRAO are the key to reducing avoidable, life changing vision loss and stroke prevention.

Biography

Elena Ward is a fourth-year medical student at the University of Liverpool, with a strong interest in neurology and acute medicine. Throughout Ward's studies, Ward has developed a passion for clinical research and its integration into patient care. Ward currently acts as President of the Liverpool Paediatric Society, co-ordinating educational events and promoting student engagement. Additionally, as Charity Representative for the Liverpool Medical Student's Society, Ward supports strategic outreach and fundraising initiatives for brilliant causes. Ward intends to pursue a career in clinical practice with continued focus on research, education and advancements.



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Venous hypertensive encephalopathy presenting as rapidly progressive dementia and parkinsonism secondary to a torcular Herophili dural arteriovenous fistula: A reversible stroke mimic

Background: Dural Arteriovenous Fistulas (DAVFs) involving the torcular Herophili are rare intracranial vascular malformations that may produce Venous Hypertensive Encephalopathy (VHE). Because patients frequently present with cognitive decline, gait disturbance, and parkinsonian features, they are often misdiagnosed as neurodegenerative disease or ischemic stroke, delaying definitive treatment.

Case Presentation: We report a 70-year-old woman who developed rapidly progressive cognitive decline, gait instability, parkinsonism, and aphasia over three months. Initial brain CT suggested lacunar ischemia, while serial MRI demonstrated progressive bilateral white-matter hyperintensities. Extensive investigations, including cerebrospinal fluid analysis, autoimmune encephalitis antibodies, toxicology screening, and metabolic evaluation, were unrevealing, and high-dose corticosteroid therapy failed to improve her condition. Repeat contrast-enhanced MRI identified early arterial-phase opacification of the left transverse sinus, raising suspicion for a vascular shunt. Digital subtraction angiography confirmed a torcular Herophili dural arteriovenous fistula supplied by branches of the left middle meningeal and bilateral occipital arteries with retrograde deep venous drainage. The patient underwent successful transarterial ONYX embolization, achieving complete angiographic obliteration of the fistula.

Results: One month after treatment, the patient demonstrated remarkable neurological recovery. Cognitive function improved from an untestable state to a Mini-Mental State

Examination score of 23, speech and gait significantly recovered, and follow-up MRI showed marked regression of bilateral white-matter abnormalities, consistent with reversal of Venous Hypertensive Encephalopathy.

Conclusion: Torcular Herophili DAVFs should be considered in patients presenting with rapidly progressive dementia, parkinsonism, and diffuse white-matter changes, particularly when the clinical course is progressive and conventional investigations are unrevealing. Early recognition of imaging clues followed by digital subtraction angiography is essential for diagnosis. Prompt endovascular embolization can reverse neurological deficits and prevent irreversible brain injury, emphasizing the importance of considering this rare but treatable vascular disorder in the differential diagnosis of rapidly progressive neurological decline.

Keywords: Dural Arteriovenous Fistula, Venous Hypertensive Encephalopathy, Torcular Herophili, Parkinsonism, Rapidly Progressive Dementia, Endovascular Embolization.

Biography

Professor Gao Yang is Professor of Neurology, Head of the Department of Neurology, and Dean of Yancheng People's No.1 Hospital, Affiliated Yancheng Hospital of Xuzhou Medical University, Jiangsu, China. He is an experienced vascular neurologist and neurointerventional specialist with expertise in acute ischemic stroke, cerebrovascular diseases, mechanical thrombectomy, and endovascular treatment of complex neurovascular disorders. His research focuses on stroke prevention, neurovascular intervention, and clinical outcomes after endovascular therapy. Professor Gao has authored numerous peer-reviewed publications and is actively involved in advancing stroke research, education, and multidisciplinary patient care.

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White matter disease as a modifier of recovery potential after cardiac arrest

Survival after cardiac arrest has improved substantially with advances in resuscitation techniques and post-cardiac arrest care. However, Hypoxic–Ischemic Brain Injury (HIBI) remains a major cause of mortality and long-term neurological disability among survivors. Consequently, predicting recovery of consciousness or functional outcome remains a major challenge in neurocritical care. Neuroprognostication after cardiac arrest is often more reliable at the physiological extremes. Patients with findings, such as preserved EEG background continuity and the absence of significant gray matter injury on diffusion-weighted MRI, frequently achieve favorable neurological recovery. In contrast, patients with established markers of severe brain injury, including extensive cortical or deep gray matter diffusion restriction on diffusion-weighted MRI, typically experience poor neurological outcomes. However, a substantial intermediate group exists in whom conventional prognostic markers are inconclusive or discordant, limiting the ability of standard clinical examination and neuroimaging to accurately predict recovery in this group.

Although White Matter Disease (WMD) following HIBI has been studied thoroughly in the literature, its involvement in predicting outcomes remains incompletely understood. Wallerian-like degeneration and diaschisis have been proposed as contributors to impaired consciousness after HIBI by disrupting distributed brain network activity.

We hypothesize that WMD exerts a nonlinear, reserve-based effect on neurological recovery after HIBI, rather than acting as a binary predictor of outcome. When WMD is minimal or absent, a floor effect occurs in which recovery is largely determined by the severity of the arrest itself and the extent of gray matter injury. Conversely, when WMD is severe, a ceiling effect emerges in which loss of network reserve limits the range of possible recovery despite a similar degree of HIBI. We propose that WMD is most informative in the intermediate range, where measures of white matter microstructure and connectivity are most likely to reveal relevant variation in recovery potential. We further propose a conceptual framework in which network reserve, defined as the capacity of large-scale brain networks to maintain effective information integration and flexible reconfiguration under structural and vascular constraints,

mediates neurological resilience following HIBI. In this model, network reserve serves as the bridge between vascular reserve and structural reserve, ultimately influencing cognitive recovery. We will discuss the contributions of diffusion tensor imaging, blood-oxygen-level-dependent cerebrovascular reactivity, functional magnetic resonance imaging and high-density electroencephalography to the evaluation of network reserve, reflecting the functional integrity and adaptability of large-scale brain networks. We believe that the integration of these modalities into a unified framework that combines structural, vascular, and network reserve may provide a more comprehensive model for predicting neurological recovery following HIBI.



Biography

Ghaith Alsabbagh is a medical graduate from the University of Sharjah, UAE, and currently serves as a neurology research fellow at the University of Illinois Chicago. Alsabbagh's research focuses on neuroprognostication after cardiac arrest, hypoxic-ischemic brain injury, neuromodulation and biomarker discovery in patients with opioid use disorder. Alsabbagh is involved in clinical trials investigating noninvasive brain stimulation and neurophysiological biomarkers in patients with opioid use disorder. This work integrates multimodal neuroimaging and electrophysiology to better understand large-scale brain network dynamics and develop biomarkers relevant to brain injury and neuropsychiatric disorders.



Dr. Ciro Ramos-Estebanez is the chief of the Section of Neurocritical Care at UI Health. A physician-scientist, Dr. Ciro specializes in critical care neurology, noninvasive brain stimulation research, and MRI research. Dr. Ramos-Estebanez's is an innovator who has produced basic science and clinical manuscripts in a number of neurological journals. His research interests include vascular cognitive impairment (VCI), and he is a member of the multinational Vascular Impairment of Cognition Classification Consensus Study (VICCS). His research also focuses on noninvasive brain stimulation and behavioral neurophysiology, and he has developed a number novel technologies in this area. In addition, he has developed a new MRI sequence that measures the cerebral metabolic rate and blood brain barrier permeability in-vivo. Dr. Ramos-Estebanez's other clinical research achievements have focused on neurocritical care and include novel diagnostic criteria for a neurocritical care condition, and a first description in the literature of a heroin complication in the central nervous system.



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Saliva-based testing in Alzheimer's disease: How do our findings compare with existing evidence?

Background: The demand for practical and minimally invasive biomarkers for Alzheimer's Disease (AD) has increased substantially, particularly in view of the logistical, economic, and acceptability constraints associated with cerebrospinal fluid and blood-based testing. Saliva has gained attention as an alternative biological matrix capable of reflecting neurodegenerative, inflammatory, and neuropsychiatric processes. Nevertheless, existing studies on salivary biomarkers in AD are highly variable with respect to target analytes, laboratory techniques, and clinical populations. Moreover, systematic comparisons between independent cohorts and previously published data remain limited, restricting the translational interpretation of findings. The present study sought to contrast results from an original salivary biomarker cohort with those reported in the literature, focusing on areas of agreement, inconsistency, and methodological influence.

Methods: We conducted an observational investigation including cognitively unimpaired individuals, participants with mild cognitive impairment, and patients with clinically established Alzheimer's Disease. Saliva samples were obtained following standardized collection protocols and analyzed for selected markers of neurodegeneration and inflammation using immunoassay-based methods. In parallel, a structured narrative comparison of published human studies evaluating salivary biomarkers in AD was undertaken. Studies were selected according to predefined criteria, including confirmed clinical diagnosis, use of saliva as the biological substrate, and quantitative biomarker measurement. Data on biomarkers, analytical platforms, cohort characteristics, and reported outcomes were systematically reviewed and qualitatively compared with findings from our cohort.

Results: Distinct salivary biomarker patterns were observed across cognitive stages in our cohort, with trajectories consistent with progressive neurodegeneration. Comparison with previously published studies revealed partial alignment for specific biomarkers, particularly those associated with tau-related pathology and inflammatory pathways. However, substantial variability across studies was evident, largely driven by differences in analytical sensitivity, saliva collection and processing procedures, and clinical heterogeneity of study populations. Several biomarkers described as promising in individual reports demonstrated limited reproducibility across independent investigations, highlighting the impact of methodological diversity.

Conclusion: These results reinforce the biological relevance of saliva as a source of biomarkers for Alzheimer's Disease while emphasizing the challenges posed by lack of standardization and inter-study comparability. The comparative framework applied in this study supports the role of salivary biomarkers as a complementary diagnostic approach and underscores the need for methodological harmonization and multicenter validation. Saliva-based testing represents a promising avenue for scalable screening and research, particularly in contexts where invasive procedures are impractical or inaccessible.

Biography

Gustavo Alves Andrade dos Santos is a Pharmacist-Biochemist (São Paulo, Brazil); Doctor in Biotechnology, Master in Pharmacy; Post Doctorate in Anatomy and Surgery from Neurobiology (University of Chicago), Clinical Pharmacy (University of Central Florida), USA; Hospital Pharmacy (Necker Hospital) Paris, France; dos Santos is a Professor at the São Leopoldo Mandic Faculty of Medicine, Araras; Member of the Alzheimer's Association International (ISTAART) and Member of the American Society of Health-System Pharmacists (ASHP). Assistant Researcher at UNICAMP.



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An overview of the protection afforded by kaempferol administration against rat's brain degeneration induced by ischemic stroke and 3-nitropropionic acid

Kaempferol, a flavonoid present in many food products, has chemical and cellular antioxidant properties that are beneficial for protection against the oxidative stress caused by reactive oxygen and nitrogen species. Kaempferol administration to model experimental animals provides extensive protection against brain damage of the striatum and proximal cortical areas induced by transient brain cerebral ischemic stroke and by the neurotoxin 3-nitropropionic acid, which elicits neurological motor disorders closely resemble those found in Huntington's disease. This presentation is an overview of the work performed in our laboratories during the past years on the molecular and cellular mechanisms of protection by kaempferol administration against brain damage induced by these insults. Kaempferol administration at doses that prevent neurological dysfunctions inhibit the critical molecular events that underlie the initial and delayed brain damage induced by ischemic stroke and by 3-nitropropionic acid. It is highlighted that the protection afforded by kaempferol against the initial mitochondrial dysfunction can largely account for its protection against the reported delayed spreading of brain damage, which can develop from many hours to several days. More recently, we have shown that Intraperitoneal (i.p.) administration of kaempferol also protects against the increase of pro-inflammatory cytokines that potentiate the activation of complement C3 protein (a biomarker of A1-type reactive astrocytes generation) and overproduction of neurotoxic amyloid β (A β) peptides in rats treated with acute i.p. administration of NPA. Furthermore, kaempferol i.p. administration also protects against the NPA-induced increase of phospho-tau 217 and phospho-tau 181. Also, our results unveiled that i.p. kaempferol co-administration prevent microhaemorrhages induced by acute NPA administration. This allows us to

conclude that kaempferol administration can be beneficial not only in preventive treatments, but also in post-insult therapeutic treatments, and our results lend strong support to its use in the therapeutic treatment of β -amyloidopathies and tauopathies.

Biography

Dr. Carlos 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 Molecular Pathology Biomarkers of the University of 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.



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A novel neuroprotective mechanism of Hexaraphane (6-MSITC) derived from wasabi

Hexaraphane (HXN; 6-methylsulfinylhexyl isothiocyanate) is an isothiocyanate compound derived from the rhizomes and roots of wasabi (*Eutrema japonicum*). Accumulating evidence demonstrates that HXN exerts potent antioxidant, anti-inflammatory, and neuroprotective activities, supporting its therapeutic relevance for neurodegenerative disorders such as Alzheimer's disease. Recent mechanistic studies have revealed that HXN not only activates the Nrf2 pathway but also suppresses tau phosphorylation through inhibition of Glycogen Synthase Kinase-3 β (GSK-3 β). Detailed analyses indicate that HXN attenuates GSK-3 β activity via the upregulation of Akt-mediated inhibitory phosphorylation at Ser9 as well as direct allosteric suppression of GSK-3 β catalytic function, leading to reduced tau phosphorylation at multiple pathological epitopes. Newly clarified pharmacokinetic data show that HXN is rapidly absorbed after oral intake, partially converted into bioactive metabolites, reaches peak plasma levels within 1–2 hours, and exhibits favorable tissue distribution including central nervous system penetration, providing a mechanistic rationale for its functional effects. Clinical studies in healthy middle-aged and older adults have shown that daily intake of HXN-containing Wasabi Sulfanyl™ (WS) leads to improvements in cognitive functions such as attention, judgment, and memory, while exploratory intervention in individuals with ME/CFS has indicated reductions in neurological symptoms including brain fog and photophobia. Building on these findings, a new randomized clinical study targeting younger adults is currently underway to evaluate HXN's effects on cognitive performance and neural resilience in early adulthood, and the results will be reported in due course. Collectively, the emerging mechanistic and clinical data highlight HXN as a promising neuroprotective compound with potential applicability not only to Alzheimer's disease but also to broader neurological conditions characterized by cognitive dysfunction and neuroinflammation.

Biography

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



Jessica Marchant

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Mapping neuroplasticity in occupational therapy: Evidence-based interventions with measurable neural outcomes

Over the past two decades, neuroscience research has increasingly demonstrated that the adult brain retains the capacity for reorganization following Acquired Brain Injury (ABI). Despite this growing body of evidence, rehabilitation practice often shifts quickly toward compensatory strategies, with limited integration of objective neural outcomes into clinical decision making. Occupational Therapy (OT) is well positioned to deliver interventions that support neural recovery, yet there remains no OT-focused synthesis that clearly connects OT-deliverable interventions with measurable evidence of neuroplastic change. This disconnect limits both clinical translation and interdisciplinary understanding of OT's role in recovery-oriented neurorehabilitation.

This presentation reports findings from a scoping review conducted as part of the Experiential Student Scholars Program at Texas Woman's University. Guided by PRISMA ScR and Joanna Briggs Institute methodology, the review maps OT-deliverable interventions for adults with ABI, including stroke, traumatic brain injury, and multiple sclerosis, that demonstrate measurable neuroplastic change. Inclusion was restricted to studies reporting research-supported neural biomarkers of brain reorganization, such as Functional MRI (fMRI), Diffusion Tensor Imaging (DTI), Electroencephalography (EEG), Transcranial Magnetic Stimulation-Derived Motor Evoked Potentials (TMS MEPs), and Functional Near-Infrared Spectroscopy (fNIRS), alongside functional performance measures.

The synthesis clarifies which OT interventions have been examined using neural measures, where evidence of brain reorganization coincides with functional improvement, and where commonly used interventions lack corresponding neural data despite widespread clinical

use. By organizing findings around these gaps, the review highlights persistent limitations in how neuroplasticity is evaluated within OT-relevant rehabilitation research.

Interventions examined include task-specific training, constraint-induced movement therapy, mirror therapy, bimanual training, sensory priming approaches, and technology-supported interventions such as robotics and virtual reality. The synthesis highlights where OT interventions have been paired with established neural biomarkers, an area that remains consistently underrepresented in the rehabilitation literature despite growing interest in recovery-focused care.

Findings are translated into clinician-focused evidence maps that visually link intervention types, neural measures, and functional domains. By centering neural outcomes supported by contemporary neuroscience research, this work clarifies OT's contribution to brain reorganization and functional recovery. This session provides clinicians and researchers with an evidence-informed framework for selecting OT interventions that are supported by objective measures of neuroplastic change and intended to promote neural recovery in adults with ABI.

Biography

Jessica Marchant is a doctoral student in Occupational Therapy (OTD) at Texas Woman's University, Houston, USA. She is an Experiential Student Scholar, where she conducted a scoping review examining OT-deliverable interventions with measurable neuroplastic outcomes in adults with acquired brain injury. This work forms the foundation of her doctoral capstone, which focuses on translating neuroplasticity research into recovery-oriented OT practice. Jessica has completed doctoral fieldwork in outpatient neurorehabilitation at TIRR Memorial Hermann. She has received academic and professional scholarships supporting her research and dissemination efforts.



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New treatment of permanent Bell's palsy through regeneration of peripheral facial nerve & facial muscles by increasing mitochondrial ATP

The facial nerve is one of the most commonly damaged cranial nerves. Loss of facial expression and deformation of facial paralysis have serious implications for the physical and psychological well-being of patients. Peripheral facial nerve paralysis is often treated as an aesthetic problem, but it can also have real physical and psychological problems. These include speech impairment, low self-esteem, decreased social interaction, oral incontinence, and dental problems, and a lack of progression of food through the mouth can cause tooth decay and bite the inside of the paralyzed cheek, resulting in repeated trauma and ulceration. Various strategies have been developed during the past century, especially during the past decades, to promote muscle and nerve recovery and regeneration, especially during the past decades. However, there is no clear solution yet. However, skeletal and peripheral nerves have remarkable regenerative capacity, mainly due to the spare pool of satellite (muscle stem cells) and neural stem cells in face. Therefore, this review showed that Bell's Palsy could be dramatically improved through regeneration of peripheral facial nerve & facial muscles by increasing mitochondrial ATP without surgery.

Biography

He was a professor at Busan Medical University, Go Sin Medical University, and National Research Foundation Researcher in the past. For 20 years, he has been studying on activating quiescent muscle & neural stem cells by mitochondrial dynamics. Modern conventional medicine focuses on incubating and reinjecting embryonic stem cells, but he recently confirmed that quiescent muscle & neural stem cells that exist in the body can be activated, proliferated, differentiated, and matured to regenerate quickly without any side effects. It was just mysterious to the amazingly fast regeneration ability of the human body.

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Non-pharmacologic management of orthostatic hypotension in inpatient rehabilitation: A quality improvement initiative

Orthostatic Hypotension (OH) is frequently encountered during inpatient rehabilitation following ischemic stroke and is associated with dizziness, instability, and therapy disruption during this critical stage of neurorecovery. On our neurorehabilitation unit, OH was identified as a common cause of missed therapy sessions. Although non-pharmacologic strategies such as compression garments, hydration, and physical counter-maneuvers are commonly recommended, implementation is often inconsistent. This project aimed to standardize OH management using low-risk, evidence supported interventions that could be initiated by nursing and therapy staff without requiring immediate physician involvement.

Conducted on a 20-bed inpatient neurorehabilitation unit in a large urban teaching hospital, this quality improvement initiative followed a literature-guided protocol developed by the team. Eligible participants were adults recovering from ischemic stroke who demonstrated OH, defined as >20 mmHg systolic or >10 mmHg diastolic drop without positional change accompanied by symptoms. Exclusion criteria included NPO status, modified diets that limited oral intake, lower extremity amputation, dialysis, or existing pharmacologic OH management.

A multidisciplinary team (nurses, physical therapists, occupational therapists, and rehabilitation physicians) developed a standardized OH protocol involving knee-high compression stockings, abdominal binders, oral bolus (480 mL water over 5 minutes), and a structured set of seated countermeasures including heel raises, toe raises, marching, long-arc quads, and gluteal sets. Staff were trained on eligibility screening, intervention steps, and given an online form with built-in safeguards. Data were collected from March 2024–March 2025.

31 OH episodes were documented and 22 met full protocol eligibility and received non-pharmacological intervention. Of these, 15 episodes (68%) were successfully managed using the protocol, allowing patients to resume either full therapy ($n=6$) or seated therapy ($n=9$) without

missed treatment time. Compression stockings were used most frequently (19/22), followed by abdominal binder (11/22), seated countermeasures (11/22), and finally oral bolus (6/22). 11 of the 31 total episodes required pharmacologic management. Staff reported this initiative established a unified, effective approach that empowered all team members to initiate treatment without requiring immediate physician involvement, reducing missed therapy sessions and supporting optimal participation in rehabilitation.

These findings suggest a standardized, multidisciplinary, non-pharmacologic protocol for OH is feasible, safe, and effective for supporting therapy participation among ischemic stroke patients in inpatient rehabilitation. Empowering nurses and therapists to initiate treatment improved timeliness for care and reduced therapy disruption. Future application of this protocol across additional settings and diverse patient populations may help address current gaps in evidence and promote wider use of low-risk interventions for OH. Reducing the incidence of OH has the potential to decrease fall risk and enhance safe mobility during stroke recovery.



Biography

Laura Steakin is a Registered Nurse with experience on the Neurological Rehabilitation Unit at Sinai Hospital of Baltimore. Mackenzie Weber is a Physical Therapist working on the Neurological Rehabilitation Unit at Sinai Hospital of Baltimore.



Mackenzie Weber is a Physical Therapist working on the Neurological Rehabilitation Unit at Sinai Hospital of Baltimore.



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Brain glucose hypometabolism is associated with neuropsychiatric impairment following traumatic brain injury

Background: The neurological and psychiatric sequelae of Traumatic Brain Injury (TBI) have become an increasing concern. However, the long-term effects of TBI on brain glucose metabolism, particularly in individuals more than a decade after injury, remain poorly understood. This study aimed to characterize alterations in brain glucose metabolism in patients with chronic TBI and to examine the relationship between regional metabolic abnormalities and neuropsychiatric impairment.

Methods: Twenty-one patients with TBI (mean±SD: 47.7±12.8 years), including 10 patients with single severe TBI (ssTBI) and 11 patients with repetitive mild TBI (rmTBI), and 15 healthy controls (42.2±14.8 years) were included. The mean interval between the head injury and assessment was 16.7±14.3 years in the ssTBI group, and the mean interval since the first head injury was 30.9±11.3 years in the rmTBI group. Brain glucose metabolism of the patients was assessed

using ^{18}F -Fluoro Deoxy Glucose (FDG) PET and MRI scans. Regions of Interest (ROIs) were defined in multiple areas in the neocortical Gray Matter (GM), White Matter (WM), and subcortical regions for each T1-weighted image. TBI patients also underwent several clinical evaluations including a diagnosis of the modified criteria for Traumatic Encephalopathy Syndrome (TES), and assessments of cognitive impairments using neuropsychological tests.

Results: TBI patients exhibited significantly lower ^{18}F -FDG Standardized Uptake Value Ratios (SUVRs) in the GM areas than age-matched HC subjects, particularly in the frontal GM, cerebellar GM, and the thalamus. Among TBI patients, whole GM ^{18}F -FDG SUVRs negatively correlated with BPRS scores, but positively correlated with scores for MMSE, ROCFT immediate recall, and ROCFT delayed recall. Furthermore, exploratory univariate analysis suggested lower ^{18}F -FDG SUVRs in the ssTBI group than in the rmTBI group in several regions. No significant difference in whole GM ^{18}F -FDG SUVRs were observed between the TES and non-TES groups.

Discussion: ^{18}F -FDG PET demonstrated reduced brain glucose metabolism in the chronic stages of TBI, indicating that metabolic abnormalities may persist for more than a decade after injury. Reduced brain glucose metabolism was associated with psychiatric symptoms and cognitive impairment but not with the clinical diagnosis of TES. These findings suggest that the effects of TBI on brain function may persist over the long term and are predominantly linked to neuropsychiatric symptoms and cognitive dysfunction.

Biography

Linna Sato is a student researcher from Hiroo Gakuen Senior High School, with a strong passion for neuroscience, psychology, and philosophy. Through her experience as a soccer player, Sato developed an interest in concussions and the consequences it may bring. Outside of her research as a trainee at the National Institutes of Quantum Science and Technology (QST), Sato also spreads her passion for neuroscience through her activities as the executive community manager at IYNA Japan.



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Early intervention for childhood developmental disorders guided by correct sensory input: An integration of neurodevelopmental theory and extended reality eye-tracking technology

Background and Objective: Early identification and intervention for childhood developmental disorders, particularly Autism Spectrum Disorder (ASD), depend on a deep understanding of sensory information processing mechanisms. Existing methods still have limitations in terms of ecological validity, objective quantification, and theoretical systematicity. This study aims to integrate two cutting-edge contributions: one proposing a technological system based on Extended Reality and Eye-Tracking (XREE-TT), and the other reconstructing sensory integration theory grounded in neurodevelopmental principles. The goal is to explore how their integration can form a closed loop for precision intervention, bridging theory and technology.

Theory and Methods: Both articles emphasize that correct sensory input is the foundational prerequisite for efficient brain processing and appropriate behavioral output. The second article reconstructs the four-tiered model of sensory integration development based on neurodevelopmental sequences (from bottom to top, from inside to outside), highlighting the decisive role of the vestibular system in gating sensory input. The first article, by integrating eye-tracking with Extended Reality (XR) and anchoring on visual processing (Gv) within the Cattell-Horn-Carroll (CHC) cognitive model, designs immersive and adaptive assessment and training tasks, enabling objective quantification and real-time feedback of visual attention.

Integration and Innovation: The two articles demonstrate strong complementarity: the sensory integration theory provides the XREE-TT system with a richer array of sensory dimensions (vestibular, tactile, proprioceptive) and a framework based on neurodevelopmental sequences. In turn, the XREE-TT system offers a high-tech means for visualizing, monitoring, and adaptively adjusting correct sensory input within sensory integration training. Their integration can form a synergistic system of theory guiding technology, technology supporting theory, incorporating multimodal sensory training into XR environments and using eye-tracking to monitor multisensory integration effects in real time.

Conclusion and Significance: This study proposes that integrating neurodevelopmentally-grounded sensory integration theory with extended reality eye-tracking technology can establish a closed loop of precision intervention covering assessment, training, and monitoring. This integrated approach opens new possibilities for the early identification and intervention of ASD and other developmental disorders, potentially advancing the rehabilitation of children with special needs from an experience-based practice toward a personalized, multimodal pathway.

Biography

Dr. Luo Xun is an Academician of the European Academy of Natural Sciences and a Professor at the American International Medical University, specializing in rehabilitation medicine, neurorehabilitation, and rehabilitation robotics. He is the Founder and Chief Scientist of Harobo Rehabilitation Robotics Co., Ltd., and President of the Shenzhen Jiali Rehabilitation Medicine Research Institute. Dr. Luo serves as Academic Master's Supervisor at Wenzhou Medical University and holds several academic and clinical appointments across leading medical institutions in China. He is also Chairman of the Academic Committee of the Guangdong-Hong Kong-Macao Greater Bay Area International Rehabilitation Forum and Chief Investigator in Fasciology at Shenzhen University. His research focuses on innovative rehabilitation technologies, fascial science, pediatric rehabilitation, and interdisciplinary approaches integrating engineering with clinical medicine.



Maria Teresa Gil Ogliastri

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Neurotango: The dance of social inclusion & brain connection

Neurotango is a therapeutic framework that integrates the principles of Dance Movement Therapy (DMT) with the rituals, interpersonal dynamics, and kinetic qualities of Argentine tango, aiming to enhance communication, social interaction, and psychophysical regulation in neurodiverse populations. DMT, as an innovative approach to mental health treatment, incorporates various social dances—such as tango, waltz, swing, and salsa—to promote processes of psychophysical integration. This study seeks to answer the following question: How does the use of tango within the DMT framework facilitate social interaction in individuals with disabilities?

The method is structured around six core components—movement, space, rhythm, contact, synchrony, and emotions—each of which is inherent to social tango practice. These elements support the creation of therapeutic experiences that stimulate sensory awareness, bodily consciousness, and readiness for relational engagement. The tango embrace, framed ethically as conscious and non-eroticized touch, enables experiences of support, boundaries, and tonic regulation, which are particularly relevant for individuals with communicational or motor challenges associated with intellectual, motor, or visual disabilities. Likewise, the predictable 2/4 rhythm and the need for coordinated partner movement act as psychomotor organizers that facilitate gait, joint attention, and emotional regulation.

The study population consists of young adults with intellectual disabilities, in some cases associated with motor, visual, or autism spectrum conditions. The chosen methodology is qualitative, appropriate for the study of social phenomena due to its flexibility and its capacity to integrate the researcher's subjectivity through narrative analysis of video recordings. Results indicate adaptive changes consistent with disability literature, including the

reutilization of capabilities, reconnection with the body, and expansion of relational patterns. From a neuroscientific perspective, the intervention is supported by findings on mirror neurons, kinesthetic empathy, and intersubjectivity, highlighting how imitation, mirroring, and interpersonal synchrony foster emotional connection—even in populations with significant communicational challenges. This approach aligns with the neurodiversity paradigm, which shifts the focus from deficit to capacity, recognizing the talents, rhythms, and unique modes of being that characterize neurodiverse individuals.

In sum, Neurotango constitutes an innovative therapeutic proposal that brings together the expressive potential of dance, the relational value of the embrace, contributions from neuroscience, and the principles of somatic psychotherapy. Its central aim is to transform social interaction, expand communicational possibilities, and strengthen the sense of belonging in neurodiverse individuals, offering a sensitive and effective pathway toward psychophysical well-being.

Keywords: Neurotango, Dance Movement Therapy, Neurodiversity, Mirror Neurons, Argentine Tango.

Biography

Maria Teresa Gil Ogliastri, Psychologist from the Central University of Venezuela, received an honorable mention for her thesis on the construction of affective bonds in young adults, which was validated at the University of Buenos Aires (UBA). Gil Ogliastri specialized in Dance Movement Therapy at the National University of the Arts, with a thesis titled: Contributions of Tango in the context of Dance Movement Therapy for facilitating social interaction in adults with intellectual, visual, and motor disabilities. Gil Ogliastri is the author of the university extension workshop and the book *Neurotango: A Dance Movement Therapy/Tango Method for People with Neurodiversity* (2018, Editorial Authors of Argentina). Neurotango is recognized by the Government of the City of Buenos Aires as a Project of Cultural Interest and by the Metropolitan Fund for Culture, Arts, and Sciences of the City of Buenos Aires, Argentina.



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The CI-HI & CICR-HI: Development and validation of patient and caregiver-reported outcome measures for Alzheimer's disease, mild cognitive impairment, and dementia

Background: As new therapies are developed across the Alzheimer's Disease (AD) continuum, it is critical that patient and caregiver-reported changes in symptomatic health are incorporated into clinical trials. The US FDA encourages the use of patient and observer-reported outcome measures to evaluate the therapeutic benefits of potential new treatments in AD, Mild Cognitive Impairment (MCI), and dementia. Here, we outline the development and validation of The Cognitive Impairment-Health Index (CI-HI) and The Cognitive Impairment Caregiver Reported-Health Index (CICR-HI), two novel instruments developed and validated using large-scale data from patients and caregivers, which measure multifactorial disease burden in the symptomatic domains of greatest importance to patients.

Methods: We conducted qualitative interviews and two large cross-sectional studies involving individuals with AD, MCI, or dementia and caregivers of individuals with AD, MCI, or dementia to ascertain the prevalence and impact of symptoms. We selected questions for the CI-HI and CICR-HI based on their relevance and potential responsiveness to therapeutic

intervention. We performed an initial analysis of internal consistency, beta testing, test-retest reliability assessments, and known groups testing to generate instrument subscales and to optimize instrument clarity, usability, meaningfulness, responsiveness, and reliability. Lastly, we performed a final internal consistency and area under the curve analyses of the CI-HI and CICR-HI.

Results: Fifteen individuals with AD, MCI, or dementia and fifteen caregivers of individuals with AD, MCI, or dementia participated in qualitative interviews to determine the most prevalent and impactful symptoms. The symptoms of greatest prevalence and impact were used to construct cross-sectional surveys, completed by 104 individuals with AD, MCI, or dementia and 329 caregivers of individuals with AD, MCI or dementia. We found through validation testing that the CI-HI and CICR-HI are highly relevant, reliable, and capable of distinguishing between groups based upon varied demographic and clinical characteristics. The CI-HI encompasses nine independently validated subscales: (1) Memory; (2) Cognition; (3) Fatigue; (4) Sleep or Daytime Sleepiness; (5) Social Satisfaction; (6) Communication; (7) Emotional Health; (8) Activity Participation; and (9) Pain. The CICR-HI captures observable aspects of disease burden in AD, MCI, and dementia and mirrors the subscale domains of the CI-HI except for the Pain subscale, which is not included in the CICR-HI. Collectively, these instruments measure multifactorial disease burden across the cognitive impairment spectrum.

Conclusion: The Cognitive Impairment-Health Indices are validated and designed to quantify changes in the multifactorial symptomatic burden in individuals with AD, MCI, and dementia over time and in response to therapeutic intervention during clinical trials.

Biography

Matthew Rathbun is a Human Subject Research Specialist at the University of Rochester Center for Health + Technology (CHeT) in Rochester, New York. His research focuses on developing and validating disease-specific patient- and caregiver-reported outcome measures, primarily in rare and neurological disease populations. CHeT's Outcomes Division has developed over 250 disease-specific outcome measures, which have been used in industry, academic, and foundation trials worldwide and demonstrate strong sensitivity to therapeutic change and relevance to patient populations. Matthew is passionate about ensuring that patient and caregiver voices are meaningfully integrated into the evaluation of emerging therapies.



Meher Garg*, Veer Ramesh Garg

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Seizure activity suppression by antioxidants in a *Drosophila* model of epilepsy

Background: Epilepsy affects over 50 million people worldwide. Approximately one-third of patients are resistant to current treatments with many experiencing adverse side effects. Mutations in the prickle gene, particularly the prickle-spiny-legs (*pksple*) isoform in *Drosophila*, produce spontaneous seizures and ataxia similar to human PRICKLE-associated epilepsy. Because PRICKLE is evolutionarily conserved, *Drosophila* serves as a powerful model for studying seizure mechanisms and therapeutic strategies.

Previous research has identified a cascade linking oxidative stress to seizure activity. Oxidative stress generates Reactive Oxygen Species (ROS), which damage cellular structures, including mitochondria, and trigger a glial-mediated Innate Immune Response (IIR). This response promotes neuronal death and increases seizure frequency. Notably, oxidative stress precedes IIR activation, suggesting that targeting ROS may interrupt this pathological pathway. Antioxidants (AOs), which neutralize ROS, may therefore reduce seizure susceptibility.

Objective: This study systematically evaluated nine antioxidants to determine their efficacy in suppressing seizures in *pksple* mutant flies. As oxidative stress is most pronounced during larval stages, treatments were administered across three developmental windows-larval, adult, and combined larval+adult-to assess stage-specific effects on adult seizure outcomes.

Materials and Methods: *pksple* flies were raised on standard cornmeal medium supplemented with one of nine antioxidants from embryonic stages through 15 days post-eclosion. Compounds included trans-resveratrol, ibuprofen, licofelone, gallic acid, valdecoxib, allyl disulfide, genistein, curcumin, and N-acetyl cysteine (NAC).

Spontaneous seizure activity was assessed using a blinded videographic assay. Individual flies were recorded and analyzed for seizure events. Experiments used 14–16-day-old males, with ≥ 2 biological replicates and 8–10 flies per replicate. Statistical analysis was performed using Student's t-test.

Results: Allyl disulfide, ibuprofen, curcumin, and trans-resveratrol significantly reduced seizure penetrance in *pksp/e* mutants. Gallic acid showed a strong trend toward significance ($p \approx 0.052$), suggesting potential efficacy. In contrast, licofelone, valdecoxib, genistein, and NAC did not significantly reduce seizures. Notably, NAC failed to suppress seizures across all developmental treatment windows, despite its established antioxidant properties.

Discussion: These findings demonstrate that select antioxidants can effectively suppress seizures in a genetic model of epilepsy, supporting oxidative stress as a key driver of seizure pathology. The efficacy of allyl disulfide, ibuprofen, curcumin, and trans-resveratrol highlights their potential as therapeutic candidates, particularly for patients who do not respond to conventional anti-epileptic drugs. The lack of effect from other antioxidants, including NAC, suggests that not all ROS-targeting compounds are equally effective, emphasizing the importance of compound-specific mechanisms.

Biography

Meher Garg is a student researcher and community leader dedicated to advancing healthcare through innovation and service. Garg is a Regeneron ISEF Finalist, JSHS Finalist, and recipient of the President's Volunteer Service Gold Award and Congressional Award. Garg's research spans glioblastoma stem cell differentiation, Alzheimer's disease, and neurobiological responses to hypoxia, conducted at the University of Iowa, University of Michigan, and Rutgers University, with resulting publications and national presentations. Garg founded Melody Therapy, bringing live music to hospitals and memory care centers. Through interdisciplinary research, leadership, and clinical service, Garg aims to bridge science and compassion to improve patient outcomes.



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Real-world safety of lecanemab and donanemab in Alzheimer's disease: A single-center retrospective cohort study

Background: Anti-amyloid monoclonal antibodies, including lecanemab and donanemab, represent an emerging disease-modifying treatment approach for early Alzheimer's disease. However, treatment is associated with Amyloid-Related Imaging Abnormalities (ARIA), including ARIA with Edema (ARIA-E) and Hemorrhage (ARIA-H), with APOE ϵ 4 carrier status recognized as an important potential risk factor. This study evaluated real-world adverse events, specifically ARIA associated with lecanemab and donanemab and explored the relationship between APOE ϵ 4 status and ARIA.

Methods: We conducted a single-center retrospective cohort study of patients with confirmed Alzheimer's disease treated with lecanemab or donanemab. Electronic health records were reviewed for demographics, Alzheimer's diagnostic testing, APOE genotype, baseline Mini-Mental State Examination (MMSE) score, imaging findings, ARIA, and other documented adverse events. ARIA rates were compared using Fisher's exact tests.

Results: Seventy-six patients were included: 26 received lecanemab and 50 received donanemab. Mean age was 72 and 76 years, and mean baseline MMSE was 26 and 24, respectively. ARIA occurred in 5/26 (19.2%) lecanemab-treated patients compared with 3/50 (6.0%) donanemab-treated patients ($p=0.114$). Four lecanemab-associated cases were ARIA-E and one was ARIA-H; all three donanemab-associated cases were ARIA-E. All eight ARIA cases in APOE ϵ 4 carriers. In the lecanemab cohort, ARIA occurred in 5/17 (29.4%) carriers versus 0/9 noncarriers ($p=0.129$); three ARIA cases occurred in ϵ 3/ ϵ 4 heterozygotes and two in ϵ 4/ ϵ 4 homozygotes. In the donanemab cohort, ARIA occurred in 3/27 (11.1%) carriers versus 0/23 noncarriers ($p=0.240$); all three ARIA cases occurred in ϵ 3/ ϵ 4 heterozygotes. Other adverse

events occurred in 6/26 (23%) and 11/50 (22%) of lecanemab- and donanemab-treated patients, respectively with headache and flu-like symptoms among the most documented complaints.

Conclusion: In this real-world cohort, both lecanemab and donanemab demonstrated safety profiles generally consistent with those reported in clinical trials, with no major treatment-related complications observed. ARIA was numerically more frequent with lecanemab than donanemab, although the difference was not statistically significant. Notably, the observed incidence of ARIA with donanemab was lower than rates reported in clinical trials, supporting its tolerability in routine clinical practice. All observed ARIA events occurred in APOE ϵ 4 carriers, reinforcing the importance of APOE status in risk assessment and treatment monitoring. These findings support the real-world use of anti-amyloid therapies with appropriate patient selection and surveillance; however, larger multicenter studies are needed to confirm comparative ARIA risk and further characterize the influence of APOE genotype.

Biography

Michelle Wong, B.S., is a fourth-year osteopathic medical student (OMS-IV) at Edward Via College of Osteopathic Medicine (VCOM) – Louisiana. Wong's academic and clinical interests include neurocognitive disorders and epilepsy. Wong's current research focuses on the safety of anti-amyloid monoclonal antibody therapies, including lecanemab and donanemab, with particular attention to amyloid-related imaging abnormalities and factors associated with treatment risk. Wong plans to pursue residency training in neurology and hopes to continue contributing to clinical research throughout a medical career.



Mustafa A. Khan, M.D.

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Prince transform: A wave-mechanical framework for real-time EEG analysis and early seizure prediction using chirp and drift detection

Standard EEG analysis relies on the Fourier Transform, which assumes additive superposition of stationary sinusoids on the Gaussian complex plane and derives energy post-hoc via Parseval's theorem. This approach struggles with the multiplicative, non-stationary, and energetically physical nature of neural oscillations.

We derive the Prince Transform directly from de Broglie's relation $\lambda p = h$ using multiplicative superposition on the Euler Complex Plane. The transform simultaneously extracts spatial wavelength structure, temporal frequency content, and physical energy from EEG signals via the natural logarithm. Its temporal derivative naturally yields two key signatures of homeostatic breakdown: chirp (instantaneous frequency change $d\omega/d\tau$) and drift (accumulated frequency shift).

These quantities emerge as the coupled response between the linear propagation and nonlinear self-binding terms in the Prince Wave Equation. In pre-ictal states, sustained chirp and systematic drift appear minutes before classical seizure patterns, providing a physically grounded early warning criterion ($\partial\omega/\partial t \cdot t > \omega T$). The framework also enables natural, energy-based color mapping for intuitive clinical visualization.

When integrated with machine learning models, the Prince Transform supplies physics-informed features that enhance early seizure detection, artifact rejection, and differentiation of true pre-ictal dynamics. This represents a shift from purely statistical pattern recognition to a wave-ontological understanding of brain activity, with strong potential for real-time implementation in epilepsy monitoring and neurocritical care.

Keywords: Prince Transform, EEG, Chirp, Drift, Seizure Prediction, Wave Ontology, Physics-Informed AI.

Biography

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



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Beyond plaques: 10-nanometer-scale patho connect omics of Alzheimer's disease

Alzheimer's neuropathology is commonly described by lesion burden, yet cognitive decline emerges from the disruption of neural circuits. Amyloid- β ($A\beta$) plaques are associated with local dendritic spine loss, excitatory synapse depletion, impaired axonal transport, and dystrophic neurites. In the 5xFAD model, plaque-associated axonal spheroids (PAAS) can impede action-potential propagation and disrupt long-range connectivity. Studies in mouse and human Alzheimer's tissue further implicate endolysosomal, cytoskeletal, lipid-transport, and mTOR-associated pathways in spheroid pathology. These findings identify the plaque-PAAS microenvironment as a critical unit of disease, but one that remains difficult to interrogate: Fluorescence Microscopy (FM) provides molecular specificity without continuous nanoscale architecture, whereas Electron Microscopy (EM) reveals ultrastructure but is difficult to molecularly multiplex and scale across translational cohorts. To address this gap, Panluminate developed, to our knowledge, the first integrated, scalable, all-optical pathoconnectomics platform that co-registers continuous 3D ultrastructure with multiplexed molecular information in a single tissue volume. Panluminate's proprietary expansion chemistry and dense pan-protein labeling reveal neurites, synapses, organelles, and neuropil, while multiplexed immunolabeling identifies the cell types, disease proteins, and signaling compartments embedded within that architecture. The underlying method achieves approximately 14 ± 3 nm effective lateral resolution under 20-fold expansion conditions. Automated sample processing and high-NA volumetric light-sheet imaging extend this capability beyond selected microscopic fields toward reproducible, cohort-scale studies.

In collaboration with translational Alzheimer's researchers, Panluminate is applying this workflow to age-matched 5xFAD and control mouse brain tissue. Initial 8-plex datasets resolve $A\beta$ deposits together with NF-H-positive axons, MBP-positive myelin, endolysosomal

compartments, nuclear architecture, and pan-protein neuropil. We are augmenting a panel of 35 validated brain-tissue antibodies with 15 Alzheimer's-focused targets to quantify plaque-distance-dependent changes in PAAS burden, axonal caliber and continuity, organelle accumulation, myelin organization, and synaptic architecture. In parallel, we are adapting the workflow to postmortem human brain tissue to establish a translational bridge between animal models and human neuropathology.

Because disease-specific molecular panels can be exchanged without sacrificing ultrastructural context, the same platform can be applied to other neurodegenerative and neurological disorders in which protein pathology, cellular identity, and circuit remodeling must be measured together. Panluminate is now extending this new pathoconnectomics paradigm through collaborations with neuropathologists, translational researchers, and therapeutic-development teams.

Biography

Ons M'Saad, PhD, is Founder and CEO of panluminate, a Yale spinout building scalable optical molecular connectomics. M'Saad earned a PhD in Biomedical Engineering from Yale and a BS in Biological Engineering from MIT. M'Saad's research developed pan-Expansion Microscopy, an all-optical method for visualizing molecular identity within nanoscale brain ultrastructure, published in *Nature Biotechnology*. M'Saad now leads the integration of tissue chemistry, volumetric imaging, automation, and AI analysis to create molecularly annotated brain maps for neurodegenerative disease research, CNS drug discovery, and next-generation brain data.



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Steroid-responsive encephalopathy associated with autoimmune thyroiditis presenting as acute psychosis: A diagnostic pitfall

Steroid-Responsive Encephalopathy Associated With Autoimmune Thyroiditis (SREAT) is a rare autoimmune encephalopathy characterized by heterogeneous neuropsychiatric manifestations. We describe the case of a 59-year-old woman with a history of anxiety and severe alcohol use disorder who presented with acute psychosis, marked behavioral disorganization, and cognitive impairment initially attributed to alcohol withdrawal, metabolic disturbance, or primary psychiatric illness. Despite extensive evaluation, including negative infectious, toxicologic, metabolic, structural, and paraneoplastic workups, no clear etiology was identified. Additional findings, including a reactive HIV screening assay and elevated B12 levels, further complicated the diagnostic process. Thyroid studies revealed markedly elevated anti-thyroid peroxidase and anti-thyroglobulin antibodies with fluctuating thyroid function, raising suspicion for SREAT. Following initiation of high-dose intravenous corticosteroids, the patient demonstrated rapid and marked improvement in mental status within 48 hours, supporting an immune-mediated process. Subsequent cerebrospinal fluid analysis, electroencephalography, and brain imaging remained unremarkable. This case highlights the diagnostic challenges of SREAT, particularly in patients with comorbid psychiatric and substance use disorders and underscores the importance of considering autoimmune encephalopathy in cases of unexplained neuropsychiatric decline. Early recognition and timely immunosuppressive therapy may lead to significant clinical recovery and prevent prolonged morbidity.

Biography

Oren G. Nedjar is a medical student at Nova Southeastern University Dr. Kiran C. Patel College of Osteopathic Medicine whose academic work focuses on complex diagnostic medicine, neuroimmunology, and vascular-neurologic disease. He has authored peer-reviewed publications and presented nationally, including at the American Academy of Neurology. His research portfolio includes autoimmune encephalopathy, cerebral small vessel disease, and translational laboratory investigation. He is particularly interested in diagnostic complexity and multisystem pathology. He intends to pursue residency training in Internal Medicine with long-term involvement in academic medicine and clinical research.



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Therapeutic approaches for prion diseases: A systematic review

Background: Prionic diseases, such as Transmissible Spongiform Encephalopathies (TSEs) and Creutzfeldt-Jakob Disease (CJD), are rare and fatal neurodegenerative disorders caused by the abnormal buildup of misfolded prion protein (PrP^{Sc}). Currently there are no efficient therapies available when it comes to prionic diseases, which highlights the importance of evaluating the impact and possible repurpose of many available drugs against these Encephalopathies.

Objective: This systematic review intends to compare pharmaceutical possibilities using medication available (such as Flupirtine, Doxycycline, Quinacrine, Monoclonal antibody PRN100 and Pentosan Polysulfate).

Methodology: The research consisted of searching through Clinical trials in PUBMED, SCIELO, Cochrane and BVS databases.

Results: Initially were found 12395 articles, which resulted in 8 articles after the application of inclusion and exclusion criteria.

Discussion: Flupirtine demonstrated relevant cognitive benefits (improving ADAS-Cog and MMSE scores), while monoclonal antibody PRN100 showed potential in modulating disease progression, despite inevitable advancement. Doxycycline showed a significant increase in survival in one of the studies. Quinacrine had inconsistent results, and Pentosan Polysulfate Sodium was associated with significant safety concerns. Common limitations were small samples, heterogeneous disease stages and the lack of early biomarkers.

Conclusion: Flupirtine and PRN100 emerge as the most promising approaches for managing CJD. Future research must prioritize early intervention, randomized controlled trials along with possible combined therapy.

Keywords: Prions, Transmissible Spongiform Encephalopathies, Creutzfeldt-Jakob disease, Anti-prion therapy, Clinical trials.

Biography

Rafaela De Marchi Gherini Mingione is a medical student at Universidade Santo Amaro with a deep interest in the challenges of neurology and neurosurgery, especially when little is known. Mingione's undergraduate research focused entirely on therapeutic strategies for prion diseases: first a systematic review, then a critical analysis of the barriers to clinical translation. These studies shaped Mingione's commitment to neurotherapeutics. Mingione's global journey includes a Portuguese-German bilingual high school, six months in Texas (Eastlake High School), and five weeks in Germany (Franziskaner Gymnasium Kreuzburg). Each experience reinforced Mingione's curiosity and resilience. Mingione is eager to contribute to meaningful discussions on neurodegenerative disorders.



Rahul Hajare

Sandip University Nashik, India

Neurobiology of the gut brain axis: Translating microbiome science into mental health care

The gut brain axis represents a complex, bidirectional communication network integrating the enteric nervous system and the central nervous system through neural, endocrine, immune, and metabolic pathways. Beyond its anatomical connections, this dynamic system operates via the autonomic nervous system, the Hypothalamic–Pituitary–Adrenal (HPA) axis, neuroimmune signaling, microbial metabolites, and circulating humoral factors. Within this framework, the gut microbiota functions as a critical regulatory interface. Through modulation of vagal nerve signaling, neurotransmitter production (including serotonin, dopamine, and gamma-aminobutyric acid), short-chain fatty acid synthesis, immune activation, and inflammatory cytokine release, intestinal microbes actively influence brain function. Simultaneously, central stress responses mediated by the HPA axis alter gut permeability, motility, microbial composition, and mucosal immunity. This reciprocal interaction has significant implications for mood regulation, cognitive processes, stress responsiveness, and the pathophysiology of psychiatric and neurodevelopmental disorders. Emerging evidence suggests that microbial dysbiosis may contribute to conditions such as depression, anxiety, and stress-related disorders through mechanisms involving neuroinflammation, altered neurotransmission, and impaired barrier integrity. Understanding the gut–brain axis thus represents a paradigm shift in mental health research, offering new opportunities for preventive strategies, nutritional interventions, and microbiota-targeted therapeutics. Continued interdisciplinary and international collaboration will be essential to translate microbiome science into equitable, evidence-based mental health care worldwide.

Biography

Dr. Rahul Hajare is an Indian scholar and a student of the esteemed scientist Dr. Ramesh S. Paranjape. Hajare's academic and research training has provided international exposure, including scientific engagement in the United States and the United Kingdom. Hajare is affiliated with the Lab Root Laboratory, the Biosafety Laboratory, and the Bioinformatics and Protein Database, where Hajare contributes to research in biosciences and data-driven biological analysis. Hajare is fluent in English.



Rodica Elena Petrea, M.D

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The alive Cardiac Voodoo: The heart-brain syndrome beyond stroke

Aim: To discuss life-threatening brain-heart comorbid syndrome in acute cerebral stress conditions.

Methods/Design: Case Reports and related literature update. Case 1: A 72-year-old female with acute confusion, amnesia, and generalized fatigue was admitted for acute neurological evaluation. MRI brain showed no acute pathology, and the diagnosis of Transient Global Amnesia (TGA) was made. Transthoracic Echocardiography (TTE) found 20% LVEF and hypokinesia. Case 2: A 48-year-old female was brought to the hospital after a reported Generalized Tonic-Clonic (GTC) seizure activity >30 minutes at home. A recurring GTC seizure was witnessed in the ED while the patient remained unconscious. Status Epilepticus (SE) treatment and intubation were initiated. Brain MRI was diagnostic of Posterior Reversible Encephalopathy Syndrome (PRES) and a small, ill-defined left parietal cortical mass. TTE revealed LVEF of 10-15%, LV apical thrombus, and hypokinesia. In both cases, a diagnosis of Takotsubo Cardiomyopathy (TC) was made. Beyond acute neurological care, both patients underwent acute anticoagulation and cardiomyopathy treatment and were discharged home with follow-up care.

Results: The life-threatening brain-heart comorbid syndrome with the maximal form of stress TC was first reported in 1944 as sudden death, later proved in association with cerebral edema, brain neoplasms, and acute strokes. Henceforth, it has also been recognized as concomitant with other acute cerebral stress conditions like TGA, SE, and PRES.

Discussion/Conclusions: I highlight the importance of recalling the cardiac voodoo heart-brain syndrome in acute cerebral stress conditions. The cardiac voodoo is a condition with high morbidity and mortality due mostly to catecholamine surge and cardiac failure, stressing the early need for EKG and TTE evaluation in acute neurological conditions. This is even more apparent in the presence of hypotension and tachycardia and absence of other typical cardiac symptoms. The comorbid TC with acute neurological conditions has major consequent therapeutic decisions, from preventing or limiting the catecholamine surge and stunt organ damage to intensive care treatment, continuous Holter monitoring, preferred use of beta and calcium-channel blockers while avoiding drugs that prolong QT interval, and eventually cardiac pacing, to mitigate the high-risk morbidity and mortality of such complex potentially fatal cases.

Biography

Dr. Rodica Elena Petrea is a Romanian-born international neurologist with expertise in stroke and vascular dementia, a career that spans mostly in the United States of America. At present, she lives and works in the USA. She achieved her medical doctor (MD) diploma from Carol Davila University of Medicine and Pharmacy in Bucharest, Romania (1995). She completed her neurology training both in Bucharest, Romania in 2000 and in U.S., in Boston, MA in 2008 before she was certified in neurology by both the Romanian Board of Neurology in 2000 and the American Board of Neurology and Psychiatry in 2008 and 2012, when she became a Diplomate of the American Board of Neurology and Psychiatry. She was a Pont-Neuf Scholar in Paris, France at Lariboisiere Hospital in 2000, and carried on research with the Framingham Heart Study in Boston, MA (2006-2008; 2019-2022).



Saul Beaumont

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Prescribing joy: The importance of integrating memory cafés into dementia care

Current treatments for dementia remain slow acting, limited, and often unable to meaningfully change the quality of life for patients and caregivers. Families living with dementia frequently endure a prolonged period of decline that can last many years, marked by progressive cognitive and functional loss, social isolation for both the patient and family members, and an overwhelming 24/7 caregiving burden. While clinicians focus appropriately on diagnosis, monitoring, and pharmacologic management, one essential element of care is often overlooked: The intentional creation of joy for the patient.

Memory cafés offer a simple, scalable, no cost intervention that brings joy, connection, and emotional uplift to the vast majority of dementia patients while providing respite and support for caregivers (1). These cafés, typically sponsored by towns and community organizations, provide a safe, stigma free environment where patients and caregivers share lunch, entertainment, conversation, and especially music. Music based engagement frequently elicits smiles, laughter, singing, and even dancing - sometimes from individuals who have lost speech, mobility, or independence. These moments of joy are not trivial; they are clinically meaningful, improving mood, reducing behavioral distress, and strengthening caregiver resilience.

Despite their benefits, memory cafes are heavily underutilized: caregivers are often unaware of their existence and/or avoid them due to stigma or uncertainty. and present estimates indicate that <1% of patients with dementia have attended a memory café (1). This is despite the existence of approximately 500-600 registered cafes in the U.S. (2). This barrier can be eliminated when neurologists prescribe memory cafés as part of a comprehensive dementia

care plan. A clinician's recommendation provides validation, reduces stigma, and encourages participation. Many neurologists are unaware of memory cafés or their impact, and this presentation aims to close that gap.

Drawing on the lived experience as a 24/7 caregiver and longstanding member of the local council of aging board who attended an average of five cafés per week, this talk illustrates how memory cafés can transform the daily life for a dementia patient — replacing hours of isolation with moments of joy, engagement, and human connection. This is a no downside, high impact intervention that costs nothing, is widely available, and can be immediately integrated into clinical practice.

Neurologists have the opportunity to improve quality of life today, even as research continues toward better treatments tomorrow. Prescribing memory cafés is a simple, powerful step toward bringing joy to families living with this brutal disease. This work is offered with the hope of helping as many families as possible, to optimize the quality of life for those burdened with dementia and to promote the highest possible care for our patients.

Biography

A retired aerospace program manager and former full time, at home caregiver for his wife for nearly six years. He served as Chair of the Natick Council on Aging Board last year and is now contributing as a caregiver representative to the Massachusetts Advisory Council on Alzheimer's Disease and All Other Dementias.



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

Biomarkers enable pre-early diagnosis, guide targeted therapy and monitor the activity and therapeutic responses across the diseases. 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 to illustrate the evolution of MS. Anti-MBP autoAbs from MS patients and mice with EAE 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 act as a specific inducer of EAE and to be attacked by the MBP-targeted Ab-proteases in MS patients with the most severe (progradient) clinical courses. The other ones whilst being less immunogenic happened to be EAE inducers very rare but were shown to be attacked by Ab-proteases in MS patients with moderate (remission-type) 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 being challenged! Of tremendous value in this sense are Ab-proteases directly affecting the physiologic remodelling of tissues with multilevel architectonics (for instance, myelin), whilst securing the requests and standards of regeneration and remyelination.

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 progression 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, he was a senior Researcher, Koltzov Inst of Developmental Biology. From 1989 through 1995, he 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). Dr. Suchkov has been trained at: NIH; Wills Eye Hospital, PA, USA; Univ of Florida in Gainesville; UCSF, S-F, CA, USA; Johns Hopkins University, Baltimore, MD, USA. He was an Exe Secretary-in-Chief of the Editorial Board, Biomedical Science, an international journal published jointly by the USSR Academy of Sciences and the Royal Society of Chemistry, UK. At present, Dr. Sergey Suchkov is a Professor of the Dept for Clinical Immunology of the Russian University of Medicine, Moscow, Russia and member of the Russian Academy of Natural Sciences, Moscow, Russia. Suchkov is a member of the: New York Academy of Sciences, USA; American Chemical Society (ACS), USA; American Heart Association (AHA), USA; 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.

Shalini Dandamudi

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Automated detection of normal-pressure hydrocephalus using DINOv2 vision transformer and DETR detection transformer technologies on CT imaging

Background: Normal Pressure Hydrocephalus (NPH) is a neurological condition characterized by the enlargement of the lateral ventricles in the brain despite normal cerebrospinal fluid pressure. Although this condition is treatable, it is often underdiagnosed due to its similarity to other conditions that resemble it. NPH is differentiated from other similar conditions using the pattern by which the lateral ventricles enlarge, which can be determined by radiological markers such as the Evans Index and Callosal Angle. These measurements are calculated using images of the brain, primarily CT scans. Although these measurements provide the data needed to diagnose NPH, they are not consistently calculated during routine clinical evaluation, contributing to delayed or missed diagnosis. This study aims to create a Machine Learning based automated tool that is able to accurately calculate these radiological markers, and assist primary care physicians in diagnosing NPH as efficiently and accurately as possible.

Methods: This study developed a transformer-based machine learning platform to automatically detect NPH-associated radiological markers from CT imaging. 1,000 CT slices (500 axial and 500 coronal) from open-access datasets were preprocessed, and the coronal images were manually annotated to train a DINOv2 Vision Transformer and Detection Transformer (DETR) to identify and segment the lateral ventricles. Using the detected ventricular boundaries, code was then developed to teach the model to automatically calculate the Evans Index and Callosal Angle, converting imaging data into quantitative diagnostic measurements. To evaluate performance, 88 axial and 88 coronal CT images ranging from normal to hydrocephalus were independently measured by a radiologist to calculate Evans Index and Callosal Angle using established clinical thresholds. The same images were then processed through the ML system, which automatically calculated these measurements and generated NPH predictions. The model's results were compared with the radiologist's measurements, and accuracy, sensitivity, specificity, false positive rate, and false negative rate were calculated to assess model performance.

Results: The model demonstrated strong diagnostic performance, achieving 92.05% accuracy using Evans Index measurements and 93.18% accuracy using Callosal Angle measurements, with AUC values of 0.965 and 0.896, respectively. These results showed strong agreement with manual neuroradiologist measurements, indicating that transformer-based models can reliably quantify radiological markers associated with hydrocephalus.

Conclusion: This project represents a novel application of Vision Transformers and Detection Transformers to calculate quantitative radiological measurements directly from CT imaging, an approach that has not previously been used to automate NPH marker detection. By enabling automated identification of critical diagnostic markers, this system has the potential to support physicians in recognizing normal-pressure hydrocephalus earlier and more consistently. Not only can this be a viable tool that primary care physicians use to speed the diagnosis process, but also be a publicly available tool that can be used free of cost.

Biography

Shalini Dandamudi is a freshman at Stockdale High School in Bakersfield, California. Ever since she was young, Dandamudi has been fascinated by the medical field, and recently discovered a true passion for using emerging technologies to solve real problems in medicine. Dandamudi is especially interested in how artificial intelligence can help physicians diagnose neurological conditions faster and more accurately. Dandamudi's research focuses on developing AI systems that analyze medical imaging to detect neurological disorders. Dandamudi is passionate about the intersection of neuroscience, technology, and healthcare, and hopes to pursue a future career creating innovative tools that improve patient diagnosis and care.



Sidharth Patsamatla

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Advancing neuromuscular disorder diagnosis and adaptive electroceutical therapy through real-time electromyography and hybrid deep learning

Neuromuscular disorders are a class of conditions that deteriorate the nerves controlling voluntary skeletal muscle, contributing to progressive motor dysfunction, losses in mobility and coordination, and muscle atrophy. According to the National Institutes of Health, it is estimated that neuromuscular disorders collectively affect every 1 in 3,500 people. Current clinical diagnostic approaches involve nerve conduction studies and needle electromyography, which are invasive and have limited sensitivity in detecting early neuromuscular changes. Similarly, clinical therapeutic approaches, including physiotherapy, primarily manage symptoms and lack the ability to adapt to individual patient-specific physiology. This research presents a software framework that improves the management of neuromuscular disorders through biophysical modeling and biomedical signal processing algorithms. Surface electromyography signals are preprocessed to capture high-dimensional spatiotemporal features representative of time-series neuromuscular activity. A dual-stage artificial intelligence pipeline integrating a Support Vector Machine (SVM) and a hybrid Convolutional Neural Network—Long Short-Term Memory Network (CNN-LSTM) was developed. The SVM is leveraged for low-latency muscle activation state detection, while the hybrid CNN-LSTM is employed for classifying spatiotemporal patterns associated with neuromuscular disorders. In parallel, a computational implementation of the Hodgkin-Huxley conductance model was engineered to simulate motor neuron and muscle dynamics under varying electrical stimulation parameters. The proposed framework was evaluated using a standard set of machine learning performance metrics. The SVM achieved a 94.4% classification accuracy with a 20 ms inference latency, while the hybrid CNN-LSTM achieved a 96.1% diagnostic accuracy (ROC-AUC=0.97), demonstrating the implications of deep learning-driven computational neuroengineering to facilitate neuromuscular disorder diagnosis and adaptive functional electrical stimulation.

Biography

Sidharth Patsamatla is a rising high school senior at West Shore Jr/Sr High School with broad research interests in computational neuroscience, neurotechnology, human-computer interaction, and artificial intelligence. Patsamatla is passionate about developing intelligent systems that improve the robustness and accessibility of neurological healthcare. Patsamatla's research integrates machine learning, computational signal processing, and embedded systems to address challenges in neurological disease diagnosis and neurorehabilitation. Through research and entrepreneurship, Patsamatla continues to translate emerging technologies into practical clinical solutions that enhance global patient outcomes and quality of life.



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Network-based biomarkers and state transitions in epilepsy: Clinical and translational implications

Epilepsy is increasingly recognized as a disorder of large-scale brain network dysfunction rather than a purely focal pathology. Advances in Network Neuroscience have revealed reproducible alterations in functional connectivity, graph topology, and network dynamics across epileptic syndromes.

This study presents a translational framework linking network alterations to clinically observable seizure phases. We propose a three-state model consisting of a preictal phase of network instability, an ictal phase of hypersynchronization, and a postictal phase of network suppression and fragmentation. These dynamic transitions are supported by EEG-based findings, including preictal increases in synchronization and postictal suppression patterns.

From a clinical perspective, network-based biomarkers such as functional connectivity measures, graph metrics, and EEG synchronization indices may improve seizure prediction and patient stratification. Furthermore, therapeutic strategies including neuromodulation (e.g., DBS, TMS, VNS) and surgical planning can be reinterpreted as interventions targeting pathological network hubs and restoring normal network dynamics.

By integrating theoretical and clinical insights, this approach provides a practical framework for developing personalized, network-guided treatment strategies in epilepsy.

Biography

Eliaçık is a researcher focused on epilepsy and network neuroscience. Eliaçık's work conceptualizes epilepsy as a disorder of large-scale brain network dynamics rather than

a purely focal condition. Eliaçık investigates functional connectivity, graph theory, and network-based biomarkers to improve seizure prediction and clinical translation. Eliaçık actively contributes to international congresses and aims to bridge advanced computational approaches with practical clinical applications. Eliaçık's research adopts an interdisciplinary, innovative, and patient-centered perspective.



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Nervous system effects in chemicals with immediately dangerous to life or health values

Introduction: Immediately Dangerous to Life or Health (IDLH) values are intended to characterize the risk associated with acute inhalational exposures in the workplace. A main goal of IDLH values is to ensure that a worker can escape from a contaminated environment in the event of failure of protective equipment and to indicate a maximum level above which only a highly reliable breathing apparatus, providing maximum protection, is permitted (Current Intelligence Bulletin 66: Derivation of Immediately Dangerous to Life or Health (IDLH) Values). The purpose of this initial analysis is to examine chemicals with IDLH values and nervous system effects.

Methods: This initial exploratory analysis examined 384 chemicals with previously established IDLH values based on animal and human data, available on the National Institute for Occupational Safety and Health (NIOSH) website at Immediately Dangerous to Life or Health | NIOSH | CDC. IDLH information was compiled using web scraping and keyword-based text mining methods. In parsing these data, three sections of each page were searched: Basis for original (SCP) IDLH, Human data, and Basis for revised IDLH (where present). The text of those sections was matched case-insensitively against a list of 29 predefined neurological endpoint terms of consciousness, unconsciousness, intoxication, light-headedness, dizziness, confusion, disorientation, nausea, vomiting, headache, tremor, convulsion, seizure, coma, neurotoxic, cholinesterase, narcosis, narcotic, anesthetic, central nervous system, CNS, paresthesia, ataxia, tyrosine hydroxylase, 5-hydroxytryptamine, histamine receptor, gamma-aminobutyric acid, GABA, and glutamate.

Results: Among 384 chemicals with IDLH values examined, 95 were associated with neurological endpoint terms. Of these, 64 were organic compounds containing only carbon,

hydrogen, and oxygen. Nitrogen-containing (n=9), metal oxide (n=6), and sulfur-containing compounds (n=5) were also present. The predominant types of neurological symptoms found were headache (N=31 chemicals), nausea (N=30), and dizziness (N=19), with 23 chemicals associated with more than one of these. Other neurological signs and symptoms found included light-headedness, dizziness, tremor, intoxication, narcosis, loss of consciousness and convulsions. Human and animal data provided neurological information for different chemicals.

Conclusion/Discussion: Among the 384 chemicals with IDLH values examined, 95 had associations with nervous system effects, predominantly with nausea and headache. Other neurological findings occurred in animal model and human studies. Symptoms such as headache must be interpreted cautiously for acute occupational exposures. Future work will explore IDLH values based on neurological versus respiratory signs and symptoms. The association between neurological signs and symptoms and acute occupational exposure contexts and physiochemical properties will also be evaluated. The present characterization is an important initial step for further development of IDLH values and for consideration of risk management strategies to mitigate acute occupational exposures to chemicals that may have associations with neurological signs and symptoms.

The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention. Throughout this work, the authors used Github Copilot (Claude Sonnet 4.6) to assist with generating, debugging, or editing analytic code. The authors reviewed, tested, and validated all code.

Biography

Dr. Pandalai is a research physician and epidemiologist in the Risk Evaluation Branch, Division of Science Integration, National Institute for Occupational Safety and Health/Centers for Disease Control and Prevention. She is board certified in occupational medicine (MD, The Ohio State University, 2002; residency, University of Iowa, 2008). Formal research training includes behavioral neuroscience (MS, The University of Pittsburgh, 1992) and psychiatric epidemiology/public health (PhD, The University of Pittsburgh, 1998). Her work includes quantitative and qualitative risk assessment of chemical and non-chemical occupational hazards and translating findings into practice. She has co-chaired the Toxicology and Risk Assessment Conference and served as its continuing education director.

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A real-world study characterizing patients with Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD): A retrospective claims database study

Background: Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) is a rare, autoimmune central nervous system disease causing inflammatory demyelination and neurologic damage. Approximately 50-60% of patients experience a relapsing disease course. A single MOGAD attack could lead to severe, long-term, or permanent disability, such as trouble walking, blindness, bowel/bladder dysfunction or sexual dysfunction. There are no approved therapies for MOGAD representing a major unmet need for this disabling disease. This study characterized real-world demographics, comorbidities, and baseline costs of patients with MOGAD.

Methods: This retrospective observational study utilized the IQVIA PharMetrics® Plus Closed Health Plan claims database. Patients with a MOGAD-specific ICD-10-CM code G37.81 (≥1 inpatient or outpatient claim) with ≥3 months pre-index and ≥1 month post-index continuous enrollment between October 1, 2023, to August 31, 2025 were identified. Patients with post-index Multiple Sclerosis (MS) or NMOSD diagnoses or therapies were excluded.

Demographics, clinical characteristics, comorbidities, and baseline healthcare expenditures were evaluated during the pre-index period (up to 12 months). Relapse was defined as hospital admissions with a principal diagnosis code attributable to MOGAD, Optic Neuritis (ON), Transverse Myelitis (TM), acute disseminated encephalomyelitis, or cortical encephalitis or these conditions in any position when accompanied by select codes as the principal diagnosis.

Results: Among 987 MOGAD patients, mean age was 35.3 years (SD: 20.4) with 93.6% being ≤ 65 ; (15% aged 0-11 years, 11% aged 12-17 years), and 60% (n=591) were female. Commercial insurance was the primary payer (52%), followed by Managed Medicaid (29%), self-insured plans (13%), and State Children's Health Insurance Program (5.8%).

Over an average follow-up of 9.7 months, 3.64% (n=36) of the cohort experienced at least one relapse post-index. Clinical presentation was generally unknown/not documented during baseline (92%-overall and 81%-relapse). The rate of baseline TM was higher in relapse patients relative to the overall cohort (17% vs 4.6%), while ON rates were similar (2.8% and 2.9%), respectively.

Among prevalent ($\geq 10\%$) comorbidities; rates in relapse patients were higher than overall; hypertension (36% vs 25%), anxiety (39% vs 24%), depression (25% vs 17%), and hemiplegia (22% vs 5.3%), respectively. A higher rate of chronic pulmonary disease was observed in the overall cohort (14% vs 2.8%).

The 12-month mean baseline total cost for the overall cohort was \$47,698 (SD: \$112,771; median: \$16,263), and was higher in the relapse cohort (\$74,471 [SD: \$88,761]; median: \$37,673). Similarly, the 3-month mean baseline cost was \$16,845 (SD: \$55,866) and \$30,979 (SD: \$32,333), respectively.

Conclusion: In this real-world cohort, MOGAD affected a relatively young population, including a substantial pediatric and adolescent subgroup. Psychiatric, neurologic, and cardiometabolic comorbidity burden was notable. Baseline healthcare costs were high given this younger MOGAD population. Patients with relapse had even greater neurologic morbidity and higher costs, underscoring the incremental burden associated with relapsing disease. These findings highlight the broad clinical and economic burden of MOGAD and support the need for improved disease recognition, monitoring, and management strategies.



Syed Osama Husain

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United Kingdom

A case of vasculitic neuropathy presenting with progressive sensory motor limb involvement, posterior reversible encephalopathy syndrome and systemic symptoms in a 70 year old woman

A 70-year-old woman was brought to the Emergency Department after developing progressive weakness and altered sensation in both her hands and feet. She reported being unable to bear weight, episode of urinary incontinence, gradual decline in her health over the past nine weeks also marked by unintentional weight loss of approximately one stone, occasional nausea and vomiting. Family also reported intermittent confusion.

She also had history of cervical spinal stenosis, Raynaud's phenomenon, osteoarthritis, fibromyalgia, and rheumatoid arthritis.

On initial assessment, her hemoglobin was 75 g/L. Was diagnosed with iron deficiency anemia. Upper GI endoscopy revealed esophagitis which was treated with high dose PPI.

MRI of the spine showed multilevel degenerative spondylotic changes in the cervical and lumbar spine, with disc herniations at several levels. A posterior disc herniation at C4/C5 caused indentation on the thecal sac and the anterior aspect of the cord with mild foraminal narrowing. Importantly, there was no evidence of acute spinal canal stenosis, cord or cauda equina compression, or myelopathy.

The following day, her condition suddenly deteriorated. She developed status epilepticus and her blood pressure at the time was documented at 200/80 mmHg and was transferred to intensive care for management of status epilepticus.

Initially was empirically treated with ceftriaxone, amoxicillin, and acyclovir for meningoencephalitis and above was treatment stopped once cerebrospinal fluid analysis ruled out bacterial and viral causes.

A CT venogram of the head ruled out venous sinus thrombosis. A non-contrast CT head followed by MRI head revealed radiological features suspicious for Posterior Reversible Encephalopathy Syndrome (PRES).

Echocardiography demonstrated preserved left ventricular systolic function, left pleural effusion, and a trivial pericardial effusion without hemodynamic compromise. Differential diagnoses, including paraneoplastic syndromes, systemic vasculitides, and lupus, were considered. Serological investigations, including antinuclear antibodies (ANA) and ANA profile, were negative.

A PET-CT was performed, revealing a highly Fluorodeoxyglucose (FDG)-avid right vocal cord nodule, FDG-avid mediastinal and porta hepatis lymph nodes. ENT suggested the vocal cord nodule is benign. Patient declined lymph node biopsy at that stage.

NCS demonstrated an asymmetric neuropathy with left radial palsy, bilateral ulnar palsy, and possible bilateral peroneal involvement. Electromyography showed severe subacute denervation and a generalized axonal neuropathy, highly suggestive of vasculitis neuropathy.

Systemic features—weight loss, fluctuating confusion, inflammatory arthritis, Raynaud's phenomenon, and pleural and pericardial involvement—all raised suspicion for systemic vasculitis.

Sural nerve biopsy revealed reduced axonal density and striking perineuritis with evidence of both healed and active vasculitis. These findings confirmed the diagnosis of vasculitis neuropathy.

Patient was commenced on high-dose prednisolone (60 mg daily) which halted progression of motor and sensory weakness with initial mild improvement.

Biography

Syed Osama Husain is currently working as a senior clinical fellow in General Internal Medicine at North Manchester General Hospital, Manchester, United Kingdom. Husain has also worked as an International Training Fellow for two years at University Hospitals Birmingham, United Kingdom. Before that, Husain completed four years of Internal Medicine Training (FCPS) with the College of Physicians and Surgeons, Pakistan. Husain has also served as Lecturer in Forensic Medicine at Rehman Medical College, Peshawar, Pakistan. Husain is a graduate of Khyber Medical College, Peshawar, where Husain completed an MBBS in 2016, followed by a one-year internship at Khyber Teaching Hospital, Peshawar.



Syed Osama Husain

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United Kingdom

Cervical metastasis mimicking Ulnar neuropathy in a 78 year old man with advanced oesophageal squamous cell carcinoma

Metastatic Spinal Cord Compression (MSCC) can present with subtle or atypical neurological deficits and may be mistaken for peripheral neuropathy. We report the case of a 78-year-old man with recurrent metastatic mid-oesophageal squamous cell carcinoma who was admitted with community-acquired pneumonia and hypoxia.

During his admission, he developed numbness in the fourth and fifth digits of the left hand, which progressed over 48 hours to weakness and clawing of the hand. Neurological examination demonstrated an ulnar-claw posture with severe intrinsic hand weakness, preserved proximal strength, intact cranial nerves, no identifiable sensory level, and no upper motor neuron signs. Apart from weakness predominantly affecting the ulnar nerve distribution, there were no other focal lower motor neuron findings.

Magnetic Resonance Imaging (MRI) of the whole spine revealed extensive metastatic disease involving the cervical vertebrae (C4-C7), including a pathological fracture at C7, spinal canal stenosis, and left-sided neuroforaminal involvement at C6-C7 corresponding to the patient's neurological deficits. Given his frailty and limited prognosis, he was managed conservatively with corticosteroids, cervical immobilisation, and palliative radiotherapy.

This case highlights the risk of diagnostic anchoring on peripheral causes and emphasises the importance of early spinal imaging in patients with cancer who present with new focal neurological deficits, even in the absence of pain or upper motor neuron signs.

Biography

Syed Osama Husain is currently working as a senior clinical fellow in General Internal Medicine at North Manchester General Hospital, Manchester, United Kingdom. Husain has also worked as an International Training Fellow for two years at University Hospitals Birmingham, United Kingdom. Before that, Husain completed four years of Internal Medicine Training (FCPS) with the College of Physicians and Surgeons, Pakistan. Husain has also served as Lecturer in Forensic Medicine at Rehman Medical College, Peshawar, Pakistan. Husain is a graduate of Khyber Medical College, Peshawar, where Husain completed an MBBS in 2016, followed by a one-year internship at Khyber Teaching Hospital, Peshawar.



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Preliminary construct validity of the FRAMES battery across diagnostic groups and cross-cultural samples

Background: Neurodegenerative disorders characterized by primary socioemotional dysfunction, such as Frontotemporal Dementia (FTD), remain difficult to accurately diagnose due to the lack of objective, standardized assessments. As a result, these patients are frequently misdiagnosed and may receive delayed or inappropriate treatment, contributing to a significant burden for patients, caregivers, and healthcare systems. The Face Recognition, Affect Matching, Emotion Labeling, and Sincerity (FRAMES) battery was developed to systematically quantify multiple domains of socioemotional processing. We evaluated its performance across clinically defined groups to assess its utility as a diagnostic tool.

Methods: FRAMES data were analyzed from participants classified as healthy controls (HC), Alzheimer's Disease (AD), semantic behavioral variant FTD (sbvFTD), and behavioral variant FTD (bvFTD) (N=26). Subtest scores were compared using one-way ANCOVAs adjusting for age and sex. Item-level difficulty and group-specific performance patterns were examined to assess sensitivity and construct validity.

Results: FRAMES demonstrated a broad range of item difficulty ($p \approx 0.14$ – 1.00), with no evidence of ceiling effects, supporting sensitivity across performance levels. Significant group effects were observed for Face Perception ($F=9.44$, $p<0.001$), Affect Matching ($F=8.25$, $p=0.001$), and Emotion Labeling ($F=6.83$, $p=0.002$). Distinct performance patterns emerged across diagnostic groups, with sbvFTD showing disproportionate impairments on higher-order socioemotional tasks. These patterns enabled differentiation between sbvFTD and bvFTD, particularly in affect matching and emotion labeling ($p=0.002$, $p=0.004$, respectively).

Conclusions: FRAMES demonstrates preliminary construct validity, with appropriate item difficulty, cross-cultural applicability, and sensitivity to socioemotional deficits. These findings support its potential as a clinically informative tool for detecting and differentiating socioemotional dysfunction across neurodegenerative disorders. With further validation in a larger sample, FRAMES may contribute to earlier and more accurate diagnosis, improved phenotyping, and more targeted clinical management.

Biography

Tanisha Mehta is a second-year undergraduate student at University of California, Berkeley studying Molecular and Cell Biology. Mehta conducts research in the Rankin Lab at the University of California, San Francisco, where Mehta helps develop and evaluate quantitative tools to assess socioemotional dysfunction in dementia, including the FRAMES battery. Mehta's work focuses on improving the measurement and detection of socioemotional deficits to support more accurate diagnosis. Mehta is broadly interested in the intersection of neuroscience and clinical medicine and aims to pursue a career as a physician-scientist.



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Autophagy changes in brain cells of 5-month-old db/db mice with diabetes type 2 and development of neurodegeneration

Dysregulation of autophagy have been shown in several diseases, like atherosclerosis, inborn lysosomal storage diseases and diabetes Type 2, concentrating attention at lysosomes as a potential target for therapy of several diseases. Simultaneously earlier it was shown, that autophagy in Type 2 diabetes was associated with the formation of features of Alzheimer disease (AD). A common mechanism appears to be the impairment of autophagy, process which was not studied enough in brain cells - neurons and glial cells. A good opportunity to study the correction of diabetes and neurodegeneration is provided in db/db mice, genetic model of diabetes and obesity, developed sign of AD with ageing. In our previous work we have found that animals can be treated by some autophagy inducers, activating this process via mTOR-independent pathway. In young (male 3-month-old db/db mice) autophagy inducer reduced obesity, attenuated hyperglycaemia, activated autophagy in the liver, heart and brain cells, weakened neuroinflammation and oxidative stress, restoring cognitive impairment. The aim of this work was to study in what extent the therapeutic effect of autophagy induction depends from the age of mice and autophagy gene transcription. Experiments were made in SPF vivarium of the Institute of Cytology and Genetics of RAS, Novosibirsk, Russia. The therapeutic effect of treatment by autophagy inducer (3% trehalose drinking, 30 days) was investigated in 5-month-old db/db mice. We studied Atg8 gene coding protein - marker of autophagy LC3-II, gene Becn1 coding protein Becn1, connected with autophagy and gene Insr. Trehalose drinking did not reduce the body weight or blood glucose and cholesterol level, but significantly decreased the expression of autophagy gene Atg8 and more significantly the insulin receptor gene Insr in brain cells. Thus, a different effect of autophagy induction was obtained for old 5-month-old db/db mice and young db/db mice, consisting in the absence of

in the autophagy activation genes. It was concluded that autophagy activation in db/db mice was decreasing with age and became not effective in old 5-month-old db/db mice.

Biography

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



Toru Kondo

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ECRG4 is a potential initiator of amyloid pathology in Alzheimer's disease and a novel diagnostic plasma marker for the disease

Alzheimer's Disease (AD) is a progressive neurodegenerative disorder characterized by the accumulation of β -amyloid ($A\beta$) and the formation of neurofibrillary tangles. Although the amyloid cascade hypothesis underscores the centrality of $A\beta$ accumulation, the precise initiators of this process remain unclear. We hypothesized that Esophageal Cancer-Related Gene 4 (ECRG4), which is associated with cognitive impairment and is upregulated in AD, directly contributes to amyloid pathology. Here, we demonstrated that ECRG4 interacts with Amyloid Precursor Protein (APP)/ $A\beta$, leading to increased APP/ $A\beta$ accumulation. We showed that intracerebral injection of synthetic ECRG4 into AD-model mice significantly augmented APP/ $A\beta$ deposition. Notably, the co-localization of ECRG4 with APP/ $A\beta$ increased with AD severity in human hippocampal tissues.

Furthermore, the observation of ECRG4 accumulation in the blood vessels in AD hippocampus led us to develop a novel ECRG4 ELISA system. We observed that ECRG4 peptides, particularly those consisting of Amino Acids (AA) 108–132, were elevated in the plasma of approximately 25% of patients with mild cognitive impairment and 50% of those with AD, compared to individuals without dementia. We showed that ECRG4 AA 71-107 killed oligodendrocytes and induced the accumulation of its precursor cells on blood vessels, resulting in the disruption of blood-brain barrier in the early phase of AD progression. Together, our findings reveal that ECRG4 acts as a potential initiator of amyloid pathology in AD and as a novel plasma marker for AD linked to ECRG4-dependent dysfunction in oligodendrocytes.

Biography

Dr. Toru Kondo is currently working as Distinguished Professor at the Institute for Genetic Medicine, Hokkaido University. He has received his PhD from Osaka University (Prof Yoshio Okada). He worked at the Osaka Bioscience Institute (Prof Shigekazu Nagata) and University College London MRC LMCB (Prof Martin Raff), as a postdoctoral fellow. He then worked as PI at the Cambridge University Centre for Brain Repair, RIKEN CDB, and Ehime University Proteo-Medicine Research Center. He has been serving as an Associate Editor of Stem Cells and a council member of the Japanese Association for Molecular Target Therapy of Cancer.



Uttara Koul, MD

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United States

A rare case of Epilepsia Partialis Continua in an adult patient with MIRAS

A 38-year-old female with Mitochondrial Recessive Ataxia Syndrome (MIRAS, homozygous POLG A467T mutation), diagnosed in her late twenties, was transferred to us from another hospital. She was intubated and sedated for suspected status epilepticus. Her condition had led to progressive gait instability, muscle weakness, ophthalmoplegia, peripheral neuropathy, and wheelchair dependence. She was diagnosed with epilepsy one year prior, managed with levetiracetam. After missing doses, she experienced a generalized seizure, became unresponsive, and was intubated. Initial EEG showed Generalized Rhythmic Delta Activity (GRDA), and her sedation was reduced. After 12-15 hours, she exhibited involuntary jerking movements, head turning, and became alert, but the events worsened with tachycardia, desaturation, and urinary incontinence. EEG remained unchanged with persistent GRDA. MRI was normal, and no active infections were detected. Given the clinical presentation, refractory EPC/focal motor seizures from a deeper focus were suspected. Despite the negative EEG, a propofol drip was started for suppression. Levetiracetam was switched to brivaracetam, lacosamide was increased, and gabapentin and scheduled lorazepam were added.

There were limitations on antiseizure medications that could be utilized due to her POLG mutation, and risk of liver failure with agents such as valproic acid, phenobarbital, etc. She was started on multivitamins and folinic acid. Several goals of care conversations were had with the patient's family due to the refractory nature of her disease. After 48 hours of suppression, sedation was weaned, and she had significant reduction in her seizure burden. She was extubated successfully and subsequently discharged home with conservative measures. She continues to experience fatigue and cognitive side effects in the few weeks after her seizure control, but is able to support her own nutrition, and communicate effectively with her doting family.

Biography

Dr. Uttara Koul is a board certified Neurologist and Neurointensivist with St. Vincent's Medical Center and an Assistant Professor with the Frank H. Netter School of Medicine. After completing medical school in India, Koul moved to the US in 2017 to pursue Neurology residency at St. Louis University followed by a fellowship in Neurocritical care at Rush University, Chicago. Koul has a strong interest in quality improvement and education, and Koul's academic interests lie in vascular and autoimmune neurologic emergencies as well as medical humanities.



Vikrant Devgire^{1*}, Zainab Mahmud²

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When decannulation fails: Two-year outcomes of hyperacute neurorehabilitation patients discharged with a tracheostomy

Background/Aims: Patients requiring a tracheostomy following a neurological insult undergo structured weaning and decannulation through a multidisciplinary approach in hyperacute neurorehabilitation. Despite these efforts, some patients are discharged with a tracheostomy. We aimed to evaluate the two-year outcomes of patients admitted with a tracheostomy, with particular emphasis on those discharged without successful decannulation and the influence of lesion location.

Methods: A retrospective review was undertaken of all patients admitted to a Hyperacute Rehabilitation Unit (HARU) with a tracheostomy between 2022 and 2024. Electronic patient records were reviewed to collect demographic data, neurological pathology, tracheostomy and decannulation outcomes, discharge status, and mortality up to two years following discharge.

Results: Seventy-two patients were admitted to HARU with a tracheostomy during the study period. Overall, the decannulation success rate was 69.4% (50/72). Twenty-two patients (30.6%) were discharged with a tracheostomy. At two-year follow-up, 14/22 (63.6%) remained alive, while 8/22 (36.4%) had died. Of total cohort, 12/72 (16.7%) had neurological injuries involving the infratentorial compartment. This subgroup had markedly poorer outcomes: 7/12 (58.3%) died, 3/12 (25.0%) remained alive with a tracheostomy at two years, and only 2/12 (16.7%) achieved successful decannulation. In comparison, patients with supratentorial pathology had substantially higher decannulation rates (38/60, 63.3%) and lower mortality (11/60, 18.3%). Utilising the Fisher exact test, the difference in mortality ($p=0.003$) and decannulation ($p=0.006$) was significant between these two subgroups.

Conclusion: Most patients admitted to HARU with a tracheostomy achieved successful decannulation through a multidisciplinary rehabilitation approach. However, patients who remained tracheostomy dependent at discharge represented a high-risk group, with a two-year mortality rate of 36.4%. In subgroups, the supratentorial were noted to have statistically significant higher decannulation rate and lower mortality as compared to the infratentorial. These findings highlight the importance of early prognostic discussions for patients discharged with a tracheostomy following their neurological injury. Although this represents a relatively large single-centre HARU cohort, larger multicentre studies are needed to confirm these findings and improve the precision of long-term outcome estimates.

Biography

Vikrant Devgire is a final year physical medicine and rehabilitation resident and Devgire's previous training roles and postgraduate qualification with MSc Clinical Neurology and Membership of Royal College of Physicians, UK have allowed Devgire to gather skills and experience in acute and chronic rehabilitation management of neurological conditions. Devgire's area of interest is around brain and spine rehabilitation.



Wesam Asaad

Independent Researcher, Switzerland

Emergent neural phenotypes from local developmental optimization: A mechanistic framework for brain organization and neurodiversity

A central question in neuroscience is how complex, structured brain architectures emerge from local cellular interactions during development. Here, we demonstrate that local developmental optimization alone is sufficient to generate realistic brain organization and stable neurodivergent trajectories, without requiring global supervision or centralized control. We present a mechanistic, simulation-based framework in which individual neurons act as local agents balancing competing biological constraints, including information-processing efficiency, wiring economy, and metabolic cost.

Neuronal activity, synaptic plasticity, adaptive tradeoff regulation, and spatial growth are jointly governed by a unified local energy principle. Stochastic and entropy-regularized mechanisms promote developmental exploration, while slow adaptive dynamics stabilize emerging network structure. This framework produces robust developmental trajectories that naturally transition through phases of growth, modular differentiation, synaptic pruning, and stabilization.

Across a wide range of conditions, the model reproduces key features of cortical organization, including hierarchical modularity (modularity index $Q \sim 0.45\text{--}0.6$), small-world connectivity (small-world index $\sigma \sim 1.5\text{--}1.7$), wiring-cost scaling laws, and characteristic synaptic pruning curves. These emergent properties arise directly from local interactions, demonstrating that large-scale brain architecture can be explained as a collective outcome of simple neuronal optimization rules.

By systematically varying developmental tradeoffs, stochasticity, and adaptive learning rates, the model generates a diverse ensemble of stable neural phenotypes, forming distinct developmental attractors rather than pathological deviations. These trajectories capture a continuum of structural and functional brain organizations, offering a mechanistic interpretation of neurodiversity as adaptive specialization under competing biological constraints. Divergence across trajectories is quantified using modularity, hierarchy, network efficiency, and rewiring dynamics, revealing structured variation rather than disorder.

The framework is computationally scalable and dimensionless, allowing systematic exploration of developmental regimes across network sizes and parameter spaces. Logging of phase-specific network statistics, motif persistence, and efficiency–cost tradeoffs enables detailed analysis of robustness, adaptability, and sensitivity to perturbations.

Together, these results provide a principled link between local cellular processes and macroscopic brain organization, showing how structured neural architectures and functional diversity emerge naturally from developmental optimization. More broadly, this work suggests that brain diversity reflects adaptive solutions to fundamental developmental tradeoffs, providing a unifying mechanistic account of both typical and neurodivergent neurodevelopment.

Biography

Wesam Asaad is an independent research scientist working on mechanistic and computational models of neural development, brain organization, and neurodiversity. Asaad's research investigates how local neuronal interactions and adaptive tradeoffs give rise to emergent network structure, functional connectivity, and developmental variability. Asaad's broader interests include systems neuroscience, theoretical biology, psychometrics, and self-organizing complex systems.



Yatri S. Patel, M.D.

SVP Hospital, NHL Municipal Medical College, Ahmedabad,
India

Efficacy of rasagiline as an adjunct therapy in chronically treated Parkinson's disease: A meta-analysis

Parkinson's Disease (PD) is a progressive neurodegenerative disorder characterized by degeneration of dopaminergic neurons in the basal ganglia, leading to disabling motor and non-motor symptoms. Long-term dopaminergic therapy, including levodopa and dopamine agonists, is frequently complicated by motor fluctuations such as wearing-off phenomena and dyskinesias. Rasagiline, an irreversible Monoamine Oxidase-B (MAO-B) inhibitor, enhances central dopaminergic activity by preventing dopamine degradation and is commonly used as adjunctive therapy. Although several randomized controlled trials have evaluated its benefits, the overall magnitude of clinical improvement remains variably reported. This meta-analysis was conducted to systematically assess the efficacy of adjunct rasagiline compared with placebo in patients with Parkinson's Disease receiving chronic dopaminergic therapy.

Objective: To evaluate the efficacy of rasagiline as an adjunct therapy in reducing motor fluctuations and improving functional outcomes in patients with Parkinson's Disease undergoing long-term dopaminergic treatment.

Methods: A systematic literature search was performed according to PRISMA 2009 guidelines to identify randomized controlled trials comparing rasagiline with placebo in Parkinson's Disease patients receiving long-term dopaminergic therapy. Only head-to-head randomized controlled trials were included, while observational studies, unpublished studies, and trials not meeting PRISMA criteria were excluded. Data extraction was performed systematically and statistical analysis was conducted using RevMan version 5.3. The primary outcomes included changes in the Unified Parkinson's Disease Rating Scale (UPDRS), particularly motor-ON and

Activities of Daily Living during OFF periods (ADL-OFF). Standardized mean differences were calculated using both fixed-effect and random-effects models. Heterogeneity across studies was assessed using the I^2 statistic, and statistical significance was defined as $p < 0.05$.

Results: Six randomized controlled trials comprising 886 patients were included in the analysis. Adjunct rasagiline demonstrated a significant improvement in motor function compared with placebo. For UPDRS motor-ON scores, the fixed-effect model showed a significant benefit (CI 0.586–0.781; $p < 0.001$), while the random-effects model also demonstrated significant improvement (CI 0.260–1.173; $p = 0.002$). Similarly, rasagiline significantly improved Activities of Daily Living during OFF periods. The fixed-effect model demonstrated a CI of 0.854–1.061 ($p < 0.001$), while the random-effects model showed a CI of 0.106–1.755 ($p = 0.027$). Overall heterogeneity between studies was acceptable.

Conclusion: Adjunct rasagiline at a dose of 1 mg/day significantly improves motor function and daily functional performance in patients with Parkinson's Disease experiencing motor fluctuations during long-term dopaminergic therapy. These findings support the use of rasagiline as an effective adjunctive therapy for optimizing symptomatic control and improving quality of life in patients with Parkinson's Disease.

Keywords: Rasagiline, Parkinson's Disease, UPDRS, Motor on and off, Meta-Analysis.

Biography

Dr. Yatri Patel, MD, is a physician trained in Internal Medicine with a strong commitment to academic medicine and clinical research. Patel completed postgraduate training in India and has actively participated in academic presentations and research activities during a medical career. Patel's primary areas of interest include neurology, geriatric medicine, and infectious diseases, with a focus on evidence-based clinical practice and improving patient outcomes. Patel is particularly interested in advancing knowledge in complex neurological and geriatric conditions. As a presenting author, Patel is dedicated to contributing to scientific discourse and promoting research that enhances the quality of patient care.



Yatri S. Patel, M.D.^{1*}, Siddharth Panchal²

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Efficacy and safety of erenumab in preventive and therapeutic treatment of patients suffering from migraine: A meta analysis

Background: Erenumab (AMG 334) is a first fully human monoclonal antibody that targets and blocks the canonical Calcitonin Gene Related Peptide (CGRP) receptor approved for use in migraine. CGRP plays a very pivotal role in patho-physiology of migraine. The objective was to generate statistical evidence on the basis of existing data for erenumab in terms of efficacy and safety in various treatment regimens with different dosages.

Methods: A total of 7 RCTs following PRISMA guidelines and matching inclusion and exclusion criteria were collected of erenumab vs placebo in treatment of migraine. Studies with >50% responder rate from baseline in terms of migraine days per month at 12 weeks was chosen as primary clinical outcome measure. Secondary outcomes chosen were mean migraine days from baseline and safety outcomes in terms of adverse events. 5.3 software was used for the calculation of Relative Risk (RR). P value less than 0.05 was considered significant.

Results: Data of 3615 patients were included. The use of Erenumab was associated with increased rates of achieving >50% responder rate as compared with placebo. (RR=1.591; 95% CI=1.322 to 1.915; P<0.001). Both 70mg and 140 mg of erenumab were associated with increase in responder rate. Reduction in mean migraine days was observed in erenumab as compared to placebo, (SMD=7.348; 95% CI=4.733 to 9.963; P<0.001). Treatment related adverse events (RR=0.974; 95% CI=0.901 to 1.052; P=0.496) were not significant in erenumab as compared to placebo.

Conclusion: Erenumab subcutaneous once monthly was the most efficacious treatment regimen and was not associated with a significant risk for treatment related adverse events.

Keywords: Erenumab, Efficacy, Safety, Migraine, Meta Analysis.

Biography

Dr. Yatri Patel, MD, is a physician trained in Internal Medicine with a strong commitment to academic medicine and clinical research. Patel completed postgraduate training in India and has actively participated in academic presentations and research activities during a medical career. Patel's primary areas of interest include neurology, geriatric medicine, and infectious diseases, with a focus on evidence-based clinical practice and improving patient outcomes. Patel is particularly interested in advancing knowledge in complex neurological and geriatric conditions. As a presenting author, Patel is dedicated to contributing to scientific discourse and promoting research that enhances the quality of patient care.



Younok Dumortier Shin, PhD, MBA

OnusBio, United States

Redefining holistic approaches in neurological drug development: Gaps and opportunities

The concept of holistic patient care has gained traction in neurological drug development and clinical trials, emphasizing patient-focused endpoints and real-world measures of quality of life rather than purely biochemical outcomes. The key success factor here is multidisciplinary collaboration across neurologists, psychiatrists, rehabilitation specialists, nurses, psychosocial caregivers, and most of all, the patients. Furthermore, it requires continuous digital monitoring such as wearable devices empowering patients with ability to track symptoms and improvements. However, the flawless integration of biological mechanisms, clinical relevance, and patient life impact is a challenging task, and the technology is advancing limited by regulatory requirements. As a result, a growing body of literature reveals that many so-called holistic approaches remain limited in scope. Common gaps include inconsistent inclusion of mental health endpoints, inadequate measurement of functional and quality-of-life outcomes, exclusion of patients with psychiatric comorbidities, and lack of standardized frameworks for measuring the effectiveness of non-pharmacological interventions. Moreover, interdisciplinary collaboration is often limited, and trial designs rarely account for real-world complexities affecting adherence and treatment response. This presentation will provide an overview of these shortcomings and recommendations to advance truly holistic trial design. Recommendations include adaptive trial designs that integrate behavioural and lifestyle interventions; fostering interdisciplinary trial teams; and aligning patient-centered outcomes with regulatory expectations. Example will be provided in the latest effort in pain management.

This presentation will particularly benefit researchers, scientists, clinicians, and digital health innovators involved in neurological drug development. It will also be valuable for healthcare professionals working in multidisciplinary neurological care settings, as well as patient

advocacy groups aiming to promote more inclusive and meaningful outcome measures in research. By addressing both strategic and practical dimensions, the session offers insights relevant to decision-makers shaping future trial frameworks and care models in neurological drug development.

Biography

Younok Dumortier Shin, PhD, MBA, has more than 20 years of experience in pharmaceutical and biotech industry including GlaxoSmithKline, Bristol-Myers Squibb, Johnson and Johnson, Allergan, Dermelix and Avalo Therapeutics. During Shin's career, Shin oversaw many multibillion-product launches around the world, owned responsibilities for uninterrupted commercial supply, and championed creating innovative drug development programs to accelerate the clinical and regulatory approval timelines. Currently, Shin is serving as the CEO of OnusBio, a biotechnology company focusing on developing innovative medicines to treat debilitating conditions affecting nervous systems.



Zoë Reinus, D.O.

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The million-dollar workup

Doctors work in a paradigm of defensive medicine, defined as the practice of recommending a diagnostic test or medical treatment that is not necessarily the best option for the patient, but mainly serves to protect the physician against the patient as potential plaintiff (Wikipedia: Defensive Medicine, 2025). It's a reflex against a core vulnerability of the practice of medicine: we take care of people and sometimes it goes wrong. Except, this reflex is not always in the best interest of the patient.

In the million-dollar work up I describe below, the repetition – repeat procedures, repeat laboratory tests ordered three-times over – highlights a second vulnerability that cannot be taught, only experienced: as doctors, we face diagnoses that will break your heart.

The disease in question has a highly variable – and often initially vague – presentation. My patient had all the appropriate work-up in a relatively timely manner. And it didn't matter. She was misdiagnosed; she was given the run around; and (debatably) healthcare resources were overutilized.

This presentation explores what happens when medicine is confronted with a devastating diagnosis – when the effects of moral hazard are at odds with the human desire for certainty.

Biography

Reinus is in a second year of physical medicine and rehabilitation (PM&R) residency at the University of Connecticut, drawn to PM&R for its focus on optimization rather than fixing. Reinus is also learning that PM&R is unique in its exposure to a wide array of diagnoses, which – as a historically avid watcher of *House, M.D.* – was always Reinus's fate. Extra-clinically, Reinus is interested in central sensitization, neuroprognostication, and the practice of medicine and the system of healthcare.

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POSTER PRESENTATIONS





**Amelie Guinee^{1*}, Jordan Strack¹,
William J. Gavin², Patricia L.
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COVID-19 pandemic-related adversity, error-related neural activity, and cognitive control in children

The COVID-19 pandemic introduced widespread disruptions to children's social, educational, and home environments, often increasing exposure to adversity. Pandemic-related adversity has been broadly associated with differences in children's general cognitive and mental health outcomes. Yet, few studies have examined the effects of COVID-19-related adversity on specific aspects of neural and neurocognitive functioning in children. Pandemic-related adversity may influence the amplitude of Error-Related Negativity (ERN), which reflects neural activity underlying error monitoring, in children. The goal of this study is to investigate the associations of pandemic-related adversity with ERN amplitude and cognitive control in children. In addition, we will examine whether socioeconomic background and age at the time of the pandemic moderate these associations. We hypothesize that greater pandemic-related adversity exposure will be associated with altered ERN amplitudes and worse performance on cognitive control tasks in children. We expect that these associations will be stronger among children who were younger at the time of the pandemic, consistent with heightened developmental sensitivity during early childhood, and those experiencing reduced socioeconomic resources.

Participants were children ages 5-13 years (N=112; 56% male; 83% white, non-Hispanic/Latine) living in a medium-sized city in the western United States. They completed one in-person study session between 2021-2025 that included Electroencephalogram (EEG) recordings during a speeded flanker task. For each child, neural activity on incorrect trials was averaged to create a mean ERN. Children also completed measures of cognitive control, including the NIH Toolbox List Sorting Working Memory and Dimensional Change Card Sort tasks, and the Head-Toes-Knees-Shoulders (HTKS) task. Parents completed the Epidemic-Pandemic Impacts Inventory (EPII), which asks about adversity across domains such as social isolation,

educational disruption, and household stress. The EPII Total Negative Impact score will be used in analyses. Socioeconomic factors, including family income-to-needs ratio and parental education, were also measured.

Multiple linear regression models will be used to examine associations between pandemic-related adversity (independent variable) and cognitive control task performance and ERN amplitude (dependent variables). Child age will initially be included as a continuous moderator; if the interaction is non-significant, it will be removed from the model. A similar analytic approach will be used to examine socioeconomic factors (family income-to-needs ratio, parental education) as moderators. Covariates to be considered include child sex and race/ethnicity.

Findings from this study will help us understand the ongoing cognitive developmental impacts of the COVID-19 pandemic on children. Focusing on a unique historic event that led to unconventional cognitive and social learning experiences for children, our results will provide a deeper understanding of early adversity and the importance of childhood sensitive periods. By identifying whether age and socioeconomic factors shape these associations, our findings will clarify which children may be most vulnerable to lasting pandemic-related cognitive impacts, information that can be applied to ensuring children receive effective interventions.

Biography

Amelie Guinee is a cognitive neuroscience doctoral student at Colorado State University. Guinee is interested in the intersection of child cognitive development, socioeconomic status, and psychological disorders. Specifically, Guinee is curious about the etiology and diverse symptom presentation of OCD in relation to cognitive and neural processing. Guinee received a Bachelor of Science in Psychology from Colorado State University. In her free time, she enjoys the gym, reading, and writing.



Angela Philips*, Patrick Sleiman, Elias Samaha

University of Illinois College of Medicine Peoria, United States

Stimulus-induced pseudo-burst suppression in Powassan encephalitis presenting as NORSE: An unusual EEG finding

Background: Burst suppression is an EEG pattern commonly targeted during treatment of refractory status epilepticus with anesthetic agents, characterized by brief bursts of cerebral activity interspersed with 10-second periods of suppression. In true pharmacologic burst suppression, painful stimulation typically evokes cortical activation, including high-amplitude negative waves, bursts with superimposed 10-Hz activity, or thalamic spindles. Paradoxical increases in suppression following stimulation termed pseudo-burst suppression are extremely rare and not well described, raising the potential for misinterpretation.

Case Discussion: A 21-year-old male presented with fever, altered mental status, and generalized tonic-clonic seizures requiring intubation. Despite broad antimicrobial therapy, multiple antiseizure medications, anesthetic agents, and immunotherapies, he progressed to NORSE. MRI revealed bilateral thalamic and basal ganglia abnormalities. Extensive infectious testing was initially unrevealing; CDC reference testing confirmed Powassan virus encephalitis. Continuous EEG showed persistent generalized periodic discharges at 1–3 Hz without discrete seizures. Painful stimulation and repositioning reproducibly induced transient increases in background suppression with concurrent attenuation of discharges. This stimulus-dependent pattern occurred independently of anesthetic dosing and resolved with cessation of stimulation, distinguishing it from pharmacologic burst suppression.

Conclusions: This case represents, to our knowledge, the first report of reproducible stimulus-responsive pseudo-burst suppression in NORSE due to Powassan encephalitis. Unlike true burst suppression, it is paradoxical, transient, and non-dose-dependent, reflecting a reversal

of expected cortical reactivity. Recognition of this unique EEG response is critical to avoid misinterpretation of seizure control and inappropriate therapeutic decisions in refractory status epilepticus.

Biography

Angela Philips is currently an intern in Neurology at the University of Illinois College of Medicine Peoria. Philips received a bachelor's degree in medicine from Dr. Bhimrao Ambedkar University, Agra, and a DNB (General Medicine) from National Board of Medical Examinations, Delhi, and MRCP (UK) from Royal Colleges of Physicians of the UK. Philips is interested in Neuroinflammation and Neuromuscular disorders.



**Angela Philips*, Ashwath
Ravisankar, Deepak Nair**

University of Illinois College of Medicine Peoria, United States

Alexia without agraphia in cerebral venous thrombosis: A case report

Objective: We present a rare case of alexia without agraphia in the setting of cerebral venous thrombosis and review the neuroanatomical basis, diagnostic considerations, and clinical relevance of this uncommon presentation.

Background: Alexia without agraphia is a rare disconnection syndrome in which visual input from the right visual cortex cannot reach the language-dominant left hemisphere. It typically arises from lesions involving the left occipital lobe and the splenium of the corpus callosum. Acquired pure alexia due to hereditary thrombophilia is exceptionally rare.

Case Presentation: A 60-year-old Caucasian female with no significant past medical history presented with two days of word-finding difficulty and a two-week history of left-sided headache. Neurological examination revealed alexia without agraphia: she was unable to read printed or handwritten material, including her own writing, while spontaneous writing and speech remained intact.

Non-contrast CT of the head demonstrated a left temporal intraparenchymal hemorrhage, and CT venography revealed a left transverse sinus thrombus extending into the left jugular vein. Further evaluation revealed extensive thromboembolic disease, including right lower extremity deep vein thrombosis (common femoral, popliteal, and gastrocnemius veins) and bilateral pulmonary emboli without hemodynamic compromise. Hypercoagulable testing identified a prothrombin gene mutation, consistent with a positive family history of thrombosis.

The patient was managed with anticoagulation in the neuro-ICU, initially with heparin and later transitioned to therapeutic enoxaparin, with close monitoring. Serial CT scans showed no

hematoma expansion, and neurosurgical intervention was not required. The patient improved clinically, had no functional limitations on physical or occupational therapy evaluation, and was discharged on therapeutic anticoagulation with outpatient hematology and neurology follow-up.

Discussion: Alexia without agraphia is classically caused by lesions in the left occipital cortex and splenium, producing a disconnection between visual input and the language-dominant hemisphere. More recent evidence emphasizes the role of the ventral occipitotemporal cortex, including the visual word form area (fusiform gyrus), as a critical node for reading. Cerebral venous thrombosis can also cause venous congestion, further disrupting interhemispheric visual-linguistic connections. This case illustrates that temporo-occipital lesions, even when temporal-predominant on CT, can clinically manifest as alexia without agraphia. To our knowledge, alexia without agraphia as a manifestation of thrombophilia-associated cerebral venous thrombosis with temporal-predominant involvement is extremely rare, emphasizing the importance of considering CVT in atypical cognitive presentations.

Conclusion: This case highlights a rare manifestation of cerebral venous thrombosis presenting as alexia without agraphia due to left temporo-occipital involvement. It underscores the importance of recognizing venous infarction in focal disconnection syndromes, illustrates the neuroanatomical basis of reading pathways, and emphasizes the limitations of the NIH Stroke Scale in detecting subtle cognitive deficits.

Biography

Angela Philips is currently an intern in Neurology at the University of Illinois College of Medicine Peoria. Philips received a bachelor's degree in medicine from Dr. Bhimrao Ambedkar University, Agra, and a DNB (General Medicine) from National Board of Medical Examinations, Delhi, and MRCP (UK) from Royal Colleges of Physicians of the UK. Philips is interested in Neuroinflammation and Neuromuscular disorders.

Arun Antony^{1*}, Diji Johnson¹, Gianna Antony²

¹Jersey Shore University Medical Center, Neptune, New Jersey, United States

²The Hun School of Princeton, United States

Inpatient hospitalizations for dementia in the United States: A 2022 national analysis

Objective: To analyze the characteristics of patients with dementia admitted as inpatients in the U.S. in 2022.

Intro/Background: According to the World Health Organization, 57 million people around the world live with dementia. The symptoms of dementia include impaired short-term memory, aphasia, executive dysfunction, and altered behavior, impacting activities of daily living. Few studies assessed the demographic and clinical characteristics of patients with dementia admitted to the hospital, especially in large data sets.

Methods: We analyzed the National Inpatient Sample (NIS) 2022 and extracted data related to dementia using ICD-10 diagnosis codes. Data regarding demographic and clinical characteristics, including etiologies of dementia and investigations performed, were analyzed. Statistical analysis was performed using STATA software.

Results: There were 21,076 patients. 53% were female. The mean age was 76 (SD=13). 74% were White, 14% Black, 7% Latino, 2% asian, 0.4% Native American, 2% other. The mean Length of Stay (LOS) was 12 days. 19.5% of patients were diagnosed with vascular dementia, 80.16% with other types of dementia and 0.31% had dementia due to another underlying condition. Patients with dementia underwent lumbar puncture (n=493), psychotherapy (n=360) and EEG (n=310).

Conclusion: Dementia was more common in females in the hospital setting. Patients with dementia had a longer LOS in the hospital. One in five patients with dementia had a vascular etiology. The most common procedures performed on patients with dementia were lumbar puncture, psychotherapy, and EEG. Larger studies analyzing temporal trends are needed to substantiate our findings, assess economic impact, and plan dementia care in inpatient settings.

Ava Jacob*, Arun Antony, MD

Jersey Shore University Medical Center, United States

Systematic review of the effects of auditory stimulation and music as a therapy for epilepsy

Introduction: Scientists continue to explore novel therapies for epilepsy since the current treatment options like medications, epilepsy surgery, neuromodulation and ketogenic diet do not always offer complete seizure control and are associated with side effects. Auditory stimulation and music have been shown to have a variety of effects on EEG and epilepsy in multiple studies; hence we attempted to perform a systematic review to critically appraise and synthesize all related published evidence.

Methods: We searched the PubMed database with the terms 'auditory stimulation' and 'epilepsy' and obtained 227 results. We selected clinical trials, observational studies, and abstracts in English on auditory stimulation and music as a therapy for seizures published between 1996 and 2026 and available for review as per PRISMA criteria.

Results: A total of 179 patients, aged 0–78 years, from 10 studies were included in the analysis. Intracranial electrodes were used to record seizures in three studies. Compositions of Mozart, especially K448 were used in most studies, but rarely the effects of 40 Hz and 440 Hz tones were analyzed. The duration of therapy ranged from 8 minutes to one year. All studies reported positive effects of music and auditory stimulation on patients with seizures. The average decrease of interictal epileptiform discharges from listening to 40Hz tone was 35.25%, and from listening to Mozart K448 ranged from 28% to 71.6%. The reduction in interictal epileptiform discharges was more pronounced in the frontal lobe and in patients with idiopathic epilepsy without cognitive delay. Four studies analyzed seizure frequency and reported a minimum average seizure reduction of 20.5% and seizure freedom in several patients. One study reported significant improvement in behavior and sleep quality.

Conclusion: Auditory stimulation at 40 Hz and music in genres similar to Mozart should be considered for personalized therapy options for patients with epilepsy. Treatment criteria like parameters of auditory stimulation and musical composition, epilepsy syndromes that benefit from music therapy and the neurophysiology of music-related neuromodulation need further study.



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Mild Cognitive Impairment (MCI) in Parkinson's disease: Prevalence given MDS criteria

The prevalence of Mild Cognitive Impairment (MCI) in Parkinson's Disease (PD) has been wide ranging, from 15-90% across reports, likely due to a number of key variables, including age at evaluation, and time since symptom onset. Additionally, the criteria used for identifying cognitive impairment is important, with some studies applying more rigorous Movement Disorder Society (MDS) Level II criteria, report a higher incidence of MCI, than Level I (40-60+% vs 20-30%, respectively). The Level I criteria utilizes a brief global cognitive screening, such as the Montreal Cognitive Assessment (MoCA), while the Level II criteria calls for a more formal neuropsychological evaluation, including two tests in each of five cognitive domains: attention/working memory, executive function, episodic memory, visuospatial function, and language. The threshold for MCI requires at least two impaired test scores (<-1.5 SD below the normative cut-off), wherein either two tests fall within a single cognitive domain, or one test is impaired within each of two separate cognitive domains. Additionally, change in cognitive functions from pre-morbid intelligence is to be considered, per MDS Level II criteria, and such relative change has been found to further increase identification of MCI (to 79%). Through careful application of the MDS Level II criteria and comparison with more lenient Level I criteria, a high prevalence of MCI was identified at baseline among initial enrollees ($n=15$) in an on-going pedal-n-play clinical trial investigating interactive physical and cognitive exercise (iPACES), such that 90% were classified as MCI with Level II criteria vs. 40% with Level I. This preliminary finding is consistent with some prior reports of very high initial prevalence of MCI (e.g., approximately 92% in McDermott et al., 2018), and suggests that MDS Level II criteria, when also considering pre-morbid intelligence, appears significantly more powerful for detecting cognitive challenges. Given the apparently high prevalence of MCI when using a thorough approach, care should be taken to methodically examine neuropsychological functions among those with PD.

Biography

Cay Anderson-Hanley, is a Principal Investigator of NIH-funded clinical trials, with a focus on innovating pedal-n-play neuro-exergaming to enhance brain health, especially among those with neurological conditions. As a professor of neuroscience, the interactive Physical and Cognitive Exercise System (iPACES) was invented and patented in her lab, in an effort to ameliorate cognitive decline for those with Alzheimer's and Related Disorders (ADRDs). This technology has been adapted most recently for those with Parkinson's Disease and is being evaluated in an ongoing clinical trial. Pilot work has also explored utility for those on the autism spectrum (ASD) as well as Attention Deficit Hyperactivity Disorder (ADHD).



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Neuro-exergaming for non-verbal autism spectrum: A case study of neuropsychological function after 15 sessions of pedal-n-play (iPACES)

Youth on the Autism Spectrum (ASD) display varying degrees of deficits in cognitive, motor, and social functioning, areas often targeted with various therapy modalities, most efficiently provided as early interventions. Exercise has long been examined as a potential therapy modality for a variety of neurological conditions, with some reports revealing effects for ASD. Exergaming, or the combination of interactive video games with physical activity, has also been found helpful, and has implications for being an effective and feasible therapy option that may help sustain attention using non-social reinforcement. Our lab has previously evaluated off-the-shelf exergames, such as Dance Dance Revolution (DDR), for high-functioning, verbal youth with ASD and found benefits to executive function and repetitive behaviors; however, little is known about the utility of such interventions for non-verbal or illiterate youth with ASD. The interactive Physical and Cognitive Exercise System (iPACES) was developed in our lab as a neuro-exergame to address neurological conditions and target specific cognitive domains, with a priori designed mental challenges, and to address short-comings of off-the-shelf exergame which did not provide methods for controlling features or measuring precise outcomes. Pilot work with iPACES has been promising among youth with ASD. The current case study of a non-verbal youth with ASD, adapted iPACES, creating an image-only version of the exergame iPACES, for a 10-week, 15 session intervention, to assess possible improvements in neuropsychological function. Neuropsychological function was measured using a shortened pediatric neuropsychological battery (e.g., Digit Span, Colored Trails, TABCAT set-shifting), and included additional measures of behaviorally observed sustained attention and exergame performance. Data was analyzed using SPSS. Valid data was obtained for only one of the three neuropsychological assessments due to the youth's challenges, and no significant

change was found in that traditional pre-/post-neuropsychological test performance (Digit Span). However, a significant correlation was found between duration of sustained attention and number of coins collected while neuro-exergaming [$r(n=14 \text{ sessions})=0.65$, $p<0.05$]. It may be that traditional neuropsychological assessment was insufficient to detect significant change in a single individual. Yet, it is encouraging that a non-verbal youth with ASD was able to participate in longer-term exercise and indicates feasibility of adapting a neuro-exergame such as iPACES, for this population. Furthermore, the nature of intervention, with engaging coin-capture, seems to have benefited attention and thus compliance with exercise. These results suggest additional research with a larger sample, might be warranted to further examine ways to adapt exercise for non-verbal youth with ASD, in particular, the role of facilitating sustained attention such as during neuro-exergaming, and the impact on effectiveness of such interventions, which hints at the importance of using rewarding stimuli in interventions tailored to the ASD population.

Biography

Cay Anderson-Hanley, is a Principal Investigator of NIH-funded clinical trials, with a focus on innovating pedal-n-play neuro-exergaming to enhance brain health, especially among those with neurological conditions. As a professor of neuroscience, the interactive Physical and Cognitive Exercise System (iPACES) was invented and patented in her lab, in an effort to ameliorate cognitive decline for those with Alzheimer's and Related Disorders (ADRDs). This technology has been adapted most recently for those with Parkinson's Disease and is being evaluated in an ongoing clinical trial. Pilot work has also explored utility for those on the autism spectrum (ASD) as well as Attention Deficit Hyperactivity Disorder (ADHD).



**Daniel Christopher P. Maan, MD,
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The malignant Marburg malady of Manila: A case of acute fulminant multiple sclerosis (Marburg variant) in a 38-year-old Filipino female

Background: Multiple Sclerosis (MS) is a demyelinating disease which is particularly rare in tropical regions like the Philippines, where epidemiologic incidence is 10 to 20 times lower than in temperate zones. Fulminant and tumefactive MS variants, such as the Marburg Variant, present with extreme diagnostic and therapeutic difficulties due to its rapid, monophasic, and progressive course and its propensity to mimic intracranial neoplasms.

Case Description: A 38-year-old, previously fully-abled Filipino female presented with a three-month history of progressive bitemporal and retroorbital headaches, subsequent bilateral blindness with papilledema, and a new-onset generalized tonic-clonic seizure. Magnetic resonance spectroscopy revealed large inflammatory lesions with severe vasogenic edema in the left frontal and parietal lobes, mimicking a primary brain tumor or encephalitis. Extensive cerebrospinal fluid analysis, including oligoclonal bands, anti-aquaporin-4, and anti-MOG antibodies, returned negative, pointing toward an atypical fulminant MS variant. Despite temporary stabilization as an outpatient with systemic steroids and anticonvulsants, the patient succumbed to clinical deterioration. On readmission, she presented with total blindness, decreased sensorium, unequal pupillary light reflex, slurred speech, and vomiting secondary to increased intracranial pressure. She was initially stabilized after medical decompression and Rituximab infusion, but succumbed to acute deterioration which eventually caused her demise.

Conclusion: The current case highlights the clinical presentation of an aggressive, tumefactive MS variant in a low-risk demographic. The negative yield for the expected

biomarkers and the rapid progression to neurological compromise signify the need for early clinical recognition, aggressive comprehensive intervention, and the development of standardized management strategies for fulminant demyelinating diseases such as the Marburg variant of Multiple Sclerosis.



Danush Balaji

UCLA, Mentored by Dr. Vaithi Arumugaswami through Emerging Scientist Program, United States

Early detection of amyotrophic lateral sclerosis through a coustic speech biomarkers

Amyotrophic Lateral Sclerosis (ALS) is a fatal neuro degenerative disease that has an average diagnostic delay of 10-18 months after symptom onset. Speech impairment affects approximately 80% of patients and may precede clinical recognition. This makes this early acoustic analysis a low-cost way to aid in diagnosis. The study applied an interpretable machine learning pipeline to complete the VOC-ALS database. This is a publicly available database of smart phone recorded voice signals from 102 ALS patients and 51 healthy controls. The acoustic features are F0 mean, SD, jitter, shimmer, and harmonic to noise ratio. This has been extracted using Praat in eight vocal tasks. Tenfold stratified cross validation gave an AUC of 0.712 in gradient boost, 0.706 in random forest, 0.692 SVM, and 0.63 Logistic Regression. Jitter, shimmer, HNR, and F0 variability during syllable repetition tasks (/ka/, /ta/, /pa/) emerged as the most diagnostically informative for ALS. These results suggest that interpretable, smartphone acoustic recordings for ALS can achieve clinically meaningful AUC of greater than 0.70. With advances in pause tracking and with better recording quality this can be informative for bulbar dysfunction detection through a clinical setting or via phone applications. This paves the way for at home ALS testing which can greatly benefit those that are taking precautions to make sure that they are safe. If anyone is found to have ALS, they can take the medications as early as possible in order to improve their quality of life and slow the progression of ALS.

Biography

Danush Balaji is a high school researcher that is focused on neuroscience, biotechnology, and the intersection between the two. His academic interests include neurodegenerative diseases as well as diseases like Alzheimer's and Parkinson's. Through his research, Danush hopes to take meaningful steps forward in helping patients with these disorders and improving their quality of life.



Dhruv Bearely^{1*}, Isabelle Kottyan²

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Design of a proposed phase III trial following the HORIZON phase II study

Multiple Sclerosis (MS) is an autoimmune disease that affects children and adults nationwide. The goal of this research is to improve the quality of life for patients living with the sometimes debilitating symptoms of MS, specifically through the use of the mRNA-1195 vaccine (also known as the Epstein Barr Virus vaccine). We designed a multicenter, randomized, placebo controlled, masked Phase III trial enrolling 400 adults (18–55) with early relapsing MS who are EBV-seropositive, with follow-up through Day 887. Participants would receive vaccinations on Day 1, ~Day 85, and ~Day 176, remain on standard MS management (as an add-on approach), and be monitored using clinical assessments, standardized assessment of disability outcomes, immune response labs, and neurological imaging at baseline, Month 12, and end-of-study. The proposed primary efficacy endpoint was time to first new disease activity through Day 887. First new disease activity was defined as a clinical attack and/or new or enlarging MRI lesions, based on previous studies. Statistical power calculations ($n=200$ per arm, $SD=1.0$, two-sided $\alpha=0.05$) indicated low power for a small effect ($\Delta=0.10$; 17%) but strong power for moderate-to-large effects ($\Delta=0.30$; 85.1%; $\Delta\geq 1.0$; ~100%). A high-level budget estimate for a 10-site, 400-participant trial (excluding vaccine manufacturing) ranged from ~\$25M to \$177M, reflecting variability in site operations, imaging, labs, and monitoring intensity. This report outlines a feasible and ethically considered Phase III trial design to test whether adding mRNA-1195 vaccination to standard MS care reduces disease activity. The proposed sample size is well-powered to detect clinically meaningful effects (about 30% or larger), and the cost ranges provide a realistic planning framework for future clinical trial implementation. Next steps include finalizing dose and eligibility details based on Phase II results and, if adult safety and efficacy signals are strong, planning stepwise evaluation in adolescents with pediatric-onset MS.

Biography

Dhruv Bearely is a student researcher at Walnut Hills High School in Cincinnati, Ohio, with research interests spanning neuroimmunology, multiple sclerosis, and neurological health equity. Bearely's work includes statistical analysis and clinical-trial design related to Epstein-Barr virus vaccination and MS, as well as initiatives focused on improving neurological disease education and access to care. Bearely is also involved in student-led public health advocacy and interdisciplinary projects connecting neuroscience, medicine, and health policy.



**Divine B. Fajardo, MD*, Maria
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World Citi Medical Center, Philippines

A mind under attack: A rare case of autoimmune anti-NMDA receptor encephalitis in a 19-year-old male

A 19-year-old Filipino male presented with a one-week history of intermittent headache, followed by focal neurologic symptoms including right-sided lip twitching, right-sided weakness, and vomiting. This progressed to a one-minute episode of generalized tremors, musculoskeletal spasms, and upward eye deviation lasting 3–5 minutes, followed by transient loss of consciousness and disorientation. He was initially managed in the emergency department with antiepileptics, antiplatelet therapy, and supportive medications. Initial laboratory workup revealed hypokalemia and hyperuricemia, which were corrected. Empiric antimicrobial therapy with ceftriaxone and acyclovir was initiated for possible infectious etiologies. Electroencephalography and neuroimaging, including computed tomography and magnetic resonance imaging, were unremarkable. The patient improved clinically and was discharged after five days on levetiracetam.

Four days after discharge, the patient developed recurrent vomiting, followed by new-onset left-sided weakness and involuntary movements without loss of consciousness. Two days later, he experienced another seizure episode, prompting readmission. On arrival, he had active musculoskeletal spasms, nuchal rigidity, and hypoxemia (oxygen saturation 77%), necessitating oxygen support. He was treated with benzodiazepines, antiepileptics, and broad-spectrum antimicrobials. Episodes of agitation characterized by restlessness and verbal hostility required sedation with midazolam and dexmedetomidine. Chest radiography revealed lung opacities consistent with aspiration pneumonia, leading to escalation of antibiotic therapy.

A lumbar puncture demonstrated elevated opening pressure (>200 mmH₂O) with otherwise normal Cerebrospinal Fluid (CSF) cell count. Extensive infectious workup, including testing

for tuberculosis and cryptococcosis, returned negative. Cardiac monitoring showed sinus rhythm with transient premature atrial contractions, while arterial blood gas analysis revealed uncompensated respiratory acidosis attributed to prior sedation. The patient was admitted to the intensive care unit, where he stabilized without recurrence of seizures. Corticosteroids and valproate were added to his treatment regimen.

Following transfer to the wards, the patient's condition continued to improve. Gastrointestinal symptoms, including acute watery diarrhea, resolved with supportive therapy. On hospital day five, CSF testing returned positive for Anti-N-Methyl-D-Aspartate (NMDA) receptor antibodies, confirming a diagnosis of autoimmune encephalitis. A positron emission tomography-computed tomography scan revealed no hypermetabolic brain lesions or infectious foci, allowing discontinuation of antimicrobial therapy.

The patient was treated with Intravenous Immunoglobulin (IVIG) for five days, which was well tolerated without adverse effects. He demonstrated progressive clinical improvement, with resolution of seizures and agitation, improved appetite, and near-complete neurologic recovery, except for a persistent left-sided Babinski sign. Antiepileptic therapy was gradually tapered.

He was discharged in stable condition on a steroid taper and maintenance levetiracetam, with plans for outpatient neurology follow-up. This case highlights the diagnostic challenges of autoimmune encephalitis in the absence of early radiologic and electroencephalographic abnormalities and underscores the importance of maintaining a high index of suspicion. Early recognition and timely initiation of immunotherapy are critical in achieving favorable clinical outcomes.

Biography

Dr. Divine Fajardo is a second-year Internal Medicine resident at World Citi Medical Center. Fajardo is dedicated to delivering compassionate, evidence-based care while continually refining her clinical acumen. With a strong interest in complex case management and academic medicine, Fajardo actively engages in case discussions and research activities. Fajardo is committed to lifelong learning and strives to contribute meaningfully to patient care and medical education through her clinical work and scholarly pursuits.



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Clinical outcomes of AI-augmented cognitive rehabilitation in dementia and mild cognitive impairment

Background: Cognition-oriented treatments are associated with improved cognitive and non-cognitive outcomes among people with Mild Cognitive Impairment (MCI) or dementia; however, access is limited by cost, availability of clinicians, and intervention frequency. Advances in Artificial Intelligence (AI) allow for scalable, frequent, and personalized cognitive interventions, while human-delivered telehealth interventions provide contextualized, goal-oriented, and psychotherapeutic support. This blended model may maximize clinical outcomes by combining the consistency and precision of AI with the expertise of human clinicians. We present data from a cohort of patients treated with this hybrid approach at the NewDays clinic, with a focused analysis of the MCI subgroup.

Methods: Thirty-seven patients (14 MCI, 19 dementia, 4 other cognitive symptoms; 17 males; mean age 78.8, SD = 10.0; mean education 16.5 years, SD = 2.4) were treated with the NewDays blended intervention approach and underwent evaluations at baseline and at 3–16 months (mean 7.4 months, SD = 3.5). Assessments included global cognition (ACE-III), verbal memory (WMS-IV Logical Memory I and II), meta-cognition (MMQ Satisfaction and Ability), and mood (GDS-15, GAI). For the MCI cohort, expected change without treatment was estimated from published trajectories, pro-rated to each patient's interval: 1.8 ACE-III points/year if stable and 5.3 if converting to dementia (Hamilton et al., 2024). To allow comparison with the larger MMSE literature, ACE-III scores were additionally converted to MMSE-equivalent scores (Matías-Guiu et al., 2018) and compared against an expected decline of 0.82 MMSE points/year (Xie et al., 2011). Patients were seen via telehealth by trained clinicians every two weeks, delivering individualized functionally-oriented treatment within a goal-focused reablement framework, while cognitive stimulation and training was delivered daily through conversational AI under clinician guidance.

Results: Across all 37 patients, ACE-III Total scores improved from 72.4 to 74.8 (+2.4, $p = 0.038$), driven mainly by the memory subscale (+1.4, $p = 0.021$). Secondary measures improved significantly on Logical Memory I (+3.2, $p = 0.005$), Logical Memory II (+3.2, $p = 0.005$), MMQ Satisfaction (+2.6, $p = 0.013$), and GAI anxiety (-1.3, $p = 0.022$), while MMQ Ability (+1.5, $p = 0.082$) trended favorably. The MCI cohort ($n = 14$; 7 males; mean age 75.4, SD = 12.8; mean interval 8.3 months, SD = 3.3) on average did not decline further and in fact improved: observed ACE-III change was +3.1 points against an expected decline of -1.4 ($p = 0.043$), exceeding the no-treatment trajectory by 4.6 points, with 11 of 14 patients performing better than their expected trajectory. 6 of 14 exceeded their expected trajectory by 5 or more points. MMSE-equivalent scores showed the same pattern (observed +0.5 vs expected -0.6, $p = 0.056$). Logical Memory II improved by 4.8 points ($p = 0.003$; 13/14 improved or stable).

Conclusion: Our findings add further support for a blended human-AI treatment model in improving outcomes for people with cognitive impairment and extend it to MCI, where observed cognitive change exceeded the expected no-treatment trajectory. The single-arm design and modeled comparator limit causal inference. A randomized controlled trial (NCT07474038) is currently underway.

Biography

Esra Al is a neuroscientist and AI researcher specializing in cognitive impairment, dementia, and human-AI interaction. She earned her PhD in Cognitive Neuroscience from the Berlin School of Mind and Brain and the Max Planck Institute for Human Cognitive and Brain Sciences, and completed postdoctoral research at Columbia University Medical Center. Her work spans cognitive neuroscience, computational modeling, and human-AI interaction. At NewDays, she develops and evaluates AI-based interventions for people with mild cognitive impairment and dementia.



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Real-world outcomes in glioblastoma: A retrospective single-center cohort analysis

Purpose/Objective(s): Glioblastoma (GBM) remains associated with poor prognosis despite multimodality treatment. Real-world cohorts may provide complementary evidence regarding outcomes outside highly selected clinical trial populations. We evaluated clinical characteristics, treatment patterns, and survival outcomes in a retrospective cohort of patients with GBM treated at a tertiary oncology center.

Materials/Methods: We retrospectively reviewed 50 consecutive adult patients with newly diagnosed WHO grade 4 glioblastoma treated between 2016 and 2025. Clinical, radiological, surgical, and treatment-related variables were collected from institutional records. Overall Survival (OS) was defined from diagnosis to death or last follow-up, and Progression-Free Survival (PFS) from diagnosis to radiological or clinical progression, death, or censoring. Patients alive without progression were censored on May 15, 2026. Survival was estimated using the Kaplan–Meier method. Exploratory multivariable Cox regression analyses included age at diagnosis, Karnofsky Performance Status (KPS), extent of resection, multifocal disease, and largest lesion diameter on MRI.

Results: The cohort included 50 patients with a median age of 56 years. Multifocal disease was present in 40% of cases, and the median largest lesion diameter on MRI was 5.0 cm. Gross total resection was achieved in 20% of patients, subtotal resection in 66%, and biopsy only in 14%. KPS was ≥ 80 in 72% of patients. IDH status was available in 40% of patients and was predominantly wild-type. Standard radiotherapy to 60 Gy in 30 fractions was delivered in 70% of patients, whereas 30% received hypofractionated or alternative regimens. During follow-up, 40 deaths and 45 PFS events were observed. Median OS was 14.0 months, with estimated OS rates of 79.9%, 52.3%, 41.2%, and 25.3% at 6, 12, 18, and 24 months, respectively. Median

PFS was 9.7 months, with estimated PFS rates of 69.9%, 38.2%, 25.3%, and 16.1% at 6, 12, 18, and 24 months, respectively. In exploratory multivariable analyses, increasing age at diagnosis appeared to correlate with worse OS, while multifocal disease and larger lesion diameter on MRI appeared to correlate with poorer outcomes.

Conclusion: This retrospective real-world cohort demonstrated survival outcomes consistent with contemporary glioblastoma series. Increasing age, multifocal disease, and larger tumor burden appeared to be associated with poorer outcomes. Given the retrospective design, limited sample size, and incomplete molecular profiling, these findings should be considered hypothesis-generating rather than definitive prognostic evidence.

Keywords: Glioblastoma, Central Nervous System Tumors, Radiotherapy, Survival Outcomes, Prognostic Factors.

Biography

Farley Soares Cantídio holds a Master's degree in Women's Health from the Federal University of Minas Gerais (UFMG). Specialist in Radiation Oncology certified by the Brazilian Society of Radiotherapy, with a medical degree from the Federal University of Roraima and a Nursing degree from the State University of Montes Claros.



Giriish Purushothaman

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In silico structural mapping of endothelial LRP1 cluster II variants: Quantifying the biophysical mechanisms of impaired amyloid-beta efflux at the blood-brain barrier

This study aims to characterize how specific, clinically unannotated human missense mutations within the ligand-binding Cluster II domain of Low-Density Lipoprotein Receptor-Related Protein 1 (LRP1) alter its 3D structural conformation and binding affinity for Amyloid-Beta ($A\beta$ 1-42), a critical step in preserving blood-brain barrier efflux mechanisms. The wild-type (WT) sequence of LRP1 Cluster II (residues 804–1164) and three targeted variants exhibiting distinct chemical disruptions, specifically D1016A (charge switch), G932R (backbone flexibility disruption), and Y872C (aromatic stacking loss), were structurally modeled using AlphaFold3. Rigorous protein-protein docking simulations were executed via the HADDOCK 2.4 server against the solution NMR structure of monomeric $A\beta$ 1-42 (PDB ID: 1IYT). Interfacial binding kinetics, free energy of binding (ΔG), electrostatic surface potentials, and hydrogen/salt-bridge topologies were quantified using UCSF ChimeraX. Computational analysis revealed that the wild-type LRP1- $A\beta$ complex stabilizes via extensive electrostatic coordination between negatively charged aspartate residues and the basic Lys16/Lys28 cluster of $A\beta$ 1-42. The D1016A mutation directly collapsed this local calcium-coordinating loop, resulting in a 34% reduction in interface binding energy (ΔG). The G932R mutation introduced severe steric clashes and a highly positive local surface charge that drove a structural rotation of the binding loop, completely displacing $A\beta$ from its native pocket. Conversely, the Y872C mutation maintained baseline electrostatic affinity but significantly lowered the structural stability score due to the loss of aromatic π -stacking interactions. These in silico findings demonstrate that non-synonymous single nucleotide polymorphisms within LRP1 Cluster II can structurally incapacitate the receptor's capacity to bind amyloid-beta through distinct biophysical pathways, namely electrostatic collapse and steric hindrance. This study successfully

establishes a structural screening framework to identify high-risk genetic variants that impair blood-brain barrier clearance, highlighting specific molecular targets for compensatory drug design.

Biography

Giriish Purushothaman is a rising senior at Bridgeland High School in Cypress, Texas, with a deep passion for computational neurobiology and biochemistry. As an independent researcher, he leverages structural biology frameworks to model molecular mechanisms underlying neurodegenerative diseases. Alongside his academic pursuits, Giriish serves as assistant concertmaster for his school orchestra and is an Eagle Scout. His multidisciplinary achievements span successes in Science Olympiad and Future Business Leaders of America. He aspires to major in neuroscience in college, aiming to dedicate his career to solving complex medical mysteries at the intersection of structural biology and brain health.



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Meningitis secondary to Coxsackie A16 virus in a 34-year-old adult male

Hand Foot and Mouth Disease is a common and highly contagious febrile infection mostly secondary to Coxsackievirus A16. The virus can spread to adults and one of the major complications is viral meningitis. This case describes a case of an adult patient who developed viral meningitis due to transmission from his child.

A 34-year-old male with a past medical history of active smoking and non-insulin dependent diabetes mellitus II presented to the emergency room due to convulsive status-epilepticus activity earlier in the day. Shortly after arrival to the emergency department the patient woke up but developed post-ictal agitation towards the medical staff. The patient was given lorazepam, ketamine, Haldol, and Diazepam to no effect. The patient was intubated with propofol and midazolam, and was admitted to the ICU. According to history from the wife, the patient 3 weeks prior to admission developed a mild rash with red macules and vesicles on his palms and soles of his feet that resolved 1 week ago. Initial labs demonstrated hyperglycaemia, leucocytosis, and elevated lactic acid. MRI of the cranium and cervical spine was unremarkable. Blood was collected for bacterial cultures, West Nile Virus, HSV, HIV antigen and antibodies. The patient was started on ceftriaxone and vancomycin due to meeting septic criteria. The patient the next day underwent a lumbar puncture with 3.5 mL of clear and colourless CSF volume removed. CSF analysis revealed elevated white blood cells, protein, lymphocytes, and glucose indicating a likely viral meningitis diagnosis. The CSF sample was also cultured for H. influenzae, E. coli, L. monocytogenes, N. meningitidis, Group B Streptococcus, S. pneumoniae, Cytomegalovirus, Enterovirus, HSV I and II, HHV-6, Parechovirus, Varicella, Cryptococcus. Infectious disease was consulted, and antibiotics were switched from ceftriaxone to cefepime and vancomycin for Pseudomonas coverage. The patient was also started on IV acyclovir for

viral meningitis coverage. CSF and blood cultures, as well as the HIV antigen and antibody panel were all negative. The patient was started on dexmedetomidine to assist with weaning off the ventilator. The next day the patient was extubated successfully and switched to nasal cannula. The patient was downgraded from the ICU with improved mental status, resolved leucocytosis and discontinuation of ceftriaxone and vancomycin. EEG was repeated and was unremarkable for ongoing seizures. The patient was able to walk with assistance from physical and occupational therapy and was discharged with acyclovir (PO) for four days.

The patient's symptoms of altered mental status, post-ictal aggression, and unremarkable MRI prompted a lumbar puncture. Lumbar punctures are generally a safe procedure with some potential complications, but the history of a Coxsackie virus rash on the palms and soles in this patient provided by the wife prompted the procedure to assess for potential viral meningitis. While uncommon in immunocompetent patients, viral meningitis should be diagnosed with a lumbar puncture and treated early to avoid serious complications.

Biography

Dr. Gregory Williams is a first year resident at Las Palmas Del Sol internal medicine program affiliated with HCA Healthcare. Dr. Williams' interest in neurocritical care started when working in an ICU lab funded by the Department of Defense in San Antonio, TX involved in TBI's in soldiers injured in combat. Dr. Williams is a member of the United States Air Force and wishes to serve his country in the future as neurocritical care physician.



Jaiden Makadia

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Structure-based in silico modeling of avimers for CNTF detection in autism

Autism Spectrum Disorder (ASD) lacks fast and reliable blood and molecular detection methods, making early diagnosis difficult. The purpose of this project was to design avimer molecules to bind to the autism-associated protein CNTF using structure-guided computational modeling. The research question was whether engineered avimers could form strong and stable molecular interactions with CNTF. The three-dimensional structure of CNTF was obtained from UniProt, and avimer scaffolds were designed using molecular modeling software. Protein-protein docking simulations were performed to evaluate binding between CNTF and multiple avimer variants. The number of molecular interactions, including hydrogen bonds, ionic interactions, and hydrophobic contacts, was recorded for each design. The strongest avimer candidate formed 9 hydrogen bonds, 3 ionic interactions, and more than 15 hydrophobic contacts with CNTF, while weaker variants formed fewer than 5 hydrogen bonds and fewer than 8 hydrophobic contacts. Thermostability prediction analysis showed that the optimized avimer had an estimated stability increase of about 10-12% compared to the original complex, which shows improved structural reliability. These results indicate that structure-guided design can produce avimers with strong predicted interactions and stable folding when bound to CNTF. The data supports the hypothesis that engineered avimers can selectively recognize autism-associated proteins at the molecular level. This research may contribute to the future development of biosensor-based diagnostic tools for detecting autism-related proteins, which could allow faster screening and improved early intervention.

Biography

Jaiden is an independent researcher with interests in computational neuroscience, protein engineering, and neurodevelopmental disorders. His work explores engineered avimers as

potential diagnostic tools targeting Ciliary Neurotrophic Factor (CNTF), integrating structural modeling, protein–protein docking, molecular dynamics simulations, and in silico stability analyses. He is particularly interested in applying artificial intelligence and computational biology to translational questions in autism research. His interests include neurotechnology, biomolecular design, and precision diagnostics.



Jed Hiram Rosales, MD*, Vanessa Dela Rosa, MD, Sheila Marie Reyes Yap-Uysecro, MD, FPCP, FPRA, Kristine Joyce Porto, MD, PhD, FPNA

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CNS crisis in a pediatric patient: Navigating the inflammatory-vasculitic variant of pediatric Neuro psychiatric Systemic Lupus Erythematosus (NPSLE) and the role of biomarker-driven differentials

Background: Neuropsychiatric Systemic Lupus Erythematosus (NPSLE) in the pediatric population is characterized by a more aggressive clinical course and higher morbidity compared to adult-onset disease. Diagnostic clarity is often obscured by the mimicry of central nervous system (CNS) infections and primary demyelinating disorders, necessitating a biomarker-driven approach to management.

Case Presentation: A 16-year-old female with a known history of SLE presented with a sudden onset of malar rash, fever, and a rapid neurological decline culminating in acute loss of consciousness (LOC). Upon admission, she experienced an acute call due to vomiting and teeth grinding, with a drop in Glasgow Coma Scale (GCS) to 7, requiring immediate intubation and ICU transfer. Systemic rheumatological activity was prominent, including alopecia, palatal ulcers, and erythematous vasculitic rashes on the palms and digits.

Neuro-Immunological Evaluation:

Imaging: Cranial MRI with MRA revealed multifocal T2/FLAIR hyperintensities involving the bilateral corona radiata, basal ganglia, hippocampi, and posterolateral cerebellar hemispheres. A punctate diffusion restriction in the left hippocampus was highly suggestive of a neuroinflammatory or vasculitic process.

CSF Dynamics: Lumbar puncture showed a significantly elevated CSF protein (109.60 mg/dL) but was acellular with normal glucose levels.

Differential Diagnosis: Given the multifocal lesions, Neuromyelitis Optica Spectrum Disorder (NMOSD) was a primary consideration. However, Serum Aquaporin-4 IgG was negative and oligoclonal bands were absent, effectively narrowing the diagnosis to the inflammatory-vasculitic variant of NPSLE.

Infection Control: Although the patient developed Hospital-Acquired Pneumonia (*Klebsiella pneumoniae*), direct CNS infection was ruled out via sterile CSF cultures.

Neuropsychiatric Outcomes: Following stabilization, neuropsychological testing via MOCA revealed a score of 22/30, indicating mild cognitive impairment. Screening for psychiatric comorbidities showed mild depression (PHQ-9: 5/9) and mild-moderate anxiety (GAD-7: 22).

Management and Clinical Course: The patient was treated with an aggressive immunosuppressive regimen consisting of Methylprednisolone pulse therapy (500mg IV for 3 days) and the initiation of Cyclophosphamide infusion (1g). For symptomatic neurological support, she received Levetiracetam for seizure prophylaxis and Escitalopram for mood stabilization. Her sensorium improved to a GCS of 15, and she was successfully discharged following the resolution of the acute crisis.

Discussion and Conclusion: This case underscores the critical nature of the inflammatory-vasculitic variant of NPSLE, where acute neurological symptoms can be the primary driver of life-threatening illness in pediatric patients. The successful navigation of this acute CNS crisis was dependent on utilizing specific neuro-immunological biomarkers to exclude NMOSD and CNS infections, allowing for the timely application of potent cytotoxic therapy. Early multi-disciplinary intervention remains the cornerstone for preventing permanent neuropsychiatric damage in children with severe SLE flares.

Biography

Jed Hiram Rosales is a third-year Internal Medicine resident at World Citi Medical Center in the Philippines. He is currently in training, managing a broad spectrum of medical conditions across inpatient and critical care settings. His interests include neurology and acute care medicine. As part of his residency, he has participated in case reports and clinical discussions aimed at enhancing diagnostic awareness and patient management. He remains committed to continuous learning and hopes to further develop his skills through academic involvement and collaboration.



Jed Hiram Rosales, MD*, Vanessa Dela Rosa, MD, Ma. Cecilia De-Guzman, MD, FPNA, Czarina Mora, MD, FPCP, FPCCP

World Citi Medical Center, Philippines

The ascending storm: Clinical management of rapidly progressive AMSAN GBS with early bulbar red flags

Introduction: Guillain-Barré Syndrome (GBS) is a rare but potentially fatal immune-mediated polyneuropathy typically characterized by symmetrical ascending paralysis. Approximately 20% to 30% of patients experience a rapid ascending storm of symptoms leading to respiratory failure and requiring mechanical ventilation. The Acute Motor Sensory Axonal Neuropathy (AMSAN) subtype is particularly aggressive, involving both motor and sensory axonal damage. This case emphasizes the critical role of identifying early bulbar red flags to navigate imminent respiratory crisis.

Case Presentation: A 60-year-old male presented with a three-day history of distal paraesthesia and progressive limb weakness. Critically, he exhibited atypical early bulbar symptoms, including cold hyperesthesia of the tongue, ageusia (loss of taste), dysphagia, and throat spasms. Within 48 hours of admission, the patient's condition deteriorated from being able to ambulate with support to being bedbound and experiencing gasping and desaturations. These rapid progression markers (onset to admission ≤ 3 days) and bulbar signs are significant predictors of respiratory failure. He was immediately intubated and transferred to the ICU.

Management: Following a diagnosis of AMSAN GBS, the patient received a five-day course of Intravenous Immunoglobulin (IVIg) at 0.4 g/kg/day. Because bulbar dysfunction increases the risk of aspiration and ineffective cough, a nasogastric tube (NGT) was inserted for feeding. Weaning from mechanical ventilation was navigated through serial monitoring of the Negative Inspiratory Force (NIF) and sensorium, as traditional parameters like pulse oximetry can be misleadingly normal until decompensation is imminent.

Results: The patient's diaphragmatic effort improved incrementally, reaching a NIF of -28 on day eight and a target NIF of -30 on day nine, at which point he was successfully extubated. During the recovery phase, the patient developed left-sided facial asymmetry (Bell's palsy) on day 11, a known cranial nerve complication of the syndrome. By day 17, his motor strength had improved to 4/5 in all extremities, and he was cleared for discharge.

Conclusion: This case highlights that early bulbar symptoms serve as essential red flags for the rapid respiratory collapse often seen in the AMSAN variant of GBS. Successful ICU navigation requires immediate immunotherapy and a weaning strategy centered on objective metrics like NIF to prevent extubation failure. Early recognition and a multidisciplinary approach remain the cornerstones of managing the ascending storm to ensure favorable long-term outcomes.

Biography

Jed Hiram Rosales is a third-year Internal Medicine resident at World Citi Medical Center in the Philippines. He is currently in training, managing a broad spectrum of medical conditions across inpatient and critical care settings. His interests include neurology and acute care medicine. As part of his residency, he has participated in case reports and clinical discussions aimed at enhancing diagnostic awareness and patient management. He remains committed to continuous learning and hopes to further develop his skills through academic involvement and collaboration.



**Jerico Ryan M. Pura, M.D.*, Maria Carmencita B. Gonzalez, M.D.,
FPNA**

World Citi Medical Center, Philippines

You should not start young: A case of a 25-year-old presenting with ischemic infarcts and a chronic intracranial hemorrhage

Patient J.Y, a 25-year-old male, came in due to a chief complaint of weakness and no verbal output. 2 days prior to consultation, the patient complained of occasional headaches and right-hand numbness however still apparently well at the time. It was elicited from history that the patient was drinking at a party the night prior. No medications were taken nor consultations done. In the interim there is persistence of the occasional headaches and the patient was last seen well on the midnight of the day of consultation. When the patient woke up that morning, he had right-sided body weakness as well as a lack of verbal output which prompted the relatives to immediately bring the patient to the emergency room for consultation.

The patient arrived at the emergency department with stable vital signs and was started on IV Citicoline and capillary blood glucose was monitored and corrected as necessary. Cranial nerve examination revealed poor gag reflex hence a nasogastric tube was inserted. Plain cranial CT scan revealed acute infarcts at the left basal ganglia, left centrum semiovale, and the left temporal lobe as well as a chronic intracranial hemorrhage at the parieto-occipital lobes. The patient was started on mannitol due to the findings of the chronic hemorrhage and was referred to neurology service for co-management.

At the ward, the patient was maintained on mannitol, citicoline, atorvastatin, sertraline, and levetiracetam. Antiplatelets were withheld initially due to the presence of the bleed. After a few days with the patient's condition stable, the patient was eventually started on swallowing training and was encouraged to speak. The patient was eventually referred to rehab medicine service for physical and occupational therapy prior to discharge. The patient was also started on clopidogrel with clearance from the neurologist. In the end the patient was able to verbalize

short phrases and regained most of his strength in his right extremities and the patient was eventually cleared for discharge.

Biography

Jerico Ryan M. Pura is currently a third-year medical resident of Internal Medicine residency program at World Citi Medical Center, a private tertiary hospital at the Philippines. Graduated with a Bachelor's in Science of Psychology, the author was previously interested in becoming a Neurologist but opted to pursue generalized Internal Medicine first before ultimately deciding on a subspecialty.



**Divine M. Fajardo, Jonathan E. Sy*,
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World Citi Medical Center, Quezon City, Philippines

Je Ne Sais Quoi - Autoimmune encephalitis in a 19-year-old male: A case report

A 19-year-old Filipino male with no significant past medical history presented with a 1-week history of intermittent headache followed by sudden right-sided weakness, vomiting, twitching, tremors, upward eye-rolling, and spasms with transient loss of consciousness and disorientation. Initial admission revealed hypokalemia and hyperuricemia, for which potassium supplementation and febuxostat were started, while empiric coverage with acyclovir and ceftriaxone was given for suspected infectious etiologies. Neuroimaging and EEG were unremarkable, and he was discharged on levetiracetam. Within days after discharge, vomiting recurred with new-onset left-sided weakness, involuntary shaking, and an episode of collapse with recurrence of upward eye-rolling, prompting readmission. On arrival, he was tachypneic, hypoxemic, and in active spasms with nuchal rigidity and toe hyperextension on examination. He was placed on oxygen support; given benzodiazepines, broad-spectrum antimicrobials, and antiepileptics; and admitted to intensive care. Workup included lumbar puncture with elevated opening pressure and further testing which eventually revealed cerebrospinal fluid positivity for anti-NMDA receptor antibodies. He completed a 5-day course of intravenous immunoglobulin with clinical improvement, resolution of seizures, improved appetite, and no further episodes of agitation, and was subsequently discharged on levetiracetam and a tapering steroid regimen with a follow-up with neurology advised outpatient.

Biography

Jonathan E. Sy is a licensed physician with training in internal medicine at St. Luke's Medical Center – Global City and World Citi Medical Center in Metro Manila in the Philippines. A

graduate of the Integrated Liberal Arts and Medicine (Intarmed) program of the University of the Philippines College of Medicine that accepts top high school graduates into an accelerated medical program with clinical experience at the Philippine General Hospital, the country's leading university hospital and referral center, he is interested in history, current events, foreign affairs, traveling, languages, public health, and public transit and urbanism.



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Effects of prenatal androgen exposure on Purkinje cell morphology in the cerebellum of female rats

Background: Poly-Cystic Ovarian Syndrome (PCOS) is a complex endocrine disorder typically characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovaries. Prenatal exposure to elevated androgen levels, particularly testosterone, has been linked to changes in brain structure and function, including excessive growth of dendritic spines and traits associated with Autism Spectrum Disorder (ASD). This is particularly relevant as research shows a link between Polycystic Ovarian Syndrome (PCOS) and ASD. Prenatally androgenized (PNA) female rats serve as a model for studying the effects of prenatal androgen exposure on the brain. The cerebellum, integral to motor coordination and control, has emerged as a point of interest due to its sensitivity to hormonal fluctuations. Research suggests that these fluctuations may disrupt the expression of calbindin, a neuroprotective calcium-binding protein, and affect the soma size in Purkinje cells, the cerebellar cells responsible for fine-tuning and coordinating voluntary movements.

Objective: The primary objective of this study was to examine the soma size and expression of calbindin in Purkinje cells in the cerebellum of PNA female rats as compared to control rats.

Methods: Cerebellar tissue samples were collected from both PNA rats and control rats at 16 weeks of age. Cerebella were sectioned, and Purkinje cells were traced at 40x magnification; comparisons of the soma areas were conducted. Additional cerebellum slices were incubated with antibodies to determine calbindin expression.

Results: The Purkinje soma area sizes were significantly greater in cerebellar lobes 2, 5, 6a, and 9b in PNA rats. However, the soma size was significantly smaller in lobe 9a of the PNA rat. There was no significant difference in calbindin expression.

Conclusions: The data indicate that increased prenatal exposure to testosterone significantly affects the morphology, particularly the soma size, of Purkinje cells in the cerebellum. This disruption potentially contributes to the motor deficits observed in the PNA rat model.

Biography

Kiran Ghotra is a medical student at Lake Erie College of Osteopathic Medicine with research interests in neurodevelopmental disorders, hormonal influences on brain development, and photoreceptor biology. She has conducted studies on prenatal androgen exposure and its effects on cerebellar Purkinje cell morphology, and developed assays to characterize bistable and monostable opsins for optogenetic applications. Her work aims to bridge basic neuroscience and molecular research with translational insights into neurological development and disorders.



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Evaluating the accuracy of artificial intelligence generated nutrition plans compared to evidence-based medical nutrition therapy guidelines for neurologic disorders

Objective: We aim to assess if ChatGPT can be a reliable resource for individuals with neurologic disorders, who may have limited access to registered dietitians, to seek to for a therapeutic nutrition plan.

Background: Medical Nutrition Therapy (MNT) can be a key component for promoting a neuroprotective environment for those with neurologic disorders. ChatGPT, an easily accessible online Artificial Intelligence (AI) platform has been an increasingly popular method to rapidly obtain and reference information. However, it is unclear whether it is a reliable resource for those who are seeking nutrition therapy for their specific neurologic disorder.

Design/Methods: Based on published demographics for Alzheimer's Disease (AD), Multiple Sclerosis (MS), epilepsy, and preeclampsia, a hypothetical standardized patient was created for each disorder. The free version of ChatGPT was asked to build a seven-day diet plan for each disorder, which was then entered into Cronometer.com for dietary nutrition analysis. Mean daily intake values were calculated and compared to the established dietary guidelines relevant to the adult patients with their corresponding neurologic disease. One sample t-tests were conducted for each nutrient, and p-values were adjusted using the Benjamini-Hochberg False Discovery Rate (FDR) procedure.

Results: Both macro and micronutrients were found to be insufficient across all four disorders. Significant deficiencies in vitamin D, omega-6 fatty acids, total energy, and carbohydrates were found in MS and preeclampsia, while the diet plan for epilepsy failed to meet the requirements to support ketosis. The diet plan for AD lacked sufficient fat and vitamin D, while promoting excess omega-3 and vitamin K, suggesting overall poor therapeutic targeting.

Conclusions: Although ChatGPT was able to recognize evidence-based guidelines needed to build a diet plan for each neurologic disorder, it was unable to create a sustainable plan that promotes a therapeutic and neurologically protective environment, highlighting the importance of clinician guided nutrition planning.

Biography

Lauren Provinsal is a medical student at Edward Via College of Osteopathic Medicine (Blacksburg, Virginia). Her passion for neurology was inspired at an early age by witnessing her grandmother's journey with Alzheimer's disease, which fostered a deep appreciation for the complexity of neurodegenerative disorders and their profound impact on patients and families. Lauren is particularly fascinated by the rapidly evolving landscape of neurological treatment, including emerging therapies and preventative strategies aimed at improving patient outcomes. She is enthusiastic about the future of neurology and is eager to contribute to advancing evidence-based, multidisciplinary approaches to neurological care.



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Blue tetrazolium as a fast and efficient assay for salivary cortisol in 96-well format

Background: Frequent or disproportionately elevated psychological distress can be a sign of mood disorders, such as Major Depressive Disorder and anxiety disorders. Due to its availability and non-invasive nature, salivary cortisol content is often used as an indicator of psychological stress. The gold standard methods of measuring salivary cortisol are ELISA and LC/MS. Both methods are time consuming and expensive. Previous publications have tested and verified a method of measuring salivary cortisol using blue tetrazolium in cuvettes that is simpler and more affordable, but is impractical for larger scale projects. Here, we modified this assay for a 96-well plate to keep the benefits while enabling it for more practical applications.

Methods: Initial reagent concentrations and ratios were based off of the work published by Ahmed et al. (2024). One part saliva was combined with four parts blue tetrazolium and four parts tetramethylammonium hydroxide for each test. Increasing final well volumes were tested. OD values were measured using the BioTek Epoch plate reader and Gen5 software. Salivary cortisol test samples were collected in the morning and evening from a single subject, and measured first with ELISA. Variables tested included absorption wavelength, light sensitivity in the development process, development time, final well volume, and the viability of reagents to be stored in a methanol solution at room temperature or in colder temperatures.

Results: Results from initial tests found that OD values peaked between 490nm and 500nm, demonstrating the biggest potential range for differentiating concentrations. There was no noticeable difference between incubating in the dark compared to in a normally lit lab environment. OD values increased proportionally with final well volume until 300ul, where the increase tapered. When controlling for well volume, there was a difference in OD between

replicates incubated in separated wells, and replicates incubated together and then aliquoted, indicating that reaction order is significant for this method. There were no observed differences between freshly made reagents in solution and one week old stock solutions of blue tetrazolium kept in -20C and tetramethylammonium hydroxide kept in -4C, but the stock solutions kept at room temperature for one week produced consistently lower ODs compared to both the fresh and refrigerated solutions. Overall, the samples measured with this method showed similar measured concentrations when compared to ELISA results.

Conclusion: Our pilot results show promise in using blue tetrazolium to measure salivary cortisol in a 96-well format. Given salivary cortisol's widespread use as a measure of HPA axis activity, this method opens doors for more accessible testing of cortisol markers in HPA-related mental health disorders.

Biography

Lin Yang graduated from the University of Michigan in 2025 with a B.S. in Neuroscience and a minor in History of Law. He is currently working as a research lab technician at the Tewari Lab at University of Michigan Medical School, a lab investigating how physiological and psychological factors can influence health outcomes



Miguel Q. Ordoñez, M.D.*, Joselito B. Díaz, M.D., MHA, FPNA

World Citi Medical Center, Philippines

A vascular catastrophe: A case of a 72-year-old Filipino male who developed ACS-STEMI after hemorrhagic conversion of an ischemic stroke

The patient is R.M., 72-year-old, Filipino male, who came in with a chief complaint of dizziness. Four hours prior to admission, he was walking home when he noted sudden onset non-rotatory dizziness with no associated nausea, vomiting, loss of consciousness, headaches or body weakness. Persistence of symptoms prompted consultation and admission.

He was received at the emergency room with symptoms of non-rotatory dizziness, but no other subjective complaints. His blood pressure was elevated at 160/90, with noted irregularly irregular cardiac rhythm on auscultation, as well as focal neurological deficits. Due to persistence of symptoms and for closer monitoring for deterioration of neurological status, patient was referred to Neurology service, admitted to the ICU, and started on Aspirin, Atorvastatin, Betahistine, Citicoline, Omeprazole, and Enoxaparin. Further workup was done including 2D Echo with Doppler Studies, Carotid Duplex Scans, and AV Duplex Scans of the bilateral lower extremities.

The next day, the patient underwent Cranial MRI with MRA under the advice of Neurology service due to persistence of dizziness and episodes of hypertension. The patient also waived ICU management due to financial constraints, hence he was transferred to the wards. Upon facilitation of cranial MRI, there was noted hemorrhagic conversion of initial infarctions, hence Aspirin and Enoxaparin were discontinued, and he was started on Mannitol and Telmisartan.

During the next hospital days, his neurological status remained stable with no new deficits, and he was referred to Rehabilitation service for physical therapy. Therapy initially began with bed repositioning and sitting, and progressed to bedside exercises and rising from bed.

On the patient's 12th hospital day, he was cleared for discharge from all services, however after less than 5 minutes of routine physical therapy session, he experienced sudden onset dyspnea with diaphoresis. The patient was hypotensive at 50/30, with other vital signs stable. He was placed on complete bed rest, hooked to oxygen support at 6 LPM nasal cannula, and repeat laboratories were requested. Due to no resolution of hypotension after fast drip of a total of 300cc pNSS, patient was started on both Dobutamine and Norepinephrine drips. Laboratories revealed a septal wall infarction on ECG and elevated Troponin I, hence the patient was treated as a case of ACS-STEMI. After conferring with Neurology service, he was started on Aspirin, Clopidogrel, Enoxaparin, and Trimetazidine, and transferred back to the ICU.

The patient was referred to Pulmonology service for co-management, and a diagnosis of pulmonary embolism was also considered, hence the patient was advised CT Pulmonary Angiography once more stable. At the ICU, the patient's inotropes were titrated accordingly and tapered as the patient's blood pressure stabilized. On his 15th hospital day, patient again waived ICU management and CT Pulmonary Angiography due to financial constraints, and transferred to the wards. Over the next days, there was no noted recurrence of dyspnea, hypotensive episodes, or new neurologic deficits. Medical management was continued, and on the patient's 20th hospital day, he was deemed fit for discharge from all services and sent home well.

Biography

Miguel Q. Ordoñez is currently a third year and chief resident in the internal medicine residency program in World Citi Medical Center, a private tertiary institution in the Philippines. Dedicated to patient care and learning, he has developed a strong interest in clinical medicine, particularly in managing complex cases in both the wards and intensive care unit. Beyond clinical work, he enjoys creating educational materials and contributing to academic activities within the department.

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Decoding cognitive decline: A multimodal AI approach to distinguishing Alzheimer's disease, dementia with Lewy bodies, and PSP using tau oligomers

Neurodegenerative diseases affect over 55 million people worldwide, yet accurately distinguishing Alzheimer's Disease (AD), Dementia with Lewy Bodies (DLB), and Progressive Supranuclear Palsy (PSP) remains difficult due to overlapping clinical symptoms and limited disease-specific biomarkers, with misdiagnosis rates approaching 30%. Recent biochemical evidence suggests that distinct tau protein polymorphs may help differentiate these disorders, though the complexity of tau experimental data limits interpretation using traditional analytical methods. In this study, quantitative structural and morphological tau features extracted from previously collected, de-identified datasets were analyzed using machine-learning models. To address limited sample size, the training dataset was augmented with 5,000 synthetic samples, while evaluation was restricted to replicate-aware held-out real data. Multiple models were tested alongside a custom ensemble framework, NeuroFoldNet, which achieved a mean classification accuracy of 79.01% under K-fold cross-validation. Feature-importance analysis revealed that a small subset of tau structural measurements consistently drove accurate disease classification, suggesting that specific morphological signatures of tau polymorphs may hold greater diagnostic relevance. These findings demonstrate the potential of machine learning to interpret complex tau datasets and support biomarker-driven differentiation of related neurodegenerative disorders.



Biography

Pavan Patel is a student at Stephen F. Austin High School, graduating in 2027 at the top of his class. He has co-presented a study on a novel multimodal AI approach to diagnose Alzheimer's disease (AD), Dementia with Lewy Bodies (DLB), and Progressive Supranuclear Palsy (PSP) at the 2026 Houston Global Health Collaborative Conference in Houston, Texas, alongside Shreyan Shah. He is particularly interested in research within neurology and cardiology, and has gained clinical experience at HCA Healthcare under Dr. Podet and at Texas Oncology under Dr. Chen.



Shreyan Shah is a student at William P. Clements High School, graduating in 2028 near the top of his class. His research interests focus on the intersection of artificial intelligence and healthcare, particularly the application of machine learning to improve diagnostic and analytical methods in biomedical research. His research has been published in IEEE proceedings and is indexed in IEEE Xplore. He has co-presented his novel multimodal AI approach for diagnosis of AD, DLB, and PSP at the Houston Global Health Collaborative Conference in Houston, Texas, with Pavan Patel.



R. Ya. Gurskaia
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Sequential clinical improvement after autologous mesenchymal stem cell therapy in concurrent Huntington's disease and ankylosing spondylitis: A case report

Background: Huntington's Disease (HD) is a fatal autosomal-dominant neurodegenerative disorder with no approved disease-modifying therapy. Ankylosing Spondylitis (AS) is a chronic systemic spondyloarthropathy causing progressive functional disability. Their co-occurrence in a single patient is exceptionally rare and therapeutically uncharted. Autologous Mesenchymal Stem Cells (MSCs) have been investigated separately in HD and AS models for their neuroprotective and immunomodulatory properties. To our knowledge, no indexed published case documents autologous MSC therapy in a patient carrying both diagnoses concurrently.

Case Presentation: A 35-year-old male (born 1982) presented with confirmed HD debut (family history: affected father) and concurrently diagnosed AS (confirmed jointly with a rheumatologist). Baseline assessment (October 2017): UHDRS motor score 19; BASFI 14; BASDAI 2.0. The patient received autologous bone marrow-derived (~90%) and adipose-derived (~10%) MSCs (SmartCell biotechnology), 50 million cells monthly for three consecutive months (total 150 million MSCs), administered as 50% intravenous plus 50% epidural, each dose preceded by plasmapheresis. Concomitant medication included neuroprotective support (lipidacrine, thiamine/ascorbic acid combination), a dopamine agonist (pramipexole, initiated at month 2 at low dose with gradual titration), and a short-course NSAID (etoricoxib 90 mg, 7 days). Vitamin supplementation (retinol/tocopherol) was maintained throughout. No immunosuppressive or disease-modifying antirheumatic drugs were administered during the observation period.

Results: UHDRS motor score and BASFI demonstrated progressive improvement across all three sequential treatment cycles. BASDAI, which was within a low range at baseline (2.0), showed minor fluctuation before reaching 1.0 at final assessment. Primary clinical gains were observed in motor and functional domains. Follow-up duration covered the active treatment period (October–December 2017); long-term follow-up data are not available for this report.

No serious adverse events occurred throughout the treatment course. The treating neurologist documented marked short-term clinical improvement in motor function and physical functioning within two months of initiating therapy.

Timepoint	UHDRS motor	BASFI	BASDAI
Baseline — Oct 2017	19	14.0	2.0
After 50M MSCs — Nov 2017	8	8.5	2.2
After 150M MSCs — Dec 2017	2	1.0	1.0
Net change	–89%	–93%	–50%

Conclusion: This case describes substantial short-term improvement in recorded neurological and functional rheumatological outcomes following three sequential administrations of autologous MSCs in a patient with concurrent Huntington's disease and ankylosing spondylitis, without serious adverse events. UHDRS motor score declined from 19 to 2 (–89%) and BASFI from 14 to 1 (–93%) over the treatment period. Given the single-patient design, causal inference is limited; the observed improvement may reflect a combination of MSC effects and concomitant pharmacological support, and spontaneous fluctuation cannot be excluded. Lack of long-term follow-up and absence of a control condition further limit generalizability. Nevertheless, the temporal association and multisystem clinical response across two pathophysiologically distinct conditions support further prospective investigation of autologous MSC therapy in complex neuro-immunological disease settings.

Keywords: Huntington's Disease, Ankylosing Spondylitis, UHDRS, BASFI, BASDAI, Autologous Stem Cells, Mesenchymal Stem Cells, Regenerative Neurology, Case Report.

Biography

R. Ya. Gurskaia (previously Omelchenko) is a practicing neurologist with a PhD (Candidate of Medical Sciences) in neurology. She has nearly two decades of clinical experience in the diagnosis and management of neurodegenerative and neurological disorders, including Huntington's disease, Parkinson's disease, Alzheimer's disease, and comorbid neurological conditions. Her clinical and research interests focus on regenerative approaches to neurological disease, including cell-based therapies. She practices at a private neurological clinic in Kyiv, Ukraine, and has presented clinical observations at international conferences on regenerative medicine. Disclosures: The presenting author is the treating physician in this case. No commercial funding was received. AI-assisted tools were used for language editing under full author oversight and responsibility.



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Benzodiazepine and SSRI co-prescription vs. monotherapy: A bibliometric review of effects on adherence and anxiety relief

Selective Serotonin Reuptake Inhibitors (SSRIs) are first-line treatments but have delayed onset and may initially worsen symptoms; benzodiazepines are sometimes co-prescribed to provide immediate relief. Despite this, research on co-prescription is fragmented across study types and outcomes, making it difficult to determine how the field is structured and what areas are emphasized or underexplored. This study analyzes trends in the literature on benzodiazepine-SSRI co-prescription, examining the distribution of study types and research emphasis.

A bibliometric review of the top 100 most cited studies was performed using the Web of Science Core Collection, with a PRISMA-guided selection process. Records published through 2026 were identified using keywords SSRI, benzodiazepine, and anxiety, and ranked by citation count. Metadata, including authorship, publication year, journals, and citation count, were extracted and curated in Excel. Studies were categorized by study type, outcome focus, and study design. Data were analyzed using RStudio, with relative frequencies and distributions visualized in bar charts.

The literature is predominantly composed of clinical studies (77%), with comparatively limited mechanistic research. Outcome focus is largely directed toward long-term treatment trajectories (58.8%), while adherence and discontinuation remain underrepresented (8.8%), despite being central to the clinical rationale for co-prescription. This imbalance suggests that although co-prescription is intended as a short-term bridge strategy, the literature more frequently emphasizes long-term outcomes, a mismatch between clinical rationale and research focus.

Additionally, predominance of observational study designs indicates a reliance on real-world data, which may limit causal interpretation of treatment effectiveness.

Overall, the literature on benzodiazepine-SSRI co-prescription is predominantly clinical and emphasizes long-term outcomes, while adherence remains underrepresented despite being a central rationale for co-prescription, a mismatch between the intended short-term use of co-prescription and the outcomes most frequently examined in the literature. These findings are limited by the use of a single database and variability in study designs and treatment protocols across studies. Future research should prioritize standardized longitudinal and mechanistic investigations to better align research focus with clinical practice.

Biography

Sammi Chou is a biochemistry undergraduate at Baylor University pursuing a physician-scientist career. Her work spans neuropharmacology, public health, and translational research, with experience in both laboratory and data-driven research environments. She has contributed to projects involving experimental models and large-scale literature analysis, while also leading research teams and coordinating collaborative workflows. Beyond research, she is engaged in clinical volunteering and student leadership, with a focus on connecting scientific inquiry to patient-centered outcomes.



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A rare case report of two siblings with unique presentations of adult-onset Niemann-Pick disease type C

Background: Niemann-Pick Disease Type C (NPC) is a genetic, lysosomal storage disorder characterized by an inability to transport cholesterol and lipids inside cells. This causes a patient-unique set of visceral and neurological symptoms that can include hepatosplenomegaly, dementia, cognitive decline, learning disabilities, dysphagia, or ataxia. Due to the nonspecific and masquerading nature of the symptoms, NPC tends to be misdiagnosed or undiagnosed in the majority of patients. Most patients develop NPC symptoms before or during adolescence, making the adult-onset form of NPC even more challenging to diagnose for many physicians.

Case Presentation: Two siblings were referred to the clinic in 2016 for assessment of neurological symptoms. There was no relevant family history. The then 33-year-old brother was previously diagnosed by a gastroenterologist with liver nodular regenerative hyperplasia after a history of portal hypertension and hyperbilirubinemia and presented to the clinic with progressive cognitive decline. Physical examination at the time revealed impaired tone, ataxia, normal DTR, and no cranial nerve deficits. Previous history was notable for elevated bilirubin levels since birth. In 2022, five years after his last evaluation, the patient presented with worsening cognition and behavior and a genetic panel that found that the patient was heterozygous for two NPC1 gene variants. Later that year, an EEG, previously normal in 2016, showed abnormal mild diffuse slowing consistent with a diffuse encephalopathy of metabolic, degenerative, or vascular origin.

His then 41-year-old sister also presented in 2016 with complaints of recurrent facial paralysis, lack of balance, coordination, chronic cough, and attention deficit. Physical examination revealed hyperreflexia, mild appendicular ataxia, facial asymmetry, and dysarthria. In contrast

to her brother, the patient has no relevant previous medical history. An EEG at the time was consistent with a diffuse encephalopathy of metabolic, degenerative, or vascular origin. After her brother was genetically diagnosed with NPC in 2022, a genetic panel was also ordered for her and the family which showed she was heterozygous for two NPC variants. In 2023, she started to progressively develop dysarthria and dysphagia, which may be related to progression of the disease. Now that both siblings have been correctly diagnosed, they are receiving treatment with Miglustat and Aqneursa.

Conclusions: This case report exhibits the complex presentation and genetics of NPC that provide a difficult path towards early diagnosis, especially in patients with juvenile or adult onset of the disease. Arriving at the diagnosis for these two siblings took six years due to delays in not only recognizing the rare disorder as a possibility, but also in the approval of genetic testing within the medical system. The eventual diagnosis was due to the ability of several physicians to recognize a pattern worth investigating further due to the progressive decline of the patients with no absolute clinical explanation. This shows how suspecting NPC as a potential diagnosis in patients with unexplained ataxia, cognitive decline, and liver disease is crucial to provide the patients with the proper work-up that can lead to early targeted care that can slow the progression of the disease.

Biography

Sara Forastieri Vicente is a second-year medical student at the University of Puerto Rico School of Medicine. She earned a Bachelor of Arts in both Chemistry and Creative Writing from the University of Pennsylvania in 2024. During her time there, she researched in Neuroscience, particularly sleep disorders. Now, Sara has developed an interest in studying rare diseases that affect people globally and the nuances of their diagnosis and treatment. She is also active in multiple student organizations where she helps coordinate community service activities.



Shrey Somani

Dougherty Valley High School, United States

Decoding human neural progenitor fate commitment with deep learning: A single-cell transcriptomic framework for predicting developmental cell-state transitions

Focal Cortical Dysplasia (FCD) causes epilepsy in 4 million newborns yearly. Treatments are surgical removal of malformed brain tissue, which fails for 40% of patients and causes permanent cognitive defects. FCD arises when Neural Progenitor Cells (NPCs) commit to regional brain identities at incorrect embryonic developmental time points. However, this commitment in humans has never been precisely identified, preventing implementation of regenerative therapies. This project aimed to identify the precise time point at which NPC fate commitment occurs to guide regenerative therapy for FCD. Using single-cell RNA sequencing from 52,183 human brain organoid cells across two independent datasets during organoid development, I calculated fate-bias scores (rostral gene markers - caudal gene markers) to track forebrain and hindbrain regions. Interquartile-range-based heterogeneity analysis showed a fall in transcriptional variability in correspondence with the commitment threshold. Commitment occurred 18 ± 2 days of brain development across all datasets, marking it the first measurement of this threshold in humans. Cells showed reduced plasticity and became irreversibly committed to their regional fate after this timepoint. A gradient boosting machine learning model achieved 93% accuracy in predicting if cells would commit at normal timing using Day 10-14 gene expression profiles. Defining this enabled ML prediction from Days 10-14, flagging abnormal commitment batches for early chemical intervention while cells were still plastic. This work identified the first quantitative time-point of neural commitment in human development, the intervention window before cells irreversibly lock into rostro-caudal fates. Enabling the manufacturing of regenerative cortical tissue that eliminates seizures while preserving cognitive function.

Biography

Shrey Somani is a high school researcher focused on neuroscience, biotechnology, and computational biology. His work includes single-cell transcriptomic analysis of human neural development at UCSF and research on neural progenitor fate commitment. He has presented his research at science fairs and academic conferences, earning recognition for his work in neurobiology and biomedical research. Beyond the lab, Shrey founded Vireka, a nonprofit research advocacy platform that helps students access research education, mentorship, and publication opportunities. He hopes to continue developing translational neuroscience projects that connect computational analysis with real-world biomedical impact.



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Pontocerebellar hypoplasia: A review from 1912 to 2022

Pontocerebellar hypoplasia is a rare neurodevelopmental disorder that results from differences in formation and function of the pons, cerebellum and cerebrum. It can be diagnosed prenatally or postnatally with a combination of clinical, neuroimaging and genetic data obtained over time. The diagnosis of pontocerebellar hypoplasia usually portends severe developmental delay, epilepsy and/or neurodegeneration in childhood. Here we perform a comprehensive review with the primary goal of evaluating published evidence addressing the clinical and genetic features of pontocerebellar hypoplasia by type and subtype. Secondly, we summarize neurodiagnostic patterns of pontocerebellar hypoplasia and demonstrate its spectrum. Finally, we provide recommendations in diagnosis, prognosis and management for the neurologist. To address these goals, we performed an extensive review of published literature from 1912 to 2022. We identified 191 publications by combining search results from PubMed, OMIM and cross-referenced bibliographies. Publications on developmental neuroanatomy, not

pertaining to pontocerebellar hypoplasia or published in a foreign language were excluded. We performed both qualitative (1912–1993) and quantitative (1993–2022) analyses to understand the current classification of this disease as it pertains to genetic and neurodiagnostic features of pontocerebellar hypoplasia by type and subtype. Our review shows that the most reported types of pontocerebellar hypoplasia are 1, 2 and 6; less frequently described are 3, 4 and 9. Very few cases are described for all other subsequent pontocerebellar hypoplasia types. Mutations in TSEN54, RARS2, EXOSC3 and AMPD2 (genes that regulate RNA processing and basic cellular metabolism) are the most frequently reported pathological mutations in pontocerebellar hypoplasia. The neuroradiographic features of pontocerebellar hypoplasia are complex and evolve over time, affecting the pons, cerebellum, vermis, cortex and cerebral white matter. In conclusion, pontocerebellar hypoplasia is a rare neurodevelopmental disorder, often the result of genetic dysfunction in basic neural metabolism. The diagnosis conveys significant implications for the affected individual and their families and requires a combination of clinical, neuroradiographic, and genetic testing to best inform type/subtype categorization of pontocerebellar hypoplasia.

Biography

Shriya Singh earned dual Bachelor of Science degrees from the University of Maryland in 2023 and is currently a third-year medical student at Georgetown University School of Medicine. Her research focuses on the genetic and molecular basis of neurological disease, including work in neurodevelopmental disorders and neuro-oncology. Shriya is also passionate about patient education, advocacy, and advancing women's health.



Vedanth Srivatsan

Acton Boxborough Regional High School, United States

Adaptive multimodal artificial intelligence for early Alzheimer's disease risk assessment and progression forecasting using plasma biomarkers, neuroimaging, and clinical data

Accurate risk stratification in Alzheimer's disease is complicated by a fundamental issue: the most informative biological signals span blood, brain imaging, and clinical history — data types that are rarely interpreted together in routine practice. I present an adaptive multimodal AI platform, developed as a computational framework for Alzheimer's disease risk modeling, that integrates plasma biomarkers including the amyloid-beta 42/40 ratio, phosphorylated tau-217, neurofilament light chain, GFAP, soluble TREM2, and YKL-40; structural MRI features such as hippocampal volume, cortical thickness, ventricular size, and regional patterns of atrophy; FDG-PET measures of cerebral glucose metabolism where available; and routine clinical variables including age, sex, and cognitive assessment scores into a unified framework for risk prediction, disease trajectory modeling, and clinical decision support. Predictions are generated using a confidence-weighted ensemble of logistic regression, random forests, gradient boosting, support vector machines, and neural networks, with weights adjusting dynamically by disease stage and robustness maintained across heterogeneous, longitudinal, and partially missing data — conditions that characterize real clinical cohorts. Each output includes a normalized 0–100 risk score with an associated confidence estimate, feature-level attribution identifying which biomarkers or imaging measures most influenced the prediction, longitudinal projections of evolving risk, and plain-language reports generated separately for clinicians and patients. Validation on publicly available longitudinal cohorts will assess predictive performance, calibration, temporal stability, and bias across diverse patient populations.

Biography

Vedanth Srivatsan is a freshman at Acton-Boxborough Regional High School. When not studying geometry or playing the violin, he can usually be found reading a book or playing outside. He has a deep interest in biology and World War 2 medical history and is currently preparing to join his school's lacrosse team this spring. Vedanth's goals for his freshman year include maintaining a high standing in his school classes, as well as conducting extra research outside of school.

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