magnus Group

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

16-17
June, 2023

Rome
Italy

Venue:

Mercure Roma West, Viale Eroi di Cefalonia, 301,
00128 Roma RM, Italy

BOOK OF ABSTRACTS

magnus Group

16-17 JUNE

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ABSTRACTS

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

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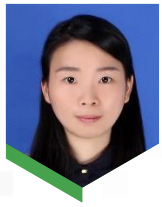

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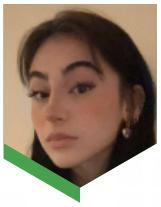

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Wei Song
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Xiaoyu Chen
Shanghai University of Medicine And Health Sciences, China

Yanjun Chen
University of Wisconsin Madison, United States

Zahra Khan
University of Toronto, Canada

Zeynep Elif Olmez
Hisar School, Turkey

Thank You All...

Welcome Message

On behalf of the organizing committee and all members of the honorable Scientific Committee, I welcome all attendees to the 7th edition of International Conference on Neurology and Neurological Disorders.

This highly prestigious conference contains no less than 31 Scientific Sessions, covering the broadest possible areas of interest and research into neurology and neurological disorders. The vast range of presenters and Keynote speakers ensures that all dedications to the greater understanding of neurology and neurological disorders, along with greatly refined treatments, better protocols, and most importantly a higher degree of accuracy in diagnosis will be of great benefit to present and future patients who have neurological disorders and lesions. This conference will assist to enhance the global collective wisdom pertaining to neurology and neurological disorders. I wish you all the very best with your presentations and thank you sincerely for your participation.

Best regards

Ken Ware

NeuroPhysics Therapy Institute, Australia

Welcome Message

Dear colleagues:

Welcome to the 7th Edition of the International Conference on Neurology and Neurological Disorders. Since the beginning of the world, humankind has looked for answers to transform the brain, the mind and the soul. It's understandable that a better brain can mean a better mind and vice versa; and that better Brains & Minds can mean a better world. It's for neurologists and neuroscientists to offer a better future to the world by creating interventions to develop better brains, minds and address neurological disorders in our society.

During the following days, we're going to hear lectures on the brain and it's neurological disorders. Human brain at its best operating on a human brain at its worst, as neurosurgeon Dr. Gazi Yasarsil once stated.

The conference will cover from induced neuroplasticity by exercise, bionutrients, or biotechnology, to the neuroplasticity created by neuromodulation with Transcranial Magnetic Stimulation or Laser Photobiomodulation. It's the congress's goal to offer a wide variety of education on pioneering research in different neurological conditions such as Alzheimer, Stroke, TBI, Pain, Addiction, and Alcoholism. Finally, wisdom on the neuroscience of consciousness and art as a modality of self-care among neurologists and caregivers will be covered to refresh our souls.

I can enthusiastically say that the future of the world depends on the best brains (you) taking care of the brain's world.

So, hope you enjoy!

Juan Moreira

CNC / Gnosis Neurointegrative Center, United States

Welcome Message

Dear congress visitors, it is an honor and pleasure to welcome you to Dementia 2023. Dementia is a devastating neurodegenerative condition. Though medications can alleviate some symptoms of dementia, the lack of robust biomarkers to facilitate early diagnosis means that current interventions are typically given too late to modify disease progression. Thus, accurate and timely diagnosis is key for optimal intervention. Dementia related changes likely occur decades before symptoms are observed underscoring the need for novel tools that can aid in the early identification. A better understanding of the early changes in the brain in Dementia and their relation to cognitive function using advanced imaging methods will ultimately facilitate the development of measures for early detection and progression of dementia.

Jun Hua

Johns Hopkins University School of Medicine, United States

Welcome Message

Dear congress visitors,

The brain has often been called the final frontier in medical research. From the intricacies of neural networks to the blood-brain barrier, it still contains so many unknowns. Neurological disorders affect the body's autonomic, peripheral, and central nervous systems and include diseases such as multiple sclerosis, Alzheimer's disease and other dementias, Parkinson's disease, epilepsy, migraines, and so many others. Especially concerning are the number of new and prolonged psychological disorders that have emerged during COVID-19 as a result of COVID-19 isolation, lost diagnoses, missing treatment, and unfortunately lost research time. It is time to make up for this lost time! Our patients need and deserve it!

As we continue to emerge from COVID-19, we need to take advantage of every conference possible to push our field ahead. While COVID-19 kept us from interacting with one another to develop solutions in neurology, that time has stopped and we need to double down and make up for lost time. Even though we could not meet over the past several years, healthcare problems in neurology continued and even grew, and the statistics are as alarming as ever. For example, in 2019, it was estimated that neurological disorders accounted for a total of 7.5 million years lost due to premature death and a total of 8.2 million years that people have lived with neurological disabilities (Burden of Neurological Conditions, www.PAHO.org, accessed Sept. 22, 2022). And the statistics are getting even worse due to lost diagnostic, treatment, and research time that occurred during COVID-19.

It is an honor and pleasure to welcome you to the 7th edition of the International Conference on Neurology and Neurological Disorders – Neurology 2023 June 16-17, 2023 where we can begin to make up for the time we lost. The conference promises to be two days of enriching conversation on what we know and don't know about neurology, and how we can move forward.

I hope to see you all in Rome where we will discuss and develop solutions to our most pressing neurological problems!

Benvenuto a Roma!

Thomas J Webster
Interstellar Therapeutics, United States

Welcome Message

As the Chair, I am very honorable and pleasurable to write these welcome notes.

Currently, more than 55 million people have dementia worldwide with that number to triple by 2050. The cost of dementia is $1.3 trillion globally and is projected to increase to $2.8 trillion by 2030. There is no cure for this disease, and the current treatments are neither specific nor always effective. Moreover, the molecular mechanisms underlying dementia are not fully understood.

Without a doubt, the conference, Dementia 2023, will provide a unique and timely fantastic opportunity for delegates, researchers, scientists, physicians, caregivers, and professionals? from all corners of the globe to meet and explore the most recent, energizing, and innovative developments as well as discoveries in every facet of this disease. With the growing prevalence and mortality of dementia, there is an increasing urgent need to develop novel and effective methods for earlier detection of the disease, elucidate the molecular pathogenesis for the initiation and progression of dementia, and generate specific and efficacious drugs and biologics for better treatment of the disease. One can expect that the developments and discoveries presented in this conference will significantly help to solve our aforementioned current urgent challenges.

Yong Xiao Wang

Albany Medical College, United States

Welcome Message

Dear congress visitors, it is an honor and pleasure to invite you to participate to the 2nd International Alzheimer's Disease & Dementia Conference (Dementia 2023) that will take place on the 16-17 June 2023, in Hybrid Format in Rome, Italy. During the conference we will explore the latest developments in basic, translational and clinical research related to Alzheimer's Disease & Dementias. One of the crucial aspects of this congress is the high-level of interdisciplinary exchange between clinicians and basic researchers with the aim to improve the clinical guidelines for diagnosis and treatment of Alzheimer's Disease & Dementias. We are convinced that to present and discuss each own work with other colleagues, sparking and sharing ideas between scientists, is the key to progress in science. We do encourage you to join us to ask questions and share knowledge during all scientific sessions.

Mario Allegra
University of Palermo, Italy

Welcome Message

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

I look forward to welcoming you to 7th Edition of International Conference on Neurology and Neurological Disorders, to be held in one of the ancient and historic, well-known, prestigious and attractive City of Rome.

The Conference will provide the ideal forum to stimulate ideas and establish collaborations as well as to initiate intense discussions. Extended networking opportunities will foster communications between delegates.

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 is thus one of the most significant global challenges of our time, with a lot of questions remaining.

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

The core of the Event is its Scientific and Teaching Program. For people entering such a prolific environment, acquiring an initial understanding of these diseases becomes more difficult each year. Graduate students, postdoctoral fellows, or other early-stage scientists and senior staff focusing on multiple sclerosis, Alzheimer's disease research, can take advantage of this opportunity to accelerate their knowledge immersion towards becoming an expert in this exciting field and discuss some of the latest trends in these research fields through close interaction with established leaders in the field. Workshop sessions are emphasized to open discussion between participants and lecturers, and immediate application of new knowledge. Students will participate in faculty-led exercises such as debates.

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

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

Sergey Suchkov

Moscow State University of Food Industry (MGUPP), Moscow, Russia

Welcome Message

Dear congress visitors, I am honored to write a few words of welcome. Artificial intelligence is currently being applied to various fields such as autonomous driving, face recognition, and voice recognition systems. It is also widely applied in the medical field, such as support for image diagnosis, and estimation of health conditions based on wearable device information. In the future, along with the development of medical data platforms, the application of AI to the dementia field is expected to progress. In this presentation, as an example of the latest clinical application of Al to the field of dementia, I will introduce a new method for estimating cognitive function and brain atrophy from health checkup data. We hope that this lecture will advance the application of Al to the field of dementia.

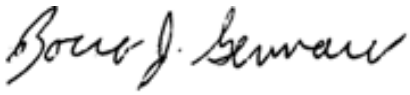
Kaoru Sakatani

The University of Tokyo, Japan

Welcome Message

Dear Attendees, Presenters, Organizing Committee and Distinguished Guests,

The invitation to write a welcome message and to present at this conference is an honor. I am very grateful to the Organizing Committee for emphasizing the important research into neurology and neurological disorders. The conference will enhance the global collective knowledge pertaining to neurological disorders and psychopathologies. The presentations and discussion will also provide crucially important hypotheses and suggestions for new research directions. My own special interest is in how specific changes and/or damage to specific neural regions can have a major impact on one's conscious mental states. Further, it is interesting to think about various philosophical implications of this line of research in terms of the so-called traditional mind-body problem. I wish you all the very best with your presentations. I only wish I could be there in person especially given its wonderful location!

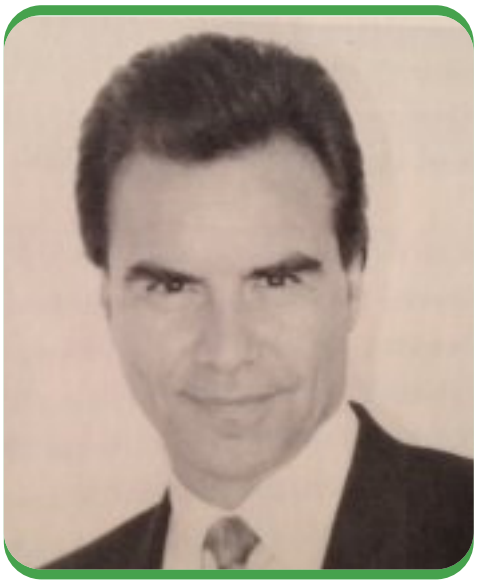


Rocco J Gennaro

University of Southern Indiana, USA

Welcome Message

Dear congress visitors, It is exciting to participate in this important conference. The significance and potential of biotechnology is clearly manifest by the incredible speed and progress that was made with the RNA Vaccines for the COVID-Pandemic which saved millions of lives. One of the lessons is how basic and clinical development can be accelerated resulting in viable life, saving products. This is a New Age for Biotechnology not only because of the new technologies being developed but also because of the progressive, new drug development processes which we now know are possible. I look forward to sharing our work on a DNA Therapeutic Vaccine for prevention and treatment of Alzheimer's Disease.



Arthur P Bollon

Vitruvian Biomedical, Inc., Dallas, Texas

Welcome Message

We greatly anticipate your presence at this conference. As a practicing neurosurgeon scientist, we are delighted to share our important discoveries that will have meaningful impact for patients. The conference is set to be packed with high quality data and engaging conversations. It will be the catalyst to push discovery forward. We look forward to your participation.

Brandon Luke Wold

University of Florida, United States

Keynote Speakers

Ken Ware
NeuroPhysics Therapy Institute, Australia

Frank Owen Bastian
Bastian Enterprises, United States

Juan Moreira
CNC / Gnosis Neurointegrative Center, United States

Juliana Fort
Louisiana State University Health Shreveport, United States

Jun Hua
Johns Hopkins University School of Medicine, United States

Roy F Baumeister
University of Queensland, United States

Thomas J Webster
Interstellar Therapeutics, United States

Yong Xiao Wang
Albany Medical College, United States

Jacqueline Tuppen
Cogs Club, United Kingdom

Mario Allegra
University of Palermo, Italy

Sergey Suchkov
Moscow State University of Food Industry (MGUPP), Russia

Kaoru Sakatani
The University of Tokyo, Japan

Lukui Chen
Southern Medical University, China

Zhenhuan Liu
Guangzhou University of Chinese Medicine, China

Thank You All...

ABOUT
MAGNUS GROUP

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

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

magnus Group

16-17 JUNE

DAY 01

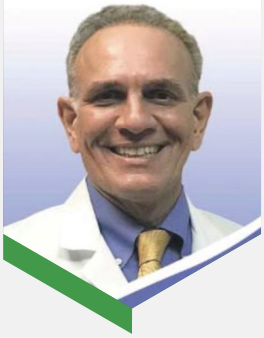
KEYNOTE FORUM

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

Transcranial laser photobiomodulation on posterior reversible encephalopathy syndrome

Posterior Reversible Encephalopathy Syndrome (PRES) have been described as a clinicoradiological syndrome characterized by acute ruptured of the Blood Brain Barrier and development of vasogenic edema secondary to acute endothelial injury. MRI shows predominantly reversible parieto-occipital T2/FLAIR hyperintense lesions, but not necessarily restricted to those brain areas. Persistent neurological sequelae are at least reported in 10-20% of the patients and can be as high as 44%. Mortality has been observed up to 19.1% of patients, where 5.7% were directly attributed to the cerebral findings causing neurological deficit. Transcranial Laser Photobiomodulation is a novel treatment which can be offered safely to a PRES's patients due to lack of adverse effects. Here, we present a small series of PRES patients who received photobiomodulation treatment during the first 72 hours followed by frequent consecutive treatment. Clinical improvement on the clinical neurological deficit and brain lesion resolution on MRI was observed. Transcranial PBM can be a novel treatment to improve neurological sequela on PRES patients.



Juan Moreira

CNC / Gnosis Neurointegrative Center, United States

Biography

Board-certified neurologist with two fellowships; vascular neurology and neuroimaging. Exceptional background with over 25 years of experience devoted to providing top quality healthcare at the hospital, ER and ambulatory setting. I have successfully run a private practice from 1995-to 2022. In addition, with experience on Botox, Transcranial Magnetic Stimulation and Laser Photobiomodulation. Interested on clinical research. Looking to work on an institution dedicated to excellence. Board-certified neurologist with two fellowships; vascular neurology and neuroimaging. Exceptional background with over 25 years of experience devoted to providing top quality healthcare at the hospital, ER and ambulatory setting. I have successfully run a private practice from 1995-to 2022. In addition, with experience on Botox, Transcranial Magnetic Stimulation and Laser Photobiomodulation. Interested on clinical research. Looking to work on an institution dedicated to excellence.

Neuro nanomedicine: Penetrating the blood brain barrier, delivering stem cells, treating stroke and meningitis, and so much more

Nanotechnology has revolutionized numerous industries, particularly medicine. Nanoparticles, nanotubes, drug delivery nanoparticles, and nanotextured neural probes are just several of many examples where nanomedicine is positively impacting neuroscience. This presentation will summarize some of the more prominent studies where nanomedicine has been used to treat various neurological disorders. Specifically, it will cover in vivo studies which have demonstrated a faster and greater return of motor function to stroke induced rats when implanted with stem cells and carbon nanotubes. Further, it will cover the design of nanoparticles that can penetrate the blood brain barrier to more effectively deliver drugs to treat Parkinson's disease. New self-assembled nanomaterials will also be covered which can kill bacteria in the brain. This presentation will also present what studies are needed for the field of nanomedicine to continue to positively impact neurological diseases.

Audience Take Away Notes

- How nanomedicine is being used to diagnose and treat neurological diseases
- The future of using nanomedicine to deliver stem cells to the brain
- How to design nanoparticles to penetrate the blood brain barrier
- How to use nanomaterials to treat stroke and meningitis as well as other neurological diseases

Thomas J. Webster

Interstellar Therapeutics,
United States

Biography

Thomas J. Webster's (H index: 111; Google Scholar) degrees are in chemical engineering from the University of Pittsburgh (B.S., 1995; USA) and in biomedical engineering from RPI (Ph.D., 2000; USA). He has served as a professor at Purdue (2000-2005), Brown (2005-2012), and Northeastern (2012-2021; serving as Chemical Engineering Department Chair from 2012 - 2019) Universities and has formed over a dozen companies who have numerous FDA approved medical products currently improving human health. Dr. Webster has numerous awards including: 2020, World Top 2% Scientist by Citations (PLOS); 2020, SCOPUS Highly Cited Research (Top 1% Materials Science and Mixed Fields); 2021, Clarivate Top 0.1% Most Influential Researchers (Pharmacology and Toxicology); and is a fellow of over 8 societies.

Cerebral vascular calcium signaling in diabetic Alzheimer's disease-related dementias

Alzheimer's disease (AD) and AD-related dementias (ADRD) are the common incurable neurodegenerative conditions characterized by progressive cognitive deterioration, memory decline and even death. The major forms of ADRD include vascular contributions to cognitive impairment and dementia (VCID). In agreement, vascular dementia is the second most common form of dementia and the most frequent comorbidity with AD. Diabetes is a leading factor in the development of VCID. The molecular mechanisms that link diabetes to the development of VCID remain to be elucidated, and the current treatments for these diseases are neither always effective nor specific. We and other investigators have found that the severity of diabetes is highly associated with the cognitive and memory impairments. The role of diabetes may primarily occur due to the abnormal glucose metabolism and increased reactive oxygen species (ROS) production in cerebral vascular smooth muscle cells (CASMCs). In support, antioxidant therapies have shown promising results in protecting against diabetes-induced VCID and other dementias. Diabetes increases mitochondrial ROS. The increased mitochondrial ROS could dissociate FK506 binding protein 12.6 (FKBP12.6) from type-2 ryanodine receptor (RyR2) to remove its inhibitory effect on RyR2 channel and induce Ca^{2+} release, which causes cerebral vasoconstriction and cerebral blood flow reduction, thereby leading to progressive memory loss, cognitive decline and VCID. These novel findings may not only enhance our understanding of the molecular mechanisms for VCID, ADRD and AD, but also help to identify novel therapeutic targets for these common devastating diseases.

Audience Take Away Notes

- Our current presentation will greatly help the audience to create their future research directions
- The finding presented may significantly assist the audience to develop new drugs, establish novel preventive and therapeutic strategies for VCID and other dementias
- Our research could also be used by other faculty to expand their research or teaching

Yong-Xiao Wang

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

Biography

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

Cogs Club: Making a difference

The NICE guidelines in November 2006 stated:

People with mild/moderate dementia of all types should be given the opportunity to participate in a structured group cognitive stimulation programme. This should be commissioned and provided by a range of health and social care workers with training and supervision. This should be delivered irrespective of any anti-dementia drug received by the person with dementia.

Cognitive Stimulation Therapy (CST) is an evidence-based group programme of activity and stimulation grounded in person-centred care which is normally run as a two-hour session, twice a week, over seven weeks and can be run by professional staff or volunteers.

In Kent, its importance is recognised and it is offered routinely by the local mental health teams. However, after the sessions have finished the participants have nothing as stimulating to continue with.

COGS Clubs aim to fill this gap. It uses a variety of approaches and is consistent with *Everybody's Business* (2005) which is committed to improving people's quality of life, meeting complex needs in a co-ordinated way, providing a person-centred approach and promoting age equality

The activities are based on and run in the same structured way as a Cognitive Stimulation Therapy session but instead of two hours a COGS Club offers a day of activity, stimulation, music and fun for a person with mild dementia.

It provides the opportunity for them to recall and/or develop new skills and facilitate the transfer of these skills to activities at home. It values and respects the unique qualities of each individual, whilst giving a sense of connectedness. It also provides the family/carer/significant other with a day's respite.

Fun is a key component to the day. Laughter dissolves distressing emotions

Jacqueline Tuppen

Cogs Club, United Kingdom

Biography

Mrs Jaqueline Tuppen graduated in 1997 with a BSc Hons and Specialist Practitioner from the University of Greenwich, London. She worked with the local Community Mental Health team, ending as their Acting Community Services Manager. She became an Admiral Nurse in 2008, and retired from the NHS in 2011 when as an independent specialist nurse practitioner she started - COGS Club for people in the early stages of Dementia. She continues to work for Dementia UK as a sessional Admiral Nurse on their Dementia Helpline. She has published articles, and presented at a variety of events in the UK, Eire and Italy.

Audience Take Away Notes

- To raise awareness of the innovative use of Cognitive Stimulation and other approaches for people living with mild to moderate dementia in the community consistent with *Everybody's Business* (2005) which is committed to valuing and respecting the unique qualities of each individual, improving people's quality of life, meeting complex needs in a co-ordinated way, providing a person-centred approach and promoting age equality
- Attendees of the conference would gain awareness of the COGS Club initiative
- Attendees would have a greater understanding of the value and clinical worth of Cognitive Stimulation Therapy
- Attendees would gain greater appreciation of the clinical worth of a variety of interventions for people with mild to moderate dementia

BOOK OF ABSTRACTS

magnus Group

16-17 JUNE

DAY 01

SPEAKERS

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

Dr. Denise DuPree*, DOA, AP, Robert Knable MS
Homewatch Caregivers of St Pete Beach, St Petersburg, Florida, USA

Aging in place with dementia: Strategies for successfully aging in place

Aging in place is the term used to describe seniors who are able to live independently in their own homes for as long as possible. The reasons for aging in place vary from individual to individual. Some seniors want to remain active and independent in their own home for as long as possible. Others may want to stay close to their family and friends or are worried about losing their identity if they move into a retirement community. The objective of this presentation is to help family members and caregivers who support older adults understand the complexities of dementia and aging in place. It will provide an overview of the unique challenges of dementia, as well as helpful suggestions for working with people who have dementia in their homes. It will share practical strategies for making an aging in place plan for older adults with memory loss. What you should expect as dementia progresses in terms of memory, emotion, and behaviour along with suggestions for interacting with older adults residing with dementia in their homes will also be shared. Dr. DuPree will share strategies for successful aging in place and practical tips to help older adults with memory loss live independently. She will introduce the complexities of dementia and aging in place, providing an overview of the challenges that come with each stage of dementia and outlining solutions to address them. These solutions will cover a range of essential topics, such as safety concerns, home modifications, environment management, communication issues, behavioral episodes, and tools for maintaining independence.

Audience Take Away Notes

- Presentation will help family members and caregivers who support older adults understand the complexities of dementia and aging in place
- Will share practical strategies for making an aging in place plan for older adults with memory loss
- Strategies for successful aging in place and practical tips to help older adults with memory loss live independently
- Introduce the complexities of dementia and aging in place, providing an overview of the challenges that come with each stage of dementia and outlining solutions to address them L

Biography

Dr. Denise DuPree, DOA, AP, is the co-owner and Administrator of Homewatch CareGivers of St Pete Beach. She has been in the health field as an Acupuncture physician for over 21 years. Denise is a natural caretaker and loves helping clients and families age in place and navigates the health journey they may be on. She has extensive experience in elder care, oncology, women's health, and postpartum care. Dr. DuPree received he undergraduate Degree from California State Polytechnic University, Humboldt (Cal Poly Humboldt), her masters from Northwest Institute of Acupuncture and Oriental Medicine (NIAOM) NIAOM and received her Doctorate of Acupuncture from Pacific College of Health and Science. She has worked in hospitals pre and post-surgery and attended labor and delivery using Acupuncture for pain management. Lived in Siem Reap Cambodia volunteering at the maternity and children's hospital and educated many women about their health and their children's health. She also lived in Tianjin China were she volunteered at the Heping YiYuan Hospital and the Tianjin TCM hospital in the brain clinic.

Constantina Mizis

President and CEO, The Latino Alzheimer's, and Memory Disorders Alliance, Chicago, Illinois USA

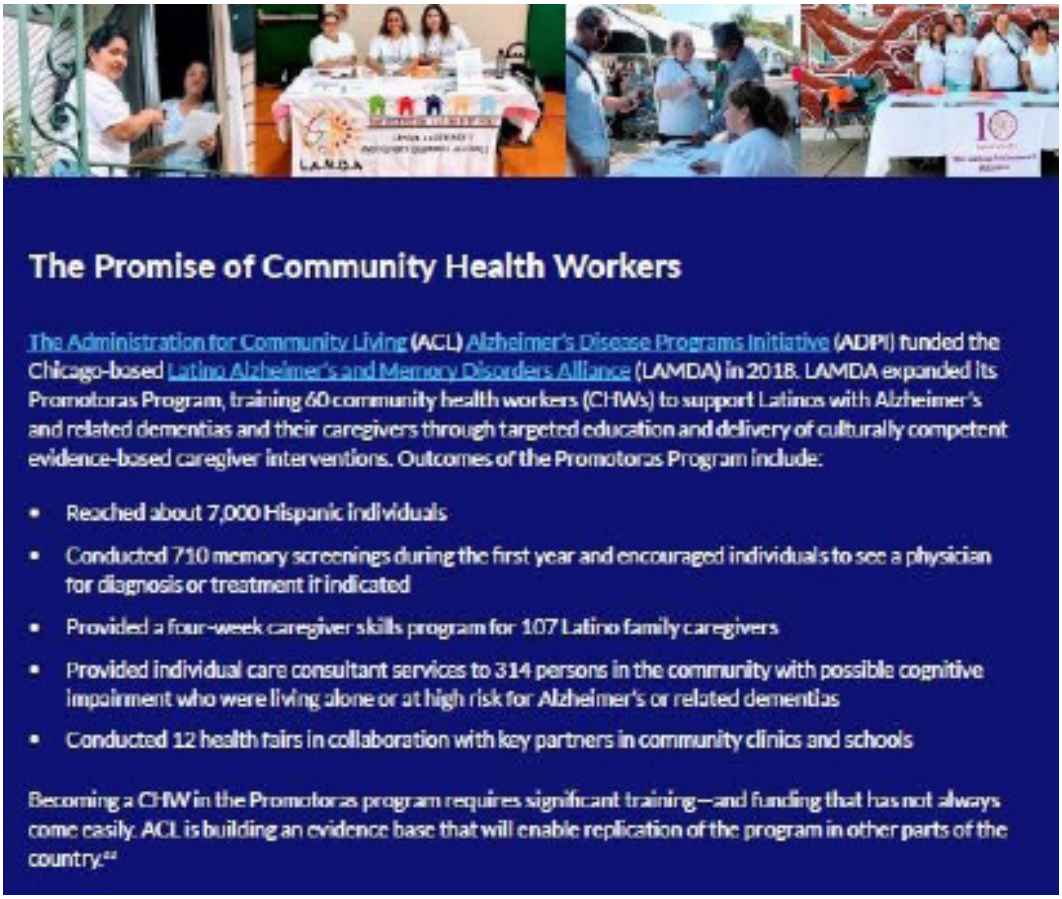
Closing gaps by addressing racial & ethnic disparities in Alzheimer's disease and reducing the cost and risk of dementia in diverse communities

Statement of the Problem: More than 5 million of Americans are living with Alzheimer's, and that number is expected to increase to 14 million in 2050. According to UsAgainstAlz, Alzheimer's will cost Latino families a cumulative $2.3 trillion by 2060 and the number of Latinos with AD is expected to increase by 832% (379,000 to 1.1 million) from 2012 to 2060 (USC).

Addressing disparities: In 2018 the US Administration for Community Living funded LAMDAs Community Health Workers model. By 2021, this program reached about 7,000 individuals in Chicago, conducted 710 MS, and encouraged individuals to see a physician for diagnosis. In October 2020, the Milken Institute's report Better Brain Health through Equity highlighted the CHW Model to address their recommendations:

1) Build a dementia-capable and culturally competent workforce through recruitment, retention, and increased interdisciplinary training. 2) Increase community outreach and engagement to improve brain health awareness in racially and ethnically diverse communities. In November 2020, the US-National Alzheimer's Project Act (NAPA) Advisory Council on Alzheimer's Research also recommended the CHW Model to be replicated in other States of the USA.

About CHWs Model: This is an evidence base program that proven mechanisms for trusted community members to deliver culturally competent education and services into diverse communities. CHWs are community leaders and have previous training on known AD risk factors to 1) conduct outreach and community education activities to identify families needing services; 2) assess, educate, support, and connect families to community services in a culturally appropriate way; and 3) facilitate caregiver trainings and assist families to better understand available service options.



Milken Institute's report Better Brain Health through Equity

Audience Take Away Notes

- Audience will learn how the Community Health Worker Model closes the gaps of services for people suffering from Alzheimer's disease and their Caregivers

Service Gap:

1. Provision of effective supportive services to persons living alone with ADRD in the community
2. Improvement of the quality and effectiveness of programs and services dedicated to individuals aging with intellectual and developmental disabilities with ADRD or those at high risk of developing ADRD
3. Delivery of behavioral symptom management training and expert consultation for family caregivers

- After this presentation, audience will learn how to use the Cultural Component to addresses Racial & Ethnic Disparities in Alzheimer's disease and Reducing the Cost and Risk of Dementia in Diverse Communities
- The Community Health Workers Curriculum can be replicate and use after 35 hours of training from The CHWs program increases community outreach and engagement to improve brain health awareness in racially and ethnically diverse and other underserved communities. The Administration of Community Living of the USA recommend the use of Evaluation Tools like: Dementia Quality of Life (DEMQOL), Dementia Capability Assessment Tool, and CHW Training Act Alzheimer's Collaborative Training tool
- Community Health Workers model provides evidence base methods of intervention that builds a dementia-capable and culturally competent workforce through recruitment, retention, and increased interdisciplinary training

Biography

Mrs. Mizis is the founding executive director of The Latino Alzheimer's and Memory Disorders Alliance (LAMDA) based in Chicago, IL. LAMDA is on the frontline of addressing Alzheimer's growing impact on the Latino community and provides direct services to thousands of Latinos struggling with Alzheimer's annually. Constantina Mizis has dedicated over 25 years of service to Latino older adults and their family caregivers. She has provided consulting and training services throughout the United States and has worked with national organizations to develop programs for Latino older adults. Have been successful in creating a partnership network for LAMDA with 76 Latino organizations in the Nation. CNN, Univision, Chicago Tribune, Los Angeles Times, ABC, Comcast, and the Democrat and Chronicle New York Newspaper are some of the media that highlight the efforts of Constantina Mizis.

Paul Y. Song[1*], Lucia Hui[1], Sean Hong[1], Yoonmi Kang[1], Yongman Kim[2]

[1]NKGen Biotherapeutics, Santa Ana, CA
[2]NKMax, Seoul, South Korea

Use of highly enhanced autologus natural killer cells for advanced Alzheimer's disease

Background: Alzheimer's Disease (AD) remains a neurodegenerative diseases with little to no disease modifying treatments. As more evidence emerges on how misfolded amyloid deposits elicit a cascade of autoreactive neuroinflammation and damage, it is clear that removing these proteins by themselves will not fully address the complex process. Natural Killer (NK) cells are an essential part of the innate immune system that have been found to slow progression of amyloid protein deposition. NK cells have also been reported to identify and eliminate autoreactive T-cell generated neuroinflammation and damaged neurons via DNAM-1 and NKG2D. But immunosenescent NK cells have been reported in many patients with neurodegenerative diseases. While NK cells have always been challenging to grow and enhance ex vivo especially when derived from older or heavily pretreated donors, SNK01 is a first-in- kind, autologous non-genetically modified NK cell product with significant increased cytotoxicity and over 90% activating receptor expression that can be consistently produced from any donor.

Methods: In preparation for a full Phase I dose escalation study, SNK01 was administered intravenously (IV) to three patients under compassionate use approval with confirmed advanced AD. NK cells were successfully activated and expanded from each patient. Average cytotoxicity was increased over 400% and average activating receptor expression was greater than 90%. There were no adverse events reported in the over 20 total doses given. Each patient had noticeable improvement from their pre- treatment baseline evaluation. In subsequent analysis of serial CSF samples obtained from additional patients, SNK01 was found to cross the blood brain barrier to reduce tau, p-tau protein levels as well as neuroinflammatory markers GFAP, NF-L, YKL-40.

Conclusion: SNK01 with high cytotoxicity and activating receptor expression can be consistently produced from patients with advanced AD. Preliminary data suggests that SNK01 is very safe and may reduce protein levels and neuroinflammation. This will be expanded into a full Phase I/II trial with a more prolonged dosing schedule.

Audience Take Away Notes

- How amyloid/tau protein deposition elicits an autoimmune cascade which causes neuroinflammation and damage
- How the innate and adaptive immune systems contribute to the overall disease process
- Understand how Natural Killer Cells may play a role in reducing protein deposition, neuroinflammation, and clearing damaged neurons and cellular debris
- Review the preliminary clinical experience of patients with advanced Alzheimer's disease treated with autologous enhanced NK cells

Biography

Paul Y. Song, MD is a board-certified radiation oncologist and the CEO of NKGen Biotech. Dr. Song graduated with honors from the University of Chicago and received his M.D. degree from George Washington University. He completed his residency in radiation oncology at the University of Chicago where he served as Chief Resident and did a brachytherapy fellowship at the Institute Gustave Roussy in Villejuif, France. He was also awarded an ASTRO research fellowship in 1995 for his research in radiation inducible gene therapy.

Tommy Kwan Hin Fong[1], Teris Cheung[2], Joanna Ngan Sze Ting[1], Vivian Lui Wai Yan[1], Wai Chi Chan[1], Corine Wong Sau Man[3], Calvin Cheng Pak Wing[*1]

1. Department of Psychiatry, The University of Hong Kong, Hong Kong, China
2. School of Nursing, The Hong Kong Polytechnic University, Hong Kong, China
3. Division of Community Medicine and Public Health Practice, The University of Hong Kong, Hong Kong, China

Efficacy and safety of Transcranial Pulse Stimulation (TPS) in older adults with mild neurocognitive disorder – An open-label trial

There are limited effectiveness and potential alarming side effects of pharmacological approach for the Neurocognitive disorder (NCD). Transcranial pulse stimulation (TPS) has been shown to be a potential tool to bring the benefit. We have conducted an open-label study. Older adults with the diagnosis of mild NCD received 2-week 6 sessions neuro-navigated TPS interventional. 19 eligible subjects (with 12 females and 7 males) were recruited in this study and completed the whole TPS interventions. Repeated measures ANOVA showed statistically significant effects of time on HK-MoCA ($F(3,54) = 4.99$, $p = 0.004$), 30-second interval of Verbal Fluency Test ($F(3,54) = 2.94$, $p = 0.041$), Stroop interference ($F(3,54) = 3.46$, $p = 0.023$), and Chinese IADL ($F(3,54) = 2.78$, $p = 0.050$) after received the intervention. Bonferroni post-hoc comparisons on HK-MoCA showed that both scores from immediate post (Mean = 21.16, SD = 3.98) and 12-week follow-up (Mean = 20.58, SD = 4.29) were significantly higher that of 12-week TAU (Mean = 18.74, SD = 3.87) ($p < 0.05$). There were no serious adverse effects reported. TPS has brought significant improvement in cognition of elderly with mild NCD. It has a great potential to delay the deterioration of cognition in older adults. The effectiveness and the long-term effect of TPS in cognition still need to have further large scale randomized controlled trial to support.

Audience Take Away Notes

- The first TPS study targeting older adults with mild NCD
- TPS has brought significant improvement in cognition of elderly with mild NCD
- TPS has significantly improved the global cognition and the effect maintained for at least 3 months after the intervention

Biography

Dr. Calvin Cheng joined the department in November 2017 as a clinical assistant professor. He is also the honorary associate consultant of psychogeriatric services in the Hong Kong West Cluster. Before joining the department, he obtained Fellowship of the Hong Kong College of Psychiatrists in 2014. His current research interests reside in pharmacological and non-pharmacological interventions for geriatric depression, structural and functional brain mapping, non-invasive transcranial brain stimulation for mood disorder and cognitive impairment in elderly. He obtained Distinguished Young Fellow Award given by the Hong Kong Academy of Medicine in 2018.

Yanjun (Judy) Chen[1*], MD/PhD, Laura M. Hancock[2], PhD, Adam J. Paulsen[3], MS, Alex A. Pinto[1], MS, Carla R. Schubert[1], MS, Natascha Merten[4], PhD

[1]Department of Ophthalmology and Visual Sciences, University of Wisconsin Madison, Wisconsin, United States of America

[2]Department of Neurology, University of Wisconsin Madison, Wisconsin, United States of America

[3]Survey of the Health of Wisconsin, University of Wisconsin Madison, Wisconsin, United States of America

[4]Department of Population Health Sciences, University of Wisconsin Madison, Wisconsin, United States of America

The association between pupil reactivity and cognitive measures among community-dwelling middle-aged and older adults

Aims: The pupillary light reflex (PLR) has been widely used to reflect visual function and brain activities, making it a potential biomarker for central nervous system (CNS) neurodegeneration. In this cross-sectional study, we evaluated the association between the PLRs and cognition in community-dwelling middle-aged and older individuals.

Methods: Pupil reactivity was recorded using a binocular infrared pupillometer (Neur-Optics, Inc., Irvine, CA) in 403 participants (mean age 60.7 years, 57.3% females) from the Beaver Dam Offspring Study, an epidemiologic cohort study of aging. Thirteen pupil parameters were calculated to describe the PLRs. Cognitive testing consisted of Trail Making Test A and B, Rey Auditory Verbal Learning Test, Digit Symbol Substitution Test, and Verbal Fluency Test. Principal Component analysis (PCA) was used to calculate an overall cognitive function score. Linear regression was used to assess the association between pupil parameters and PCA scores, adjusting for age, sex, education, medications, health-related quality of life, and systemic and ocular comorbidities.

Results: Cognitive scores decreased by 0.039 (95% CI [-0.050, -0.028]) per year increase in age and were lower in males than females by 0.76 (95% CI [-0.96, -0.55]) ($p < 0.001$). Pupil constriction amplitude in millimeters, the duration from stimulus onset to maximal constriction velocity, and the post-illumination pupil response were significantly associated with cognition after adjusting for (1) age and sex and (2) age, sex, and multiple covariates ($p < 0.05$).

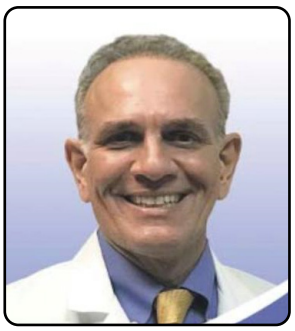
Conclusions: In this study, we found an association between multiple PLR measures and cognitive function in middle-aged and older adults. The findings suggest the potential of pupil reactivity to serve as a biomarker of brain aging. Future longitudinal research needs to assess if changes in the PLR can predict cognitive decline over time.

Audience Take Away Notes

- The anatomy and physiology of the PLRs pathway
- How brain aging may affect the neuronal substrates of the PLRs pathway
- The PLRs are associated with neuropsychological cognitive measures
- The potential of the PLRs to serve as a biomarker of brain aging and neurodegeneration

Biography

Dr. Chen received her MD at Beijing Medical University, China, in 1994 and a PhD at the State University of New York, U.S.A., in 2004. She completed a neurology residency at Saint Louis University and a neuro-ophthalmology fellowship at the University of Iowa before joining the faculty in the Department of Ophthalmology and Visual Sciences at the University of Wisconsin Madison in 2011. As a Neuro-ophthalmology, Dr. Chen's research focuses on using the pupil as a biomarker for brain aging and neurodegeneration. She has published about 30 research articles in SCI (E) journals.



Juan Moreira
CNC / Gnosis Neurointegrative Center, United States

Transcranial photobiomodulation on acute & subacute stroke patients

The limited time for a therapeutic window of intravenous thrombolysis have been a problem since its approval twenty eight years ago. The availability of a stroke team for thrombectomy or intravenous thrombolysis, and the complexity of the eligibility criteria may be factors that can limit its use. Transcranial Laser Photobiomodulation is a novel treatment which can be offered safely to an acute and subacute stroke patient due to lack of adverse effects. Here, we present a small series of acute and subacute stroke patients who received photobiomodulation treatment during the first 72 hours followed by frequent consecutive treatment. Clinical improvement on the clinical neurological deficit and partial brain lesion resolution on MRI was observed. Acute and subacute stroke patients may benefit of transcranial photobiomodulation therapy with laser as observed clinically and on neuroimaging.

Biography

Board-certified neurologist with two fellowships; vascular neurology and neuroimaging. Exceptional background with over 25 years of experience devoted to providing top quality healthcare at the hospital, ER and ambulatory setting. I have successfully run a private practice from 1995-to 2022. In addition, with experience on Botox, Transcranial Magnetic Stimulation and Laser Photobiomodulation. Interested on clinical research. Looking to work on an institution dedicated to excellence. Board-certified neurologist with two fellowships; vascular neurology and neuroimaging. Exceptional background with over 25 years of experience devoted to providing top quality healthcare at the hospital, ER and ambulatory setting. I have successfully run a private practice from 1995-to 2022. In addition, with experience on Botox, Transcranial Magnetic Stimulation and Laser Photobiomodulation. Interested on clinical research. Looking to work on an institution dedicated to excellence.

Leger C[1*], Quirie A[1], Lemaire S[1], Meloux A[1], Garnier P[1,2], Prigent-Tessier A[1]

[1]Inserm umr1093-CAPS, Universite Bourgogne Franche-Comte, UFR Sciences de sante, F-21000 Dijon, France

[2]Departement Genie Biologique, IUT, 21000 Dijon, France

Impact of exercise intensity on cerebral BDNF levels: Role of FNDC5/ Irisin

Physical exercise (EX) is an essential approach to improve physical and brain health. It is well known that positive effects of EX on brain involve the upregulation of cerebral BDNF (brain-derived neurotrophic factor), which promotes neuronal plasticity and cognitive functions. However, the underlying molecular mechanisms leading to an elevation of this neurotrophin remain not well known. One of these mechanisms is associated with myokine production induced by skeletal muscle contraction during EX. Recently, it has been reported that irisin, a peptide secreted into the blood and derived from the enzymatic cleavage of FNDC5 (Fibronectin type III domain-containing protein 5) was identified as a positive modulator of cerebral BDNF production. While the influence of EX intensity and modality on cerebral BDNF levels was characterized, the impact on muscular FNDC5/Irisin expression and circulating irisin level remains to be explored. The experiments were conducted on adult male Wistar rats sedentary (SED, n= 6) or subjected to a horizontal treadmill exercise (EX, n= 18) protocol, 30 min/day for 7 consecutive days. The EX intensity was modulated by the speed of the treadmill set at 12 m/min for the EX12 (40% of maximal aerobic speed, MAS), 14 m/min for the EX14 (50% of MAS), and 18m/min for the EX18 (70% of MAS) groups. Tissues were harvested 24 hours after the last EX session whereas blood was collected immediately. Expression of FNDC5/Irisin in oxidative muscle soleus (SOL) versus glycolytic muscle gastrocnemius (GAS) as well as hippocampal BDNF level were studied by both Western blotting and immunofluorescence methods. Serum irisin level was determined by the ELISA test. Our data showed that 1) the increase in FNDC5/ Irisin protein level was observed only in GAS and from a corresponding threshold of 50% of MAS in EX compared to SED rats; 2) FNDC5/Irisin immunostaining was localized only in fast-type fibers which are predominant in GAS 3) serum irisin level increased from EX at 50% of MAS and, 4) a positive correlation was obtained between serum irisin and hippocampal BDNF levels when SED and EX rats were studied simultaneously. In conclusion, our study reveals that an intensity-dependent increase in hippocampal BDNF level is dependent on circulating irisin level and that the upregulation of FNDC5/Irisin expression is observed only in glycolytic muscle.

Audience Take Away Notes

- The purpose of this study is to promote circulating irisin as a peripheral marker of cognitive abilities
- List all other benefits
- We believe that there will be a positive association between irisin blood levels and cognitive abilities in healthy subjects or patients with cognitive impairment. At the societal level, our findings will provide a strong case to promote brain health through physical exercise. At the scientific level, our results will allow to design exercise protocols able to improve cognition with a simple blood assay

Ali Riza Günes[1*], N. Eminaga[1], M. Beglau[1], M. Köhne[1], U. Sprick[1]
[1]Department of Brain Stimulation, Alexius/ Josef Hospital, Neuss, Germany

Case series on brain stimulation using TPS: Sustained therapeutic effects even in early Alzheimer's disease

Background: Alzheimer's dementia (AD) is the most common form of dementia worldwide, affecting 1.8 million people in Germany alone. Various forms of therapy are available, yet AD cannot be stopped, even with innovative approaches such as monoclonal antibodies. The therapeutic effects of deep brain stimulation are still under investigation

Target: Initial studies of noninvasive brain stimulation using transcranial, pulse stimulation (TPS) has been able to objectify improvements in cognition by neuropsychological testing in AD patients. The aim of our observational study was to investigate the effects of TPS over a longer period of time and in younger patients with AD.

Research question: Are sustained positive effects of TPS also achievable in patients with early onset AD (EOAD), who often have a more rapid disease progression?

Methodology: In this case series, we report on three patients with EOAD who were treated by TPS over a 12-month period at ages 41, 53, and 53 years, respectively. Each patient received 6 therapy sessions within 2 weeks with an energy level of TPS 0.2 $mJ/mm^{2=}$ per single pulse at a total pulse count of 6000 per session and a frequency of 4 Hz. Thanks to 3D navigation based on MRI images of the patients, pulses were applied individually with the Neurolith device from Storz Medical. Pulses were applied bilaterally to the frontal, parietal and temporal cortex. Neuropsychologically, the test battery CERAD and for close examination of executive function, the color-word interference test (Stroop test), were applied with the following pre/ post design, Stroop test: t0 pre-stimulation: t1 after 6 treatments, t2 after 6 Weeks later, and t3 after an interval of 12 months. CERAD test: t0 pre-stimulation: t1 after l2 months.

Results: All patients showed no serious deterioration in the Stroop test even after 12 months of TPS treatment, and one patient even improved. Also, in the MMST as a subcategory of the CERAD test battery, 2/3 patients managed to achieve a higher total score. In some other subcategories of the CERAD test battery, such as verbal production ability, executive functions, and cognitive flexibility, 2/3 patients were also able to maintain the level of their baseline scores. In contrast, all patients showed deterioration in the visuo-constructive abilities subcategory.

Discussion: The results of this case series show that, contrary to previous doctrine, stabilization of certain cognitive abilities sustained over 12 months is possible even in EOAD using regular TPS treatment. Especially in the area of general cognitive function and executive function in particular, sustained improvements are even possible. However, a progression of deficits, especially in the area of visuoconstructive abilities, which are affected early in AD, could not be prevented. The conclusion of the present study is limited due to the small number of cases, so that further studies with larger samples are necessary.

Audience Take Away Notes

- Presenting innovative non-invasive brain-stimulation procedures: showing alternative/additional stimulation methods to TMS
- To show various treatment options for TPS: expanding the treatment spectrum thanks to specific stimulation targets
- Identifying treatment alternatives for treatment-resistant depression: supplementing the range of treatment options by TPS

Biography

Dr. Günes studied Medicine at the University of Dusseldorf and Cologne, Germany and graduated as MD in 2012. He then joined the research group of Prof. Peter Berlit (General Secretary of the German Society of Neurology/DGN) at Alfried Krupp Hospital, Essen, Germany. He received his PhD degree in 2018 at the same institution researching on vasculary diseases, especially Moyamoya-Disease. After four years of neurological fellowship supervised by Prof. Berlit, he received his board certification for Neurology in 2019. From 2018 until 2022 he completed his psychiatric fellowship at University Hospital Dusseldorf, Germany and received his Board certifivation for Psychiatry and Psychotherapy in 2022. Besides of publishing on research articles and writing chapters for neurological textbooks (SOPs in Neurology), he presented posters and held oral speeches during national and international (EAN) neurological Congresses.

Anastasia Demina[1, 2*], Agnes Soudry-Faure[3], Adrien Guilloteau[3], Vincent Meille[1], Benjamin Petit[1], Benoit Trojak[1, 2]

[1]Addiction medicine, Dijon Bourgogne University Hospital, Dijon, France

[2]INSERM U1093 Cognition, Action ET Plasticite Sensorimotrice, University of Burgundy, Dijon, France

[3]DRCI, Dijon Bourgogne University Hospital

A randomized double-blind clinical trial on the efficacy of transcranial direct current stimulation in reducing alcohol consumption in non-abstinent patients with alcohol use disorder

Background: Recent studies suggest that transcranial direct current stimulation (tDCS) targeting the dorsolateral prefrontal cortex (DLPFC) results in significant alcohol craving reduction. To study the impact of tDCS on alcohol use outcomes, we designed a randomized double-blind trial of active tDCS targeting DLPFC versus sham tDCS in alcohol use disorder (AUD).

Methods: Individuals with current AUD willing to reduce their consumption were randomized to receive active or sham anodal tDCS of the right DLPFC. As recommended, we chose as primary outcomes the total alcohol consumption (TAC) and the number of heavy drinking days (HDD) changes at 6-months follow-up compared to the baseline. Participants received the assigned intervention (active or sham tDCS) during five consecutive days with two stimulation sessions per day. The group effects were estimated using four mixed models for each primary outcome, with an alpha risk at 1.25% (unilateral).

Results: Of 338 randomized participants (39% female), 253 participants provided at least partial data on daily self-report alcohol consumption and 127 provided complete data. All models showed improved outcomes favouring active tDCS group either on TAC or HDD, but only two models reached statistical significance (only on HDD). In active tDCS group there were an estimated 5.2 to 6.8 g TAC reduction and 0.8 to 2.2 less HDD.

Conclusions: Our findings support the promising potential of tDCS for AUD. Although high rates of attrition precluded the confirmatory nature of our trial, imputation models showed a limited effect of only 5 days of active tDCS on clinical AUD outcomes.

Audience Take Away Notes

- This research will help the audience to better understand the theoretical framework for non-invasive brain stimulation use in addiction
- We will discuss the results of our large randomized controlled trial and the effect size obtained in the context of other therapeutical options for AUD. This can be useful for the strategic planning of the new research, with a considerable utility of medico-economical evaluation of different therapeutical options
- We will also discuss possible developments in everyday clinical practice in light of the up-to-date evidence for brain stimulation efficacy in AUD

Biography

Dr. DEMINA studied Medicine at the University of Montpellier, France and at the University of Burgundy, France. She graduated as M.D. and completed her master's degree in Ethics and Bioethics research in 2022. In November 2022 she obtained a position of a Head of clinic in Dijon Bourgogne University Hospital in Dijon, France. This position enables Dr. DEMINA to practice addiction medicine part time and pursue her academic career through teaching addiction medicine at the University of Burgundy and developing her research. She is currently pursuing her Ph.D. in neuroscience at INSERM U1093 Cognition, Action et Plasticite Sensorimotrice at the University of Burgundy.

Emna Bouallegue[1], Kais Bouallegue[2*]

[1] Department of Biological Engineering and Chemistry, National Institute of Applied Science and Technology. Tunisia

[2] Department of Electronic, Higher Institute of Applied Science and Technology, University of Sousse, Tunisia

DNA processes in fractal cell

Computational biology faces the challenge of modeling the complex dynamical processes that underlie cellular functions, which evolve and change depending on internal functional requirements and external environmental factors. To address this challenge, researchers have turned to theories of fractals and chaos, which have long been intertwined with dynamical systems. In this research paper, we propose a novel model that combines a fractal Julia process with numerical dynamical systems to solve topological problems in DNA processes.

The model includes a representation of chromosomes with and without link points, as well as various DNA processes, such as mitosis, meiosis, duplication, separation, division, and crossing over. The study identifies the position of the centromere and its impact on the form, behavior, and scale of the fractal cell. Additionally, this paper presents two examples of cell divisions with eight nuclei, illustrating both internal and external division. The research has significant implications for the understanding and treatment of genetic diseases, as well as potential applications in various fields of biology and medicine.

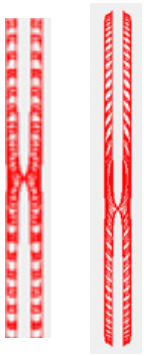


Figure 1: Model of chromosome with different position of centromeres

Figure 2: Chromosome with link points and Chromosome without centromere

The centromere, universally known as the primary constriction,is essential for chromosomal attachment to the spindle and for proper segregation. It serves as a link between sister chromatids until their dissociation at anaphase after which each chromatid converges towards a steady state. At present, however, no previous analysis was done to investigate the numerical position of centromere and to identify its position.

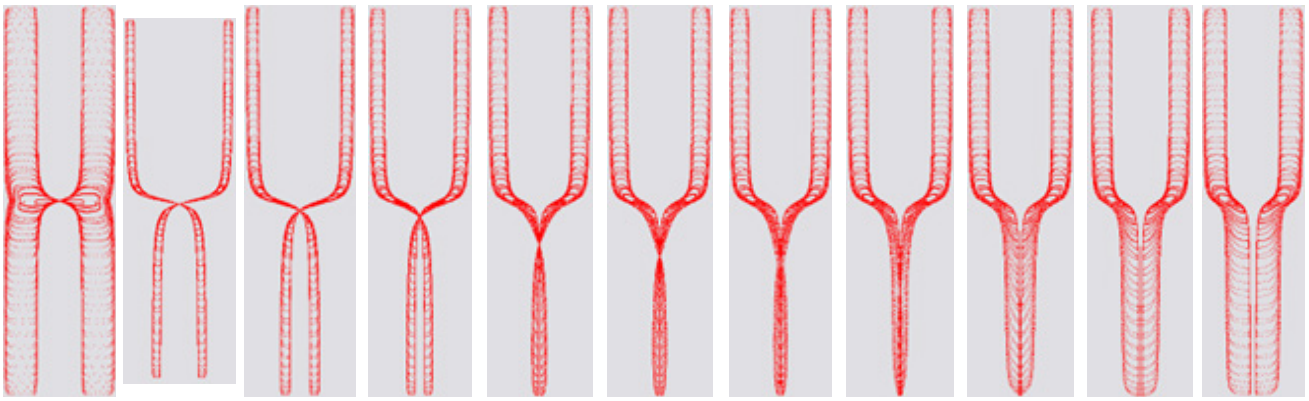


Figure 3 shows the crossing over process (mitosis process)

Figure 4 contains two behaviors of chromosomes; each chromosome has its position of centromere and without link points. We combining between them fractal algorithm, we obtain fractal flower contains eight leafs.

Figure 5 sows result of implemantion using chromosomes with link points.

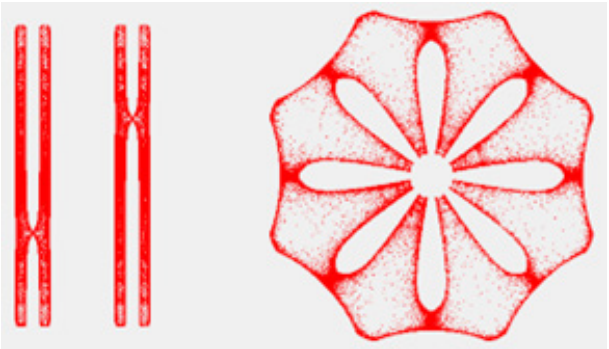


Figure 4: combining of two chromosomes without link points

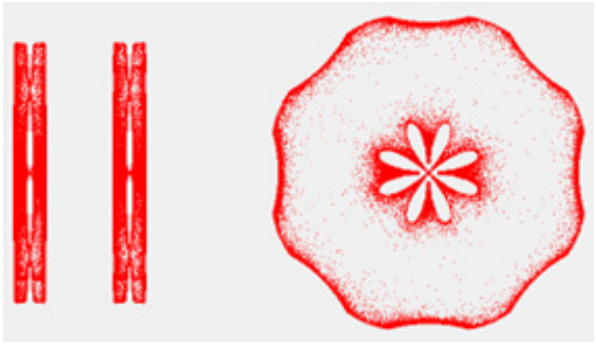


Figure 5: combining of two chromosomes with link points

In next subsection, we present only results of implementation of fractal cell contains eight nucleus and its division in two cells

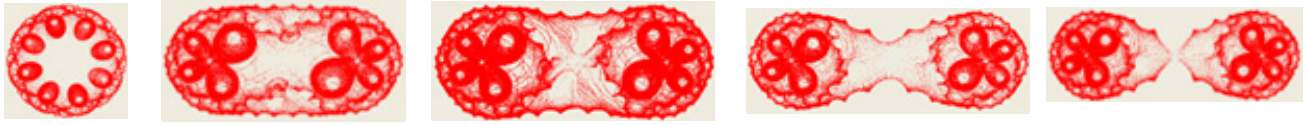
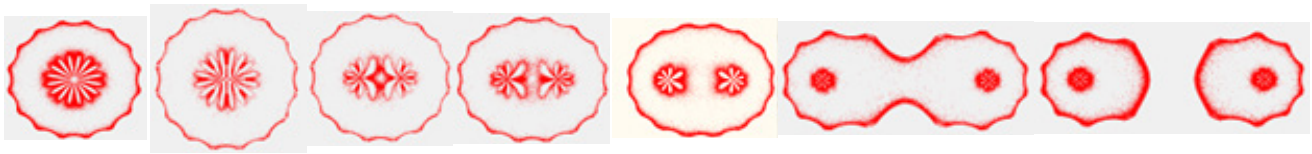


Figure 6: Cell division in fractal cell

We give an other results contains to meiosis process and cell division,in this result, we show the efficiency of our approach.



Audience Take Away Notes

- This paper has discovered the processes of the cell divisions of both mitosis and meiosis. All in all, the results show that this work could have a strong impact on the welfare of humanity and can be leveraged for the cure of genetic diseases

Biography

Kais Bouallegue studied electrical engineering at National Engineering School of Sfax, Tunisia. He received his PhD Thesis that he has defended on 2010 His thesis entitled Modeling and Chaotic control of nonlinear dynamic processes. deals with modeling of nonlinear systems using fractal processes and chaotic systems. Next, on 20l7 he has obtained the Accreditation to Supervise Research in Electrical Engineering and then he has been promoted to an associate professor at the University of Sousse. In his work, Dr. Kais Bouallegue proposed the novel fractal artificial neural cell to build new neural networks that can model complex systems and dynamics such as the meiosis in biological systems. Dr. Bouallègue is the (co)author of more than twenty publications in several international journals and conferences. He has also actively participated in the organization of several international conferences and events. He has served a reviewer for technical papers. Also, he has mentored researchers at undergraduate and graduate levels. Moreover, He has been collaborating with many different international universities. His currently research interests include fractal, chaos, and neural networks in complex systems.

Mokhtar Arazpour

Associate Professor of Orthosis and Prosthetics, Orthotics and Prosthetics Department, University of Social Welfare and Rehabilitation Sciences, Tehran, Iran

Comparison of the effects of sliding mechanism and reciprocating hinge-type medial linkage of knee-ankle-foot orthoses on walking ability in subjects with spinal cord injury

Objectives: This study evaluated the efficacy of two different medial linkage mechanisms (sliding mechanism (SM) and medial linkage mechanism associated with reciprocating gait motion (MLRGM)) used on patients with spinal cord injury (SCI).

Methods: Eight volunteer SCI subjects were fitted with a knee-ankle- foot orthoses (KAFO) equipped with 1) a sliding mechanism (SM) and 2) a medial linkage mechanism (MLRGM). Subjects walked at their self-selected speed along a flat walkway to enable a comparison of walking speed, endurance, and the resulting physiological cost index (PCI) to be performed.

Results: The use of a KAFO fitted with the MLRGM resulted in improvements to walking speed, distance walked, and energy costs when compared with the more standard SM. However, the time required to don and doff the KAFO increased with use of the MLRGM, but this difference is not statistically significant.

Conclusions: This study demonstrated that the use of a KAFO with a MLRGM could provide significant benefits for patients with a SCI when compared to a KAFO with a standard sliding mechanism.

Keywords: Spinal cord injury, Walking ability, Sliding mechanism, Medial linkage mechanism associated with reciprocating gait motion.

Biography

Dr. Mokhtar Arazpour is an associate professor in the Department of Orthotics and Prosthetics, University of Social Welfare and Rehabilitation Sciences (USWR), Tehran, Iran, where he also obtained his BS, MSc, and Ph.D. in Orthotics and Prosthetics. The title of his Ph.D. thesis is \Design, Construction, and Evaluation of the New Powered Gait Orthosis for Walking in Spinal Cord Injury Patients.\ Dr. Arazpour's research interests include lower-limb orthotics, osteoarthritis of knee and hand joints, design and construction of new lower limb orthosis, and walking analysis.

Naser Gilani[1,2*], Ehsan Razmara[3], Mehmet Ozaslan[1], Ihsan Kareem Abdulzahra[4], Saeid Arzhang[5], Ali Reza Tavasoli[6], Masoud Garshasbi[3]

[1]Department of Biology, Gaziantep University, Gaziantep, Turkey

[2]Farabi Molecular Laboratory, Irbil, Iraq

[3]Department of Medical Genetics, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran

[4]Kurdistan College of Medicine, Irbil, Iraq

[5]School of Medicine, Shahrekord University of Medical Science, Shahrekord, Iran

[6]Myelin Disorders Clinic, Pediatric Neurology Division, Children's Medical Center, Pediatrics Center of Excellence, Tehran University of Medical Sciences, Tehran, Iran

A novel deletion variant in CLN3 with highly variable expressivity is responsible for juvenile neuronal ceroid lipofuscinoses

With more than 7,000 identified rare diseases and approximately 80% being linked to genetic causes, diagnosing rare disease patients can often be difficult – resulting in lengthy, expensive, and emotional diagnostic odysseys, but with revolutionary advances in Molecular Diagnostic Techniques you have the genetic testing tool in hand to diagnose your patients in less time with high levels of certainty, with affordable cost. One of these techniques is Next Generation Sequencing that provides the possibility of doing monogenic, multigenic, whole exome, and whole genome sequencing just through days. In Neurology field with huge number of hereditary disease or disease with genetic based NGS is one of the important players that could be used to detect an array of neurological disorders from neonatal ICU cases to dementia or movement disorders in adults. In our article also thanks for advanced techniques like as NGS and other ones we found and report a novel mutation in CLN3 gene that is responsible for juvenile neuronal ceroid lipofuscinoses.

Audience Take Away Notes

- Get more information with details about some advanced techniques in molecular genetic field
- Have an idea how they can get help from these facilities for do diagnosis of some complex genetic disease
- Be informed about the usage of these procedures for providing a scientific paper in their institute or even clinic
- Learn about the limitations beside the advantages of these available investigations

Biography

Naser Gilani, Genetic Counsellor from 2013. MD, PhD in Molecular Biology and Genetics, Medical Doctor, graduated from Tehran University of Medical Science in 2003. PhD in Molecular Biology and Genetics, Turkey. Establish Farabi Medical Laboratory as the first Laboratory in North of Iraq working professionally in Medical Genetics field. Providing a wide range of Diagnostic Genetic tests. Establishing first genetic counselling clinic in area. Participation and presenting 17 lectures in Medical Genetics field, in International Congresses recent ones: Seven publications published, last one as A disorder clinically resembling cystic fibrosis caused by biallelic variants in the AGR2 gene that is published in Journal of Medical Genetics, leading human genetics journal with Impact Factor 6.318; Member of the Molecular Biology Society of Japan; Member of the editorial board of Neurology and Neuroscience Journal.

Akankunda Veronicah Karuhanga
Founder and Executive Director Golden Age Elderly Homes, Kampala, Uganda

The relevance of family involvement in the journey of dementia patients

Introduction: Dementia is an age mental disorder that makes victims lose normal functionality that need delicate attention. It has been technically defined as a clinical syndrome characterised by a cluster of symptoms manifested by difficulties in memory, disturbances in speech and and cognitive functions, changes in behaviours, and impairments in activities of daily living, and includes a range of neurological disorders characterised by memory loss and cognitive impairment. (Livingston et al,1996)

Most family members do not know that Dementia is an age-related disorder or illness and lack the requisite skills to handle the situation. The misconception about many old age-related issues like dementia is the root cause of a systematic social failure to create a conducive rehabilitation environment.

In the African context which is sometimes superstitious, in some scenarios, this condition is interpreted as a curse and hence treated with black magic which is against proved scientific solutions to the problem. This scientific ignorance concerning the problem leads to a Try and err treatment methodology and in fact, many of the patients die because of wrong prescriptions especially traditional concoctions.

Given such a misconceived approach suggesting that dementia patient is a curse, they are consequently discriminated against. They are also considered useless, labelled mad people because of the uncoordinated stories and hence their health needs are not prioritised as the case with normal people.

Why is it important for the family to play an exceptional role?

Family members are the primary health care givers and therefore the way how they handle the situation in its early stages determines future deterioration syndromes like total memory loss. Unfortunately, most family members are ignorant about this condition and in most cases the patients are brought to our facilities when their condition was already mismanaged by family members and we thus cannot do much. For example, incontinence can be managed at early stages through potty training or toilet scheduling before resorting to 24/7 diapers which are also not good.

Family is the only solace(consolation) amidst socially constructed stigmatisation practices. The moment their own family disowns them or fail to understand them, many dementia patients break down because of the sense of uselessness. Illness is the most critical moment in which family can prove a closer bond than ever (Giltin et al 2008) It is the best insurance policy that anyone can ever get. The act of sending dementia patients to elderly care facilities like us in Kampala presents a social paradox. For while on one hand the family members think that they are paying for quality care and rehabilitation of their abnormal aged relative, the dementia patients on the other hand feel socially disowned and being thrown away to third party caregivers. This therefore means that the best person for the job is actually the immediate family.

What can be done to fix the problem?

Professional Elderly care should be understood and practiced as an extension of homes not a dumping place for people considered as abnormal on account of ignorance. Immediate relatives should therefore be sensitised concerning the normalcy of dementia in the context of old age so that can be understanding and supportive to dementia patients rather than discriminating them as them present day lepers.

There is a need to skill home based care givers on how to handle dementia in its early stages. Unless this is done, many of our elderly home shall be filled with patients who should have been treated and supported from their homes. This skilling of home-based care givers is a vital intervention because until elderly care is appreciated as a human moral obligation, many transactional rehabilitation centres will crop up and this shall be one of the worst moral decadences of our times.

The task of caregiving is complex and can lead to physical, mental and financial stress for caregivers. Emotions such as guilt, resentment, sadness and the effort expended, as well as the anticipated loss of the relative, emerge (Brodaty H, Donkin M,2009). This therefore supports the need to skill home care givers.

Conclusion: Aging is a natural and inevitable dimension of human life and therefore its associated effects like dementia among others should be conceived and treated as normal. We members of society actually turn abnormal if we consider dementia an abnormal condition. The last people to be abnormal should be the immediate family members because it is old that gives forth to the new.

Biography

Veronicah Akankunda is a Ugandan Gerontologist, Neuro Researcher, social entrepreneur and advocate for elderly care. She is the Founder and CEO of Golden Age Elderly Homes (GAEH), a pioneering organization providing holistic care to seniors in Uganda.

Veronicah is a passionate geriatric care specialist, visionary leader with expertise in Gerontology, healthcare management, and social work for over 10 years. Her dedication to elderly care is inspiring. The engaging presentations, public lectures and compassionate care to seniors inspire audiences to action.

Apart from Gerontology consultancy she has innovated age-friendly living spaces. Golden Age Elderly Homes being the first care home in the country is a beacon of hope for Uganda's seniors offering a comprehensive range of services designed to cater to the diverse needs of the elderly population. From the Geriatric Training Academy that equips students with Nursing skills in Elderly care, to Mobility Aides, Personal Care, Elderly Nutrition, and Rehabilitation, the organization stands as a one-stop destination for elderly care support. The unique blend of home services, including Physiotherapy and Massage, Adult day care centre sets Golden Age Elderly Homes apart, providing a holistic approach to caregiving as seniors age gracefully with Dignity in the comfort of their homes. Veronicah's work focuses on addressing the Psychosocial, emotional, and healthcare needs of the elderly, promoting dignity, and challenging age-related discrimination. She has gained recognition for her efforts to improve elderly care in Uganda and Africa.

Her dedication to enhancing the lives of older adults has earned her respect and admiration internationally. Her work continues to inspire positive change and promote a culture of care and inclusivity for all ages. Her projects have Improved lives of countless elderly individuals and their families, Raised awareness about Geriatric care and age-related issues. Inspired a new generation of social entrepreneurs and caregivers, Contributed to policy changes and advocacy for elderly rights in Uganda.

Her selflessness, compassion, and innovative spirit makes Veronicah a true champion for the elderly and a role model for social entrepreneurship. She has won numerous Awards for Excellency in Palliative care ,Health entrepreneur , Innovation and Entrepreneurship, her Research in Geriatric Care , Neurology and Neurological disorders has been internationally published and continues to impact society.

Golden Age Elderly Homes has left an indelible mark on Ugandan communities. The organization has provided geriatric care to over 1962 elderly individuals, conducted more than 134 community health camps, and trained over 350 home care-based carers. The impact extends beyond physical care, touching on community health and general well-being.

BOOK OF ABSTRACTS

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NEUROLOGY AND DEMENTIA

Selin Edil*, Ozlem Bozkurt
Hisar School, Istanbul, Turkey

The neurological basis of narcolepsy and how it affects daily life

Extreme daytime drowsiness, cataplexy, and an early beginning of rapid eye movement sleep are all symptoms of the chronic neurologic illness narcolepsy. It can be distinguished from other conditions that can induce daytime drowsiness by its clinical signs and by sleep laboratory tests. The illness typically manifests during adolescence and lasts the entirety of a person's life. Specific HLAs that demonstrate the presence of a gene in the major histocompatibility complex on chromosome 6 that increases susceptibility to the condition is strongly linked to narcolepsy genetic predisposition. However, it is still unclear how these aberrations relate to the inherited determinants. A dysfunctional modulation of rapid eye movement sleep may be explained by altered monoaminergic and cholinergic activities, according to neurochemical investigations of human and canine narcolepsy. Tricyclic antidepressants and stimulants are used to treat cataplexy, which significantly improves symptoms in most patients but does not completely eliminate them. The most typical neurological cause of prolonged sleepiness is narcolepsy. Great strides in the research were made as a result of the revelation around 20 years ago that narcolepsy is brought on by a selective loss of the neurons that produce orexins, also known as hypocretins. Here, we go through what is currently known about how orexin neurons control sleep-wake behavior and the effects of orexin neuron loss. I will also discuss the growing body of research suggesting that narcolepsy is an autoimmune condition that may result from a T cell-mediated attack on the orexin neurons and discuss how these fresh viewpoints can guide more effective therapeutic strategies. A growing topic of research is the interaction between narcolepsy and psychiatric problems, but sadly, this relationship is little understood. Co-morbidity between the two is not rare. However, initial misdiagnoses of narcolepsy as a psychiatric disorder are common, which adds to the lengthy wait for a proper diagnosis and course of therapy. A crippling neurological disorder called narcolepsy has a significant risk of leading to social and professional disruption. Function decline may trigger the later emergence of mental disorders. Conversely, a decline in function and quality of life may follow the onset of psychiatric symptoms.

Audience Take Away Notes

- The biological basis of the disorder Narcolepsy, and how it exactly affects the brain in which way. This may be implemented into studies in order to understand how sleep disorders may relate to each other, intertwine, and maybe even cause one another
- How Narcolepsy affects the quality of a person's life and how misdiagnoses could negatively affect someone's health alongside how misdiagnosing could lead to other mental disorders, which may deteriorate someone's health
- Helps understand what neurons are affected when it comes to the disorder narcolepsy, which may help find new ways of treatment in order to help keep the disorder under control
- The symptoms of narcolepsy and how it can be differentiated from other sleep disorders in order to conduct accurate diagnoses

Biography

Selin Edil born in 2005 is a senior student at Hisar School, in Turkey. Will graduate from Hisar Schools in 2023 June.

Zeynep Elif Olmez*, Ozlem Bozkurt
Hisar School, Istanbul, Turkey

ADHD in children - Physical and psychological effects

Attention deficit/hyperactivity disorder which is popularly known as ADHD is one of the most common mental illnesses affecting children. The effects of ADHD are known to affect a person in many areas of their life, including academic and professional success, relationships with people, and daily life activities. Inability to maintain focus, excessive movement that is inappropriate for that current environment and impulsivity are all signs of ADHD. The three different forms of ADHD are: presentation that was primarily unfocused, primarily impulsive/hyperactive behavior displayed, and combinational presentation. The existence of enduring symptoms that have developed over time and have been apparent over the previous six months is the basis for a diagnosis. The unfocused type refers to people who have trouble paying attention to a certain task for a long period of time and they are usually disorganized. The hyperactive type refers to people with too much energy that they are usually unable to remain motionless, have high levels of energy, and are chatty people. Lastly, the combinational type refers to people who are both the hyperactive and the unfocused type. There are some psychological effects of ADHD in children which also lead to physical effects. Such as shortness of attention span, leads to children acting younger than their actual age. They have difficulty growing up mentally. They struggle with listening for a long amount of time. They have a hard time finishing their current work before trying to do another activity. Also, hyperactivity leads children to have sleeping problems. As toddlers, they tend to start to walk earlier than other children. Plus, they tend to be curious and create dangerous situations for themselves. In general ADHD children have a problem controlling themselves. They have a hard time controlling their energies and obeying other people. They usually act without thinking about the consequences of their actions. Although the precise causes of ADHD have not yet been determined by science, many scientists believe it could be genetics. Lastly, there is proof that the brains of children with ADHD are anatomically different from those of children without the disorder.

Keywords: ADHD, Children, Hyperactivity, Attention, Psychology.

Biography

Zeynep Elif Olmez was born in 2005 in Istanbul, Turkey. She will graduate from Hisar High School in 2024.

Tansa Nisan Guneran*, Ozlem Bozkurt
Hisar School, Istanbul, Turkey

Neural circuit and brain structure change in women with PTSD

PTSD (post-traumatic stress disorder) is a neurodegenerative disease that is caused by tragic, unfortunate events such as violation, physical/mental abuse, and combat exposure. PTSD is linked with elevated levels of cortisol and noradrenaline (hormones) responses to stressors. The symptoms include but are not limited to, nightmares, intrusive thoughts, sleep deprivation, and memory loss. The traumatization causes the brain to remain in a state of extreme vigilance. Hence it represses verbal declarative memory concluded from neuropsychological testing measures and self-control which results in the person having severe sensitivity. Females experience twofold PTSD than males. The US National Center for PTSD has announced that almost around 8% of women have PTSD compared to 4% of men. Generally, as a consequence of PTSD's effects on the female brain, the brain regions which function in the fight or flight response including the hippocampus, amygdala, and prefrontal cortex have been structurally and functionally altered greater than man. The hippocampus becomes smaller and less likely to be active. Amygdala does not distinguish the difference between past and present trauma, which causes the production of neurochemical hormones such as cortisol. In comparison, adult women with depression and a background of early childhood abuse had a higher cortisol response to a challenging and mental challenge. The prefrontal cortex becomes less active. Women who had PTSD and had experienced violence had lower levels of blood flow in the orbitofrontal cortex, medial prefrontal cortex, anterior cingulate, and fusiform gyrus than women who had not experienced abuse and had PTSD. Other than that, increased activation in the posterior cingulate left middle frontal gyrus, malfunction in hippocampal activation, left inferior parietal cortex, motor cortex, and lastly visual association. The long-term effects in neurochemical systems observed by PET cause changes in neural circuit responsiveness in women.

Audience Take Away Notes

- The audience will now have the knowledge of how PTSD affects someone who has been abused in their earlier life. The reason that the abstract was predominantly on women is that they face and experience severe and more violated life than a man does, hence the explanation on neural circuits was upon women. Now with the knowledge of the affected parts of the neural circuit system, they will be able to syncretize this information and can work on a better treatment with less limiting results because even though there are treatments on the market, due to the gaps in information on PTSD the present treatments are only effective for a few patients or on few regions in the brain
- There are still some gaps in explaining how PTSD affects the brain and how the effects could be reversed. The audience will be able to look through another point of view which might add a bonus to their job, especially in pharmaceutical companies
- In terms of teaching, it will open more sources and ideas to teach to the new generation. Also in regard to research, a type of treatment could be developed for PTSD patients based on their altered/dysfunctional circuit

- Since how the brain circuit is affected and altered will be explained meticulously, it might open doors to a practical solution/s that can be effective on more patients and less limiting on the basis of utilization. If there are projects that are already being worked on how to reverse the dysfunctionality of the brain circuit due to PTSD then with the information explained it could simplify and improve the accuracy of the designer's job. In this presentation, it will be explained the reasons why this circuit was malfunctioning and how some of the brain regions' structures have changed over time due to the long-term effect of hormones

Biography

Tansa Nisan Gunerhan born in 2005 is a junior at Hisar School, in Turkey. I will be graduating in the year of 2024 June from Hisar School.

Begum Bulgurluoglu*, Ozlem Bozkurt
Hisar School, Istanbul, Turkey

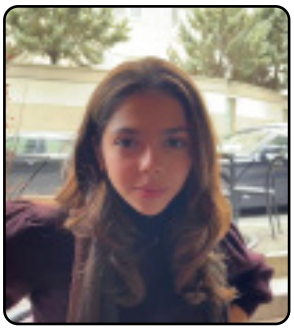
Pain control theories

Neuroscientists Ronald Melzack and Patrick Wall put forth a revolutionary new theory of pain in the 1960s. Researchers at the time were having trouble explaining the phenomenon. While some asserted that the pain signals are delivered by the intensive firing of non-specific nerve fibers, others contended that specialized nerve fibers carry pain signals up into the brain. The way that we feel pain is really complicated. Our ideas and feelings are just two of the many things that affect how we experience pain. The Gate Control Theory of Pain explains how painful sensations can be suppressed and overridden by non-painful ones. Primary afferent fibers are stimulated by a painful, nociceptive input, which is then carried to the brain by transmission cells. Pain perception increases when the transmission cells become more active. Stress and tension, mental factors, and lack of activity are the three main ways in which the gates to pain can be made more open, so that the pain feels more intense while factors such as relaxation, contentment, mental factors, activity, and other physical factors close the gate. Another theory of pain is the Labeled Line Theory of Pain: The somatosensory cortex receives a signal from the receptor as soon as pain is triggered, and the region of the cortex that the signal is sent to affects the modality of the next perception. Other pain theories include Intensity Pain Theory and Pattern Theory of Pain which defines pain as a feeling that happens when a stimulus is stronger than usual, not as a particular sensory experience and suggests that in addition to pain, other sensations may be detected by the same nerves consecutively.

Keywords: Pattern Theory of Pain, Intensity Pain Theory, Labeled Line Theory of Pain, The Gate Control Theory of Pain.

Biography

Begum Bulgurluoglu was born in 2005 in Istanbul, Turkey. She will graduate from Hisar High School in 2023.



Mia Bahar*, Ozlem Bozkurt
Hisar School, Istanbul, Turkey

The basis of decision-making

Our days are assembled from thousands of small decisions: what to do, which way to go, how to respond, whether to partake, and our brain is made of many competing networks each with its own goals and desires. One neuron itself doesn't have an efficient power. Though, when many neurons connect with each other their relationship becomes unmatched, complicated, and cyclical. When we have to make a decision, in a complex network of neurons one group of neurons will represent one choice and the other will represent the second choice. When making a decision our 5 senses work together to connect the choice to a memory or an experience. One thing certain about the decision-making process are results of communication between the prefrontal cortex (working memory) and hippocampus (long-term memory). Our emotions also play a big role in our decision-making. Without considering our emotions, our decisions wouldn't be as good as others. As much as emotions our body plays a big role in decision-making. It's easy to think about the brain commanding the body from on high – but in fact, the brain is in constant feedback with the body. The physical signals from the body give a quick summary of what's going on and what to do about it. To land on a choice, the body and the brain have to be in close communication. (Brain, David Eagle man) Your physical condition is a description of the scenario that aids you in your activity and your decision-making. Your physiological profile can be compared to a low-resolution headline that says either this is bad or this is not a problem. That aids your brain in making the following decision. One thing when our brain is making decisions is the power of now and the future. Values are associated with various possibilities. There is a twist that frequently prevents us from making wise decisions: we frequently place a higher value on the possibilities that are actually in front of us than those that we only simulate. The present is what gets in the way of making wise choices for the future. The brain can only ever see the future as a faint shadow of the present. The influence of the present explains why people make choices that seem good at the time but have negative outcomes later on. The seduction of now while we make decisions is in fact real. In order to prevent our future selves from acting inappropriately, people arrange the present. We can avoid being seduced by the moment by binding ourselves to the present. Everything we do, who we are, and how we view the world around us revolve around our decisions. We would be held captive by our most fundamental drives if we couldn't weigh our options. With no ability to plan for the future or navigate the here and now, we would be powerless. We may improve our decision-making for ourselves and for society by being more aware of how alternatives compete for attention in the brain.

Biography

Mia Bahar was born in 2007 in İstanbul, Turkey. She will graduate from Hisar High School in 2025.

Lara Hanci*, Ozlem Bozkurt
Hisar School, Istanbul, Turkey

Human brain mapping - The golden future of neuroscience

The brain, being the most complex organ in the human body, can't be completely understood with today's technology. Therefore, there mostly aren't certain cures for brain diseases such as Alzheimer's disease, dementias, brain cancer, epilepsy, mental disorders Parkinson's disease and stroke, but only treatment methods. These treatment methods don't offer a permanent result, suggesting that unless there is an exact cure, it is very hard for a patient with a brain disorder to be completely healed. In order to overcome this impediment, new technology to visualize the brain is being developed. Brain scan studies on an unprecedented scale were prompted by the search to comprehend how hereditary materials influence human brain development and disease. Researchers from all around the world worked together to generate genetic maps of the human cortex by performing statistically robust assessments of common and unusual genetic variants on brain measurements and rates of brain development and aging. One significant invention regarding these types of studies is called the Human Brain Mapping. Its history goes back to early 1990s. Main techniques used to map the brain include positron emission tomography, neoplasm, magnetic resonance imaging, transcranial magnetic stimulation, neuroimaging, stimulation and electroencephalogram. Recently, the Allen Institute organized a new international cooperation to map the roughly 200 billion cells in the human brain according to their type and function, just like how Human Genome Project mapped the entire genetic code. According to a member of the group led by Ed Lein, Ph.D., Senior Investigator at the Allen Institute for Brain Science, This is critical work: We need to understand the human brain better if we hope to treat diseases of the brain, and specifically we need a better understanding of brain function and structure. The cell atlases we're building with the support of the BRAIN Initiative promise to lead to a more rapid understanding of the basis of many brain diseases. This invention can be considered as the future of neuroscience as it can provide answers and cures to brain disorders.

Keywords: Brain, Brain disorders, Human brain mapping.

Audience Take Away Notes

- What is known and unknown about the brain
- Types of vital brain disorders and their possibility to be cured by human brain mapping
- History of human brain mapping
- Techniques used in human brain mapping
- Where can human brain mapping be used in future

Biography

Lara Hanci was born in 2006 in İstanbul, Turkey. She will graduate from Hisar High School in 2024.

Lara Ann Tureli*, Bozkurt
Hisar School, Istanbul, Turkey

The application of brain-machine interface for prosthetic limb development

The applications of Brain-Machine Interface (BMI) are relatively recent. It is believed that BMI first started being researched and tested in the 1970's. In its first applications, noninvasive methods were used to control a cursor- like graphical object on a machine screen through the transmission of electrical signals that were collected from the brain. Currently the applications of BMI are mainly researched in the medical field for development of robotic prosthetic limbs, awareness detection for people in a coma or vegetative state, and recovery for patients who suffered a stroke. The use of BMI in neural prosthetics and bionic limbs is based on detecting and quantifying brain signals from the patient. After this the BMI translates the patient's intent to device commands, making the prosthetic limb function as if it is a part of the patient's body. This system consists of four main steps: signal acquisition, feature extraction, feature translation, and device output. Signal acquisition is generally collected through non-invasive methods such as electroencephalograms (EEG's) or invasive methods like electrocorticography (ECoG). To provide the most productive signal acquisition, the limb being replaced and the functions of that limb are both considered in order to choose the most optimal placement of the device (e.g. EEG or ECoG). Feature extraction, the next step, is the process by which the signal collected from the patient is analyzed to determine whether the signal can be used for the prosthetic limb or if it is a sort of extraneous content. Through feature translation the collected signal features are passed onto a translational algorithm where the brain signals are converted into commands for the prosthetic limb. Lastly these commands are applied in the prosthetic limb resulting in the desired movement. In order to translate the signals collected from the brain, BMI systems rely on machine learning. Therefore, development in machine learning and artificial intelligence will also benefit the quality of BMI systems. Neural prosthetics go beyond device output; they also aim to send signals collected from the prosthetic limb to the brain. Through this, the patient not only can regain motor skills but also regain a certain level of sensation. The restoration of natural sensory feedback from the prosthetic limb to the patient is still being researched and developed. If this restoration can be provided the amputee could live a safer and healthier lifestyle. Therefore, further research and development of BMI systems can increase life quality, recovery rates, and further research in neuroscience.

Audience Take Away Notes

- The audience will have a general understanding of how a brain-machine interface works and the fields in which it is used
- The audience will learn about both the benefits and limitations of brain-machine interface systems
- The audience will learn about how this system is used to develop more efficient and optimal robotic prosthetic limbs
- The audience will understand the role of machine learning and artificial intelligence in the development of brain machine interface systems

- Through this presentation the audience will have a better understanding of how machine learning, artificial intelligence, and brain-machine interface systems can be developed in new fields of research, allowing for new developments in the field of neuroscience

Biography

Lara Ann Tureli was born in 2006 in Istanbul, Turkey. She will graduate from Hisar High School in 2024.

Arda Ozkurt
Hisar School, Istanbul, Turkey

Optogenetics on nonhuman primates

Optogenetics is a method to activate or deactivate genetically modified neurons by using light. It has been a useful tool to discover new neural circuits since it was first used in 2004. In the early experiments, especially mice, rats, and fruit flies were used. These experiments contributed to our knowledge of the neural circuits in the brain broadly. Since then, new opsins were discovered, new methods to deliver opsins into the brain, and less invasive optogenetics techniques were developed. Optogenetics experiments still cannot be conducted on humans. However, one of the most essential improvements in the field to understand the human brain without conducting experiments on humans was to start conducting optogenetics experiments on nonhuman primates (NHPs). Experiments on NHPs provided us to discover new neural circuits more similar to the human brain and predict the consequences of a possible optogenetics experiment on humans. The experiments on NHPs differ from those on rodents in some ways. Even though conducting optogenetics experiments on NHPs provides us with a better understanding of the human brain than of rodent brains, it has several obstacles to working on NHPs due to some reasons. First, it is harder to target the specific regions of an NHP's brain than of a rodent's brain because an NHP's brain is more complex than a rodent's. Moreover, rodents' DNAs are less complex, so neurons being modified to be light-sensitive in a primate brain is tougher than in a rodent brain. In addition, implanting fiber optics is a significant issue when conducting an optogenetics experiment on NHPs. Therefore, it is necessary to use non-invasive techniques to deliver light into a primate's brain, which makes the experiment more demanding. Essentially, conducting optogenetics experiments on NHPs has been and will continue to be one of the leading methods in neuroscience research to understand the human brain. Furthermore, testing these experiments on NHPs will make optogenetics experiments and treatments on humans possible, which might be crucial to find solutions for neurodegenerative diseases, such as Parkinson's disease and anxiety, in humans and for discovering new neural circuits to understand human behaviors more comprehensively in the future.

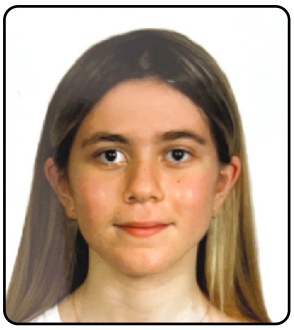
Keywords: Optogenetics, NHPs, Neural Circuits, Opsin Delivery.

Audience Take Away Notes

- The significance of optogenetics on NHPs
- The main issues of optogenetics on NHPs
- Less invasive optogenetics techniques to be used on NHPs
- Optogenetics as a tool to discover new neural circuits

Biography

Arda Ozkurt was born in 2006 in Istanbul, Turkey. He will graduate from Hisar High School in 2025.



Nil Atay*, Samet Teke
Enka Schools, Istanbul, Turkey

How trauma changes the brain

Researchers are learning more about how traumatic events affect our brains physically. Changes in a brain mechanism used for learning and survival have been discovered by neurologists to play a role in how someone responds to a threat following a traumatic experience. Another study discovered that a mechanism involved in emotion and memory is impacted, making it difficult for someone suffering from Post-Traumatic Stress Disorder (PTSD) to distinguish between safety, danger, and reward. It overgeneralizes in favor of danger. The hippocampus, amygdala, and medial prefrontal cortex are thought to be key players in PTSD. Two neurochemical systems that are vital to the stress response are cortisol and norepinephrine. These findings have the potential to significantly advance future treatments. The delicate chemical structure and equilibrium of the brain can be altered by traumatic stress. Depending on the type of traumatic stress we are experiencing, these consequences, which may affect how we function, can range from mild to severe. For instance, some individuals get PTSD, which affects 8% of Americans at some point in their lives and can also cause depression, substance misuse, dissociation, personality problems, and health issues. Even though the brain is extremely complicated, one of its main jobs is to keep us safe. Our brain turns experiences into memories as we move through life so that we can give higher priority to activities that produce positive outcomes and steer clear of those that do not. Our brains work extra hard to keep us safe after tragedy. Long after the threat or traumatic experience has passed, the brain continues to alert us of the present danger based on negative patterns of the past. The way the brain functions is altered by this response. Furthermore, trauma is a life changing event which affects humans and their brains in many ways.

Keywords: Trauma, PTSD, physical, Brain.

Audience Take Away Notes

- The significance of trauma in human neurological system
- The main issues of trauma affecting the brain
- Hormonal pathway related to trauma

16-17 JUNE

DAY 02

VIRTUAL ROOM 01

KEYNOTE FORUM

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

Neuro Physics Thearpy (NPT) shows to be a highly effective psychophysical treatment for its patients enduring advanced Facioscapulohumeral Muscular Dystrophy (FSH, FSHD) symptoms, with significant sustainable generation of lost or highly compromised functions being realized in very small time scales

Facioscapulohumeral muscular dystrophy (FSHD) is one of the most frequent hereditary muscle disorders. It is said to be a 'genetic' muscle disorder in which the muscles of the face, shoulder blades, and upper arms are among the most affected. This presentation will include the outcomes of four FSHD patients (case studies) who all presented suffering from varying degrees of advanced FSHD symptoms. The length of time since these patients were diagnosed with FSHD varied from 30 years pre-NPT treatment to 4 years pre-NPT treatment, with the most phenomenal sustainable generation of lost or highly compromised functions being realized in the patients having the longest-term diagnosis pre Neurophysics Therapy treatment. All patients received NPT treatment over four consecutive days involving 2-hour treatments each day. This presentation will also include published data and video testimonials. The profound outcomes for each of these patients excites obvious questions and levels of inquiry. Each patient's symptoms progressively advanced since their diagnosis despite pharmacological intervention. 'There is a consensus that FSHD is caused by the aberrant production of the double home box protein 4 (DUX4) transcription factor in skeletal muscle. DUX4 is normally expressed during early embryonic development, and is then effectively silenced in all tissues except the testis and thymus. Its reactivation in skeletal muscle disrupts numerous signaling pathways that mostly converge on cell death' Kenji Rowel Q. Lim, if this is the case, than how is possible for these patients to generate such profound outcomes and in such very small time frames? This will be discussed during this presentation.

Keywords: Facioscapulohumeral muscular dystrophy (FSHD), Neuro Physics Therapy Treatment, Genetic Disorders, Psychophysical Treatment.

Ken Ware

Head of Therapeutic Research and Development, Founder of Neurotricional Sciences Pty Ltd and NeuroPhysics Therapy, Gold Coast, Queensland, Australia

Biography

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

Neuroimaging by evaluation nerve repair and remodeling of acupuncture in children with cerebral palsy

Objective: To investigate the effect of and Acupuncture on brain plasticity and motor development in children with cerebral palsy. Investigate effect on mechanism of apoptosis of brain nerve cells, regulating the expression of neurotrophic factors, promoting the remodeling of nerve synaptic structure and motor development in young rats with cerebral palsy. Two: To evaluate the effect and mechanism of acupuncture on cerebral palsy. Three: The nerve repair effect of acupuncture on cerebral palsy. Methods: In this study, 146 cases of brain injury and 1078 cases of cerebral palsy were included by randomized controlled study with ICF Gross motor function measure, Peabody fine motor function, Gesell, muscle tension, joint activity, activity of daily living transcranial doppler,, skull B ultrasound, Brain Nuclear Magnetic Resonance Imaging MRI, Positron Emission Tomography SPECT, Diffusion tensor tractography evaluation method.

Results: the recovery rate of extracellular space (92.3%) was significantly higher than that of the control group (70.8%) (P <0.05), Transcranial Doppler, TCD total efficiency (79.3%) was significantly higher than that in the control group (51.8%) (P <0.05). Acupuncture to promoting the development of neurological and cognitive movement under 6 months children, effectively reduce the neurological sequelae. The total effective rate of the children with cerebral palsy was 87% in the acupuncture group, which was significantly higher than that of the control group (P <0.01). The total effective rate of Brain MRI was 59.55% in the acupuncture group and 13.25% higher than that in the control group (P <0.01). The total effective rate was 91.3% in the 1 year follow-up group, which was significantly higher than that in the control group (P <0.01). The FA value of white matter fiber bundle was significantly higher than that of acupuncture at 60 times (P <0.05). The recovery rate of ultras nous brain injury (86.7%) in acupuncture group was significantly higher than that in control group (64.4%) (P <0.05). The recovery rate of brain SPECT in acupuncture group was 96.4%, which was significantly higher than that in the control group (P <0.01).

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

Keywords: Cerebral palsy, Acupuncture, Nerve repair, Remodeling, Motor function.

Zhenhuan LIU

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

Biography

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

Addiction: A problem of motivation, free will, or self-destructive behavior?

Popular stereotypes of addiction emphasize loss of free will, sometimes based on irresistible urges. The recently popular brain disease theory of addiction likewise emphasizes on the inability to stop using despite wanting to quit. But evidence suggests that addicts retain voluntary control over their actions, and cravings are generally weak and resistible. This talk integrates many findings from the research literature on addiction to develop the idea that addiction has somewhat separate effects on liking, wanting, and doing, any of which can derail attempts to quit. Improved understanding of addiction may require replacing the long-running quest for a single formula with a pluralistic framework.

Roy F Baumeister

University of Queensland, United States

Biography

Roy F. Baumeister is one of the world's most prolific and influential psychologists. He has published over 700 scientific works, including over 40 books. In 2013, he received the highest award given by the Association for Psychological Science, the William James Fellow award, in recognition of his lifetime achievements. He is currently president-elect of the International Positive Psychology Association and has ties to the University of Queensland (Australia), Florida State University (USA), and the University of Bamberg (Germany). Although Roy made his name with laboratory research, his recognition extends beyond the narrow confines of academia. His 2011 book Willpower: Rediscovering the Greatest Human Strength (with John Tierney) was a New York Times bestseller. He has appeared on television shows such as Dateline NBC and ABC's 20/20, as well as on PBS, National Public Radio, and countless local news shows. His work has been covered or quoted in the New York Times, The Washington Post, The Wall Street Journal, Los Angeles Times, The Economist, Newsweek, TIME, Psychology Today, Self, Men's Health, BusinessWeek, and many other outlets.

Live test for chronic wasting disease based on consistent association with an extreme thermoacidphilic bacterium

The problem with controlling chronic wasting disease (CWD) infection in ruminants is there is no reliable live test for this devastating fatal encephalopathy. Our strategy is to use a disease biomarker to develop a live test for CWD. The most recognized biomarker of CWD and other transmissible spongiform encephalopathies (TSE) is prion amyloid that accumulates in brain and spinal cord during the course of the disease. However, attempts developing a live test for CWD based upon detection of the prion amyloid protein has been troublesome since the quaking test for identification of prion biomarker has proved to be less than satisfactory as the test is only 50% accurate. As used in the diagnosis of CJD in humans, the test shows numerous false positives. A problem with this approach is the prion amyloid is absent in 5% of Creutzfeldt-Jakob disease (CJD) cases, and the new variant of scrapie in sheep does not produce prion amyloid. The concept of the quaking test does not fit with the prion theory since severe shaking of the unknown brain sample in the presence of normal prion isoform may lead to increased prion amyloid, likely by self- assembly, while there is no increase in infectivity. Nonetheless, much of the monies appropriated by the USDA go to efforts of improving the quaking test rather than trying to develop alternate methodology. Since the protein sequence of the normal prion protein isoform on the cell surface is identical to that of the disease-associated prion amyloid, a credible diagnostic test based on this strategy will likely not be practical. A second biomarker is the scrapie-associated fibrils (SAF) that are present in 100% of TSE cases, even in absence of the prion amyloid. However, the use of this biomarker is impractical since SAF is identified by negative stained electron microscopy in synaptosomal ultracentrifuge fractions. Prion researchers have declared SAF to be the morphological marker of prion amyloid, but this is total conjecture. It is noteworthy that SAF are identical to fibril proteins within a third bacterial biomarker of TSE infection and show immune cross-reactivity. Our laboratory has found the consistent association of a wall-less bacterium with CWD and other TSEs, documented by morphological and molecular studies. In fact, we are able to isolate this novel Spiro plasma bacterium from 100% of CWD-affected tissues. The biological properties of this isolate are identical to those of the transmissible TSE agent including surviving boiling for one-hour, massive doses of gamma irradiation, formalin exposure for 18 hours, standard autoclaving and hyperacidity (pH 2). This novel bacterial isolate grows in cell free media and forms subsurface colonies on special agar plates prepared with Brucella media. These extreme thermoacidophiles represent a third biomarker of CWD infection and provide the best opportunity to develop a live test since they are foreign to the animals. Our strategy is to identify a protein epitope on the bacterial surface that can be used as a diagnostic serological test. That protein may also provide immune protection as a vaccine against CWD and other TSEs.

Frank O. Bastian MD

Research Professor Adjunct Department of Pathology, Tulane Medical School, New Orleans, Louisiana, United States of America

Research Scientist, CEO, Bastian Enterprises LLC, 2000 Lakeshore Drive, James Lynch BSc Research Technician, Bastian Enterprises LLC

Biography

Dr. Bastian studied Medicine at University of SASK, Canada (1960-1964). After internship at Charity Hospital, New Orleans (1964-1965) and brief time in general practice, he took residency in Neuropathology at Duke College of Medicine (1968-1972). He was Research Fellow in Dr. Rabson's Virology Research NIH laboratory in Washington DC (1972-1973) then joined department of Pathology at Baylor College of Medicine as Assistant Professor (1973- 1980). He moved to University of Maryland as Associate Professor and director of Neuropathology. Then to University of South Alabama as Professor in 1982. He became Research Professor in Pathology at Tulane Medical School in 2002.

Audience Take Away Notes

- The information presented will alert the audience that the wrong science is currently being used to address the CWD panoptic in wild and domesticated ruminants including deer, elk and reindeer
- The research also applies to the fatal CJD in humans. The prion amyloid is simply a reaction product created by the bacterial infection. Watarai et al., have shown the normal prion isoform acts as a receptor protein for a bacterium
- The audience with this information can approach TSE diseases as potentially curable with development of antibiotics and/or vaccines. A workable live test for CWD will aid in the management of the panoptic with the possibility of removing this animal reservoir of CWD infection therein preventing spread to humans
- Hopefully this presentation will result in further research investigations using this approach

Using expressive arts exercises to promote self-care among neurologists and caregivers: An experiential overview

Research suggests that the cumulative stress of caring for a person with a chronic neurological disorder can contribute to burnout, both among physicians (physician burnout) and patient families (caregiver burnout). Expressive arts techniques have been used in a variety of settings to help participants cope with overwhelming feelings and to promote resilience, including in the medical practice of neurology.

Audience Take Away Notes

- Participants will become familiar with various expressive arts disciplines, including visual art, drama, music, creative writing, and movement, and have the opportunity to engage in sample exercises from each discipline
- Participants will be instructed on incorporating mindfulness into wellness activities
- Participants will be encouraged to adapt techniques, such that they can be shared with caregivers for persons with chronic neurological illnesses

Juliana Fort[1*], MD, MPH, MBA, Kendy[l] Arden[2], Eleanore Knox[2], Michael Kenny[3], Oleg Chernyshev[4], MD, Justin Hardin[5], Shavonne Temple[5], Joshua Woo[5]

[1]Department of Psychiatry, LSU Health Shreveport, Shreveport, LA, USA

[2]MD LSU Health Shreveport, Shreveport, LA, USA

[3]Masters of Music Therapy, MT-BC, TKTN, LMT

[4]Associate Professor Department of Neurology and Sleep Medicine, LSU Health Shreveport, Shreveport, LA, USA

[5]MS, LSU Health Shreveport, Shreveport, LA, USA

Biography

Juliana Fort, MD, MPH, MBA, is the medical student clerkship director and a Clinical Associate Professor in Psychiatry at LSUHS in Shreveport. She is board certified in child and adolescent, geriatric, forensic, and addiction psychiatry. Dr Fort also has an MA in mental health counseling with a specialization in drama therapy from Lesley University and a MA in creative writing. She is a play therapist / supervisor and enjoys co-facilitating workshops with students and colleagues, and psychiatry/neurology residents. Interests include expressive arts, improvisation, Wellness, medicine and the arts/humanities, psychotherapy, and personal growth.

BOOK OF ABSTRACTS

magnus Group

16-17 JUNE

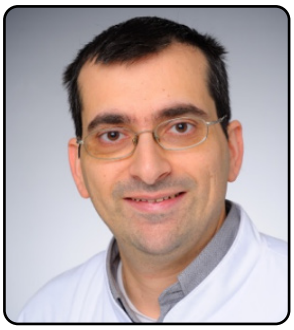
DAY 02

VIRTUAL ROOM 01

SPEAKERS

JOINT EVENT ON

NEUROLOGY AND DEMENTIA



Georgios Matis MD, MSc, PhD, FINR (CH)
University of Cologne, Faculty of Medicine and University Hospital Cologne, Department of Stereotactic, Functional Neurosurgery, 62nd Kerpener Str, 50937, Cologne, Germany

Intrathecal pain treatment & ziconotide

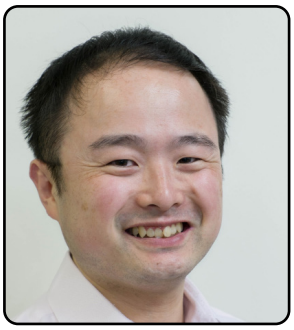
Ziconotide is a synthetic, water-soluble cone snail venom-derived peptide with a molecular weight of 2,639 Daltons. It is a nonopioid analgesic that selectively binds to N-type voltage-sensitive calcium channels on primary nociceptive afferent nerves in the dorsal horn of the spinal cord. This mechanism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

Audience Take Away Notes

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

Biography

Dr. Georgios Matis is a senior consultant for neurosurgery. He leads the chronic pain / spasticity sector of the Department of Stereotactic & Functional Neurosurgery in the University Hospital of Cologne. He has been trained in Greece (General University Hospital of Alexandroupolis, G. Papanikolaou General Hospital of Thessaloniki & 417 Army Equity Fund Hospital of Athens), USA (Department of Neurosurgery, Weill Cornell Medical College, New York, and NY), Switzerland (Department of Neuroradiology, University Hospital of Zurich, and Zurich) and Germany (Department of Stereotactic & Functional Neurosurgery, University Hospital Cologne, and Cologne). Dr. Matis is a member of two medical associations (Thessaloniki, Greece & North Rhine, Germany) and also a member of the German Neuromodulator Society (DGNM) and the International Neuromodulation Society (INS). He serves as reviewer for many international journals and is Editorial Board member for Neuromodulation: Technology at the Neural Interface and Interventional Pain Medicine and Neuromodulation. He holds the position of Editor-in-Chief of the Internet Journal of Neurosurgery. Dr. Matis has published many articles in Greek and international Pubmed-indexed journals and hold many lectures as invited speaker in numerous international congresses and webinars. At the same time, he is Public Education Committee member of the International Neuromodulation Society. Dr. Matis is involved in many international clinical studies and has been active as instructor for many colleagues in Germany and abroad. He is also an active member of the medical advisory board of the German CRPS Support Group and member of several online consultation platforms. He is actively involved in social media trying to raise awareness about spinal cord stimulation and neuromodulation.



Narinobu Juge
Advanced Science Research Center, Okayama University, Okayama, Japan

Identified new regulation of vesicular neurotransmitter transporters

Neurotransmitter is an essential for synaptic transmission in the mammalian central nervous system, and is involved in all brain functions and in various neurological diagnoses. Vesicular neurotransmitter transporters (VNTs) are one of major syanptic vesicle proteins. They transport neurotransmitter into synaptic vesicles for excitatory, inhibitory or emotional signal transmission in the central nervous system. VNTs are located not only on synaptic vesicles, but also on the plasma membrane during the transduction of neurons. VNTs are also involved in various biochemical regulation processes. I have clarified the some of molecular mechanisms of VNTs, I will talk about the lipid regulation of VNTs in this presentation.

Presynaptic terminals tightly control their lipid composition, and each lipid markedly regulates neuronal activity. The lipid compositions and protein orientations are different among synaptic vesicles and plasma membranes in presynaptic terminals and VNTs are localized on both membranes. Several physiological and pathological conditions induce changes in the lipid composition, and lipidomic changes are associated with neurological and psychiatric disorders.

In the present study, we demonstrate the lipid induced transporter activity and conformational changes. And this lipid regulation is linked to the transporter cycle during the neurotransmission. That is, VNTs actively accumulate neurotransmitters in synaptic vesicles, but upon exocytosis, they abruptly stop their transport and relase excess neurotransmitters. Because neurological and psychiatric disorders are also caused by abnormalities in vesicular neurotransmitter transporters, the lipidomic regulation of these transporters will serve as appropriate drug targets for these disorders. Thus, our findings could provide novel strategies for the treatment and prevention of neurological and psychiatric disorders.

Audience Take Away Notes

- For the biochemist, our research provides the new methodology of lipid-membrane protein interaction
- For the neuroscientist, our research provides the new lipidmic regulation of VNTs
- For the clinical person, our research provides the new drug target of several neurological disorder

Biography

I studied Pharmacological science at the Daiichi pharmaceutical University, Japan and graduated as BS in 2004. Then joined the research group of Prof. Yoshinori Moriyama at the Department of Molecular Membrane Biology, Okayama University Graduate School of Medicine, Dentistry, Pharmaceutical Sciences. I received her PhD degree in 2009 at the same institution. After two and half years postdoctoral fellowship supervised by Dr Robert Edwards at the UCSF, I obtained the position of an Assistant Professor at the Okayama University.

Xiaoyu Chen*, Qi Guo
Department of Rehabilitation Medicine, Shanghai University of Medicine and Health Sciences, Shanghai, China

Relationships between sarcopenia, depressive symptoms, and mild cognitive impairment in Chinese community-dwelling older adults

Background: Mild cognitive impairment (MCI) represents an intermediate state between normal cognitive aging and dementia. We aimed to investigate the asso- ciation and mediation pathways of sarcopenia, including its individual components (muscle mass, muscle strength, and physical performance), and depressive symptoms with MCI in the older adults.

Methods: This cross-sectional study consisting of 1394 community-dwelling Chinese older adults aged 60 years and older in Tianjin and Shanghai, China. Sarcopenia was defined according to the Asian Working Group for Sarcopenia (AWGS) criteria. Depressive symptoms were evaluated by the 30-item Geriatric Depression Scale (GDS). Cognitive function was assessed by Mini-Mental State Examination (MMSE), the Chinese version of the Dementia Rating Scale (CDRS) was used to apply the diagnostic of non-dementia, and instrument activities of daily living (IADL) were used to evaluate daily living activities. Logistic regression and mediation analyses fully adjusted for all potential confounding factors were conducted.

Results: Sarcopenia, handgrip strength, gait speed, and depressive symptoms were associated with MCI. Furthermore, depressive symptoms significantly mediated the association of sarcopenia, handgrip strength, and gait speed with cognitive function. The relationship of depressive symptoms and cognition were also mediated by sarcopenia, handgrip strength, and gait speed.

Conclusions: Our findings suggest that sarcopenia may contribute substantially to the development of MCI in the older adults via depressive symptoms, although the reverse may also be true. These findings may help guide clinicians to better diagnose and manage MCI in the context of concomitant sarcopenia and depressive symptoms.

Audience Take Away Notes

- Sarcopenia, handgrip strength, gait speed, and depressive symptoms were associated with MCI
- Depressive symptoms significantly mediated the association of sarcopenia, handgrip strength, and gait speed with cognitive function. The relationship of depressive symptoms and cognition were also mediated by sarcopenia, handgrip strength, and gait speed
- These findings may help guide clinicians to better diagnose and manage MCI in the context of concomitant sarcopenia and depressive symptoms

Biography

Dr. Xiaoyu Chen studied Rehabilitation medicine at the Tianjin Medical University, Tianjin and graduated as PhD in 2021. She then joined the research group of Prof. Qi Guo at the Department of Rehabilitation Medicine, Shanghai University of Medicine and Health Sciences, Shanghai. Her main research direction is clinical psychology. She has published more than 10 research articles in SCI (E) journals.

Siqi Peng[1*], Yan Zhuang[1], Wenzhen Gu[1], Xiuqi Yang[1], Yaochen Lv[1], Sibie Meng[1], Wenxiu Zhu[1], Wei Xie[1, 2], Moyi Li[1, 2]

[1]School of Life Science and Technology, The Key Laboratory of Developmental Genes and Human Disease, Southeast University, Nanjing, Jiangsu, China
[2]Jiangsu Co-innovation Center of Neuroregeneration, Nantong University, Nantong, Jiangsu, China

A new AAV tool specifically targeting hippocampal CA2

Mice hippocampus contains three prominent subregions, CA1, CA3 and DG and is well regarded as an essential multiple task processor for learning, memory and cognition based on tremendous studies on these three subregions. The narrow region sandwiched between CA1 and CA3 called CA2 has been neglected for a long time. But it raises great attentions recently since this region manifests the indispensable role in social memory. Its unique physical position connecting CA1 and CA3 suggests the potential novel functions besides social memory regulation. But the CA2 is too small to be accurately targeted. A flexible AAV tool capable of accurately and efficiently targeting this region is highly demanded. To fill this gap, we generate an AAV expressing Cre driven by the Map3k15 promoter (AAV/M1-Cre) which can be easily utilized to help tracing and manipulating CA2 pyramidal neurons. Furthermore, M1-Cre successfully labeled a group of Map3k15+RGS14- pyramidal neurons that do not colocalize with any RGS14+/STEP+/PEP4+/Amigo2+ pyramidal neurons. These Map3k15+RGS14- pyramidal neurons have more complex spines than previously reported. They are probably the ones projecting to the revealed CA2 downstream targets, VMH, STHY and PMV in WT mice injecting this AAV/M1-Cre virus but not in Amigo2-Cre mice. Altogether, this tool provides a completely new, more flexible and extended strategy for in-depth CA2 functional study in the future.

Audience Take Away Notes

- We successfully developed a CA2 specific Cre viral system (AAV/M1-Cre) to label and manipulate neural activity in CA2 region
- AAV/M1-Cre can efficiently drive CA2 pyramidal neurons as what Amigo2-cre transgenic mice does. Meanwhile, it even displayed an extraordinary CA2 targeting accuracy comparing to most of current available targeting systems for CA2 and it is much more flexible to be utilized into different mice to realize the CA2 specific manipulation and gene deletion
- We labeled a group of pyramidal neurons containing both Map3k15+RGS14- and Map3k15+RGS14+ neurons in Py layers of CA2
- Complex spine structures and highly diverse morphology of pyramidal neuron cell bodies can be observed not only in Map3k15+RGS14- neurons in CA2 Py layers, but also in Map3k15+RGS14+ or regular CA2 neurons in Py layer sometimes

Biography

Ms. Peng studied Biotechnology at the Huzhou University and graduated as MS in 2016. She then joined the research group of Prof. Li at the School of Life Science and Technology, The Key Laboratory of Developmental Genes and Human Disease, Southeast University. She currently is working on a Ph.D.

Lihong Li[1*], Zitong Xu[2]

[1]Research Dept.,The Second Affiliated Hospital of Zhejiang Chinese Medical University, Xinhua Hospital of Zhejiang Province, Hangzhou, China
[2]The Second Affiliated Hospital of Zhejiang Chinese Medical University, Xinhua Hospital of Zhejiang Province, Hangzhou, China

Sleep medicine: Modern medical research and acupuncture therapy

Objective: Chronic insomnia disorder (CID) affects people's daytime function and is also a risk factor for many diseases. Targeted drugs have obvious side effects. The therapeutic effect of acupuncture on CID has been confirmed by clinical studies, but there is still a lack of objective data verification.

Methods: Based on subjective-objective methods (PSQI-actigraph), we first conducted an observational study included 217 patients with CID and the clinical characteristics and the factors that aggravate CID were analyzed (study 1). We then performed an intervention study to observe the therapeutic effects of acupuncture on 61 patients with CID (study 2).

Results: Study 1 showed the male: female ratio was nearly 1:2 and patients with underlying diseases, depression and anxiety accounted for 26.7%, 39.2% and 37.3, respectively. Symptom-based stratification (severe CID: PSQI>7 but ≤14; mild CID: PSQI≥15) from subjective results showed the age of severe CID group was significantly older than those in mild CID group (P=0.000), and the ratio of male to female was around 1:1.6-2. Sub-items of PSQI scale including sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disorders, hypnotic drug usage and daytime dysfunction in severe CID group were more severe than those in mild CID group (P=0.000 or 0.002); binary logistic regression results indicated sleep quality (OR: 50.635; 95%CI: 9.078-264.102, P=0.000), sleep duration (OR: 6.747; 95%CI: 2.365-19.249, P=0.000), sleep efficiency (OR: 10.336; 95%CI: 2.899-38.865, P=0.000), hypnotic drugs (OR: 16.056; 95%CI: 5.7-45.229, P=0.000) and daytime dysfunction (OR: 27.643; 95%CI: 7.576-100.857, P=0.000) were the related factors of CID symptom aggravation. Age-based stratification showed the younger (16-44y) had lower sleep efficiency (P=0.015) and shorter sleep duration (P=0.001). Gender-based stratification showed females had longer sleep duration than males (P=0.049). The objective results from actigraph showed the difference in the number of movements during sleep (P=0.014), wake after sleep onset (P=0.009) and number of awakenings (P=0.001) of the older group (≥45y) when compared with the younger group. The sleep duration (P= 0.048) and total time in bed (P=0.02) of females were significantly longer than those of males. Study 2 showed acupuncture significantly improved sleep quality (P=0.000), sleep duration (P=0.000), sleep efficiency (P=0.000), sleep disorders (P=0.001), hypnotic drug usage (P=0.002) and daytime dysfunction (P=0.003) in patients with CID.

Results from actigraph indicated acupuncture significantly improved sleep efficiency (P=0.042), fragmentation index (P=0.009) and sleep fragmentation (P=0.03) in patients with CID.

Conclusion: There were age and sex differences in the incidence of CID. Acupuncture can significantly improve various symptoms of patients with CID. In the future, more attention should be paid to the stratified efficacy and mechanism of acupuncture intervention on CID, so as to better promote the clinical application of acupuncture intervention in CID.

Audience Take Away Notes

- In terms of age, although the physiology of sleep changes with aging, insomnia that impairing daytime functions (including work ability, daily life, etc.) deserves attention and early intervention
- In terms of gender, despite the widespread concern about sleep quality from all human beings, women seem to pay more attention to the duration of sleep they get, which is related to the physiological processes they go through during their lifetime (menstruation, pregnancy, menopause, etc.). Therefore, paying attention to women's sleep health during special periods may help them manage their physical and mental health
- The side effects of conventional hypnotic drugs cannot be ignored, while the CBT-I strategy, a first-line therapy of insomina, faces the challenge of popularization. In contrast, acupuncture has unique advantages in the treatment of insomnia. It can not only improve various symptoms of insomnia effectively, but also has a good effect on the comorbid health problems accompanied by insomnia due to its attribution of multisystem regulation. In addition, acupuncture has been confirmed safe, convenient and economical, which is a superior selection for patients with insomnia. However, due to the challenge of research strategy and specific theory of acupuncture itself, more studies with high-quality evidence are needed to promote its clinical application
- The strategy based on the combination of subjective and objective methods on the studies of sleep disorders has long been recommended. Despite the symptoms of CID are not as obvious as other sleep disorders, say, sleep apnea and REM sleep behavior disorder, researchers can also find some indirect evidence from the results obtained from objective technique, such as actigraph and polysomnography). For example, the index of movement during sleep and the movement profile during daytime. Then, is there a possible way to figure out the relations between changes of diurnal movement and the onset and persistence of insomnia and how it can promote the mining of the positive effects exerted from exercise or anything like that? Take it a step further, how does acupuncture alleviate insomnia from the aspect of the regulation of dynamic and static states
- Polysomnography is a golden standard of objective data obtainment of sleep disorders because of the superiority from multivariate detection. However, it is not that convenient for all scenes. In addition, the avoidance of sleep disturbance caused by the device itself should be noted. Portable devices like actigraph is also recommended to be introduced to sleep disorder-related study despite the functional differences compared to polysomnography. Select an appropriate strategy based on the scientific problem that someone wants to solve can maximize the utilization of a certain device and expounds reasonable explanations. Reasonable research design is of great importance of the study of acupuncture effects and mechanism on insomnia alleviation
- Population heterogeneity leads to heterogeneity in the presentation of specific diseases. Modern medical studies on the efficacy of certain interventions have moved beyond simply exploring efficiency and tried to focus on individualized treatment. Acupuncture belongs to the system of Chinese Medicine. As early as 2,000 years ago, ancient Chinese realized that the adjustment of the composition of Chinese herbs and acupuncture points can improve the therapeutic effects of the same disease in different people. While subjective results can help indicate the efficacy of an intervention, objective results not only assist in explaining the efficacy of the intervention, but also provide evidence of the mechanism by which the intervention regulate. Therefore, whether the combination of subjective and objective outcome indicators can provide evidence of stratified efficacy and mechanism, especially the syndrome differentiation and treatment of acupuncture, is a superior strategy for further study of individualized treatment. In addition, combined with the theoretical characteristics of Chinese Medicine, the combination of modern stratified analysis and syndrome differentiation will make great contributions to the clinical promotion of acupuncture.

Biography

Dr. Li studied acupuncture at Zhejiang Chinese Medical University, China and graduated as MS in 2006. She then worked in Zhejiang Provincial People's Hospital from 2006 to 2021 and during this period she graduated as PhD at the same university as she received her MS. Now she is working in The Second Affiliated Hospital of Zhejiang Chinese Medical University, Xinhua Hospital of Zhejiang Province, China, as a deputy chief doctor of Chinese Medicine. She has published more than 20 papers in English (on SCI journals) or Chinese peer-reviewed original articles and is leading many scientific research projects.

Nadezhda Khrushcheva[1*], Kalgin K.V.[1], Savelov A.A.[1], Shtark M.B.[1]
Federal Research Center for Fundamental and Translational Medicine, Novosibirsk, Russia

Controlled reconstruction of cerebral networks: Possibilities of interactive brain therapy (stimulation) on the example of stroke (fMRI-EEG-Neurofeedback research)

Neuroplasticity is the main characteristic of the adaptive properties of the brain, which ensure the self-organization of complex hierarchical and multi-relational connections within and between neural networks, during normal growth and development and under pathological conditions. Targeted mental influence on the activity of specific cerebral structures and/or on the functional connections between them hypothetically contributes to clinical improvement in brain diseases, and feedback of the actual change in neural activity during task performance can optimize such training, making its effect on local neuroplasticity more pronounced. Simultaneous registration and processing of the signal of the brain activity electrical component (EEG) and the hemodynamic response to it (fMRI) provides a unique research tool - a bimodal neuroplatform for studying the spatial (fMRI) and temporal (EEG) components of brain activity. These parameters, being built into the feedback loop, create a new class of neurotechnologies - interactive therapy (stimulation) of certain brain formations and/or their interactions. The process of neuroplasticity becomes available for the cognitive reconstruction of neural networks with therapeutic, restorative, and research aims. Using the example of stroke, the report will consider the possibilities of the fMRI-EEG platform for neurofeedback (interactive therapy (stimulation) of the brain) in the study of transformations of the functional networks of the human brain and their clinical-network correlations.

Biography

Nadezhda Khrushcheva is a neurologist, head of the neurological department of the clinic of the Federal Research Center for Fundamental and Translational Medicine (Novosibirsk, Russia), senior researcher at the Laboratory of Clinical and Experimental Medicine, PhD. In the team of Mark Shtark, Academician of the Russian Academy of Sciences, she is engaged in the research of the brain functional anatomy and the possibilities of neurofeedback based on functional MRI and EEG (fMRI-EEG-neurofeedback) in modulating brain neuroplasticity processes.

Elena Illarionova
Smolensk State Medical University, Russian Ministry of Health, Smolensk, Russia

Vestibular Migraine

The current state of the problem of polymorphism of the clinical picture of vestibular migraine and objectification of the vestibular component remains unresolved and require further research.The purpose of our study was to study the clinical features and the possibility of objectification of vestibular dysfunction in patients with vestibular migraine. The study included patients with vestibular migraine and control groups. A targeted collection of complaints was carried out with an assessment of vestibular symptoms; a detailed history; a study of neurological status, with an emphasis on the study of oculomotor reactions, vestibular tests. Functional computer stabilometric tests were used to objectify vestibular dysfunction. Clinical features, as well as features of stabilometric parameters in patients with vestibular migraine have been identified, which can be used to obtain a quantitative assessment of vestibular dysfunction and objectification of vertigo in this category of patients. A purposeful and complete collection of complaints and anamnesis detailing the features of the symptoms in patients with vestibular migraine helps to identify the clinical polymorphism of the pathology in question and contributes to the correct diagnosis. And the use of functional stabilometric tests contributes to the objectification of vestibular dysfunction in this category of patients, quantifying the distinctive features of the balance system in vestibular migraine.

Audience Take Away Notes

- The great theoretical and practical relevance of this topic drove the present study to deepen our knowledge of the clinical features of vestibular migraine and develop additional instrumented investigation criteria for diagnosis of the vestibular component of this pathology
- Analyzing the results presented here indicates that the targeted collection of complaints and history in patients with vestibular migraine helps in making correct diagnosis, while the use of functional stabilometric tests as presented here allows the state of the posture system to be determined by quantifying vestibular dysfunction in patients with vestibular migraine; functional computerized stabilometry can be regarded as a possible method for objectively confi rming failure of the balance control system in patients with vestibular migraine. Identification of the features of loss of balance in patients with vestibular migraine may have important diagnostic, preventive, and therapeutic implications

Biography

Dr. Elena M. Illarionova – Candidate of Medical Sciences, associate professor at the Department of Neurology, Physiotherapy and Reflexology of the Faculty of Additional Professional Education Smolensk State Medical University. Graduated with honors from the Medical University in 2006. She studied for three years in residency in neurology, after that, three years in full-time graduate school in neurology, she defended her thesis in 2012. Currently working at a Medical University as an assistant professor, detailing on the issues of vertigo and vestibular migraine for more than 10 years. She actively published in leading scientific journals and actively publishes in leading scientific journals and speaks at conferences and congresses of various levels.

Malka Cohen Armon

Tel-Aviv University, the Sackler School of Medicine and Sagol School of Neuroscience, Israel

Long-term memory acquisition and loss are dependent on PARP1-Erk2 synergism

We found a small piece in the puzzle of long-term memory. This finding identified the odd phenomenon implicating PARP1, a protein that initiates the repair of DNA breaks, in synaptic potentiation and in long-term memory formation during learning. The identified molecular mechanism underlying this phenomenon disclosed the link between the stimulation of receptors in the membrane and the expression of immediate early genes that are implicated in synaptic plasticity. This link is based on a synergism between PARP1 and Erk2 activation, causing localized chromatin remodeling which is required for histone modifications and activation of Erk targeted transcription factors of immediate early genes that are implicated in synaptic plasticity. Long-term memory formation in Aplysia and rodents was dependent on PARP1-Erk2 synergism including learning, pleasant and fearful memories.

Amir Hadanny
Chief Researcher, Aviv Clinics, The Villages, FL, USA

Treating chronic-stage stroke with hyperbaric oxygen therapy

Due to damage brought on by cerebrovascular occlusion or hemorrhage, the brain struggles to obtain the same amount of oxygen to the afflicted regions of a stroke. A novel protocol utilizing hyperbaric oxygen therapy (HBOT), has been proven to improve neurocognitive and neurological functions in post-stroke patients and promote neuroplasticity in recent studies. During this presentation, Dr. Amir Hadanny will present his most recent study and HBOT's impact on cognitive function in post-stroke patients.

A retrospective analysis was conducted on patients who received HBOT for chronic stroke (>3 months) between 2008 and 2018. The protocol was performed in a multiplace hyperbaric chamber, where participants were subject to the 60 daily sessions in 12 weeks, 5 sessions a week for 90 minutes.

The study included 162 patients: 77 (47.53%) had cortical strokes, 87 (53.7%) strokes were located in the left hemisphere and 121 suffered ischemic strokes (74.6%). HBOT induced a significant increase in all the cognitive function domains ($p < 0.05$), with 86% of the stroke victims achieving a clinically significant improvement. In all cognitive domains, the baseline cognitive function was a significant predictor of improvement ($p < 0.05$), while stroke type, location and side were not significant predictors. Results continue to show that the unique HBOT protocol has made significant improvements in stroke recovery outcomes, even many years after the incident. The clinical improvements were achieved regardless of the type of stroke or where it occurred in the brain.

Audience Take Away Notes

- Through the presentation of this research, the audience will learn about a unique and successful method that has been proven to reverse cognitive and physical damage that has occurred as a result of a stroke
- The research presented will provide hope for stroke survivors who suffer from post-stroke symptoms and impairments that were once thought to be chronic
- The audience will receive an in-depth analysis of what happens to the brain during a stroke and how the unique HBOT protocol can improve neurocognitive function in post-stroke patients and promote neuroplasticity

Biography

Dr. Hadanny is the Chief Researcher and Head of Global Clinical Operations at Aviv Clinics. He is a certified neuro-surgeon, hyperbaric physician, and Chief Medical Research Officer at the Sagol Center. For the past decade, he has worked alongside Dr. Shai Efrati researching neurorehabilitation, neuroplasticity and physiology, publishing more than 45 papers on the effects of HBOT on cognitive and physical performance. Before joining the Sagol Center, Dr. Hadanny was Chief Resident in the Galil Medical Center neurosurgery department. He earned his MD from Tel Aviv University and his Ph.D. in Bioinformatics and Machine Learning from Bar Ilan University.

Dr. Rocco J. Gennaro
Department of Political Science and Philosophy, University of Southern Indiana, Evansville, Indiana 47712, United States of America

The neuroscience of mental disorders and the nature of consciousness

In this talk I first review and discuss empirical evidence regarding brain damage and neural abnormalities associated with some psychopathologies and cognitive deficits, such as hemispatial neglect, agnosia, schizophrenia, amnesia, somatoparaphrenia, akinetopsia, Capgras syndrome, simultanagnosia, alexithymia, out-of-body and near-death experiences, and others. It becomes clear just how closely normal conscious mental functioning depends upon normal brain functioning as well as how some very specific mental changes occur when, and only when, very specific brain damage occurs. As a philosopher, I then also explore the implications of these results with respect to the nature of the so-called mind-body problem, consciousness, and the problem of personal identity. In particular, I examine the plausibility of materialism, roughly the view that mental processes are brain processes, in light of the evidence discussed and in contrast to a dualist conception of the mind (whereby mental states are not physical in some sense). For example, how should we interpret the empirical results in terms of the mind-brain connection, e.g. as a correlation, cause, or as an identity relation? I then also briefly examine the prospects for a conscious afterlife based both on the brain evidence adduced and the other considerations discussed. For example, even if conscious mentality merely depends upon proper neural function, does it then stand to reason that all of one's conscious mental activity ceases when all neural functioning ceases?

Audience Take Away Notes

- The audience will better understand how specific brain damage affects various specific mental abilities in often rather strange ways
- Attendees can be helped in terms of their teaching and research by becoming more aware of how neural abnormalities bear on the nature of consciousness and personal identity
- Examining brain damage and its effects can also aid research in the search for the so-called neural correlates of consciousness and in the attempt to solve the binding problem
- It can also help one to think about the philosophical implications of some brain research, e.g. to what extent conscious mental activity can continue after brain injury or damage (or even after brain death)

Biography

Dr. Rocco J. Gennaro is Professor of Philosophy at the University of Southern Indiana. His primary research and teaching interests are in Philosophy of Mind/Cognitive Science (especially consciousness), Metaphysics, Neuroethics, and Early Modern History of Philosophy. Dr. Gennaro has published twelve books (as either sole author or editor) and over sixty articles and book chapters in these areas. He has most recently published Consciousness (Routledge Press, 2017), Mind and Brain: A Dialogue on the Mind-Body Problem, 2nd edition (Hackett, 2020) as well as edited The Routledge Handbook of Consciousness (2018). He has also published The Consciousness Paradox: Consciousness, Concepts, and Higher-Order Thoughts (MIT Press, 2012), and edited an anthology entitled Disturbed Consciousness: New Essays on Psychopathologies and Theories of Consciousness (MIT Press, 2015).

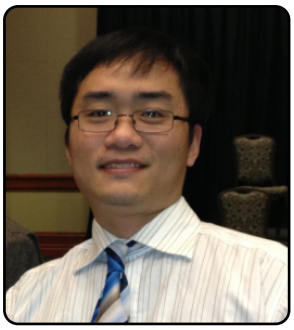
Brandon Lucke-Wold MD, PhD, MCTS
Department of Neurosurgery University of Florida Brandon Lucke

Arteriovenous malformations: An update on models and therapeutic targets

Arteriovenous malformations (AVMs) are an anomaly of the vascular system where feeding arteries are directly connected to the venous drainage network. While AVMs can arise anywhere in the body and have been described in most tissues, brain AVMs are of significant concern because of the risk of hemorrhage which carries significant morbidity and mortality. The prevalence of AVM□s and the mechanisms underlying their formation are not well understood. For this reason, patients who undergo treatment for symptomatic AVM□s remain at increased risk of subsequent bleeds and adverse outcomes. The cerebrovascular network is delicate and novel animal models continue to provide insight into its dynamics in the context of AVM□s. As the molecular players in the formation of familial and sporadic AVM□s are better understood, novel therapeutic approaches have been developed to mitigate their associated risks. Here we discuss the current literature surrounding AVM□s including the development of models and therapeutic targets which are currently being investigated.

Biography

Brandon Lucke-Wold was born and raised in Colorado Springs, CO. He graduated magna cum laude with a BS in Neuroscience and distinction in honors from Baylor University. He completed his MD/PhD, Master's in Clinical and Translational Research, and the Global Health Track at West Virginia University School of Medicine. His research focus was on traumatic brain injury, neurosurgical simulation, and stroke. At West Virginia University, he also served as a health coach for the Diabetes Prevention and Management program in Morgantown and Charleston, WV, which significantly improved health outcomes for participants. In addition to his research and public health projects, he is a co-founder of the biotechnology company Wright-Wold Scientific, the pharmaceutical company CTE cure, and was a science advocate on Capitol Hill through the Washington Fellow's program. He has also served as president of the WVU chapters for the American Association of Pharmaceutical Scientists, Neurosurgery Interest group, and Erlenmeyer Initiative Entrepreneur group. In addition, he has served as vice president for the graduate student neuroscience interest group, Nu Rho Psi Honor Society, and medical students for global health. He was an active member of the Gold Humanism Honor Society and Alpha Omega Alpha Honor Society. He is currently a member of the Young Neurosurgeons' Committee. He is married to Noelle Lucke-Wold, and has a toddler daughter named Esme. As a family, they enjoy running with their dogs, rock climbing, and traveling the world. In his spare time, Brandon frequently runs half marathons and 10ks together with his wife. Brandon also enjoys reading and discussing philosophy and playing chess. He is excited to join the neurosurgery residency program at University of Florida.



Wei Song
National Institutes of Health, United States

Predicting noncoding disease causal mutations in central nervous system through Deep Learning

The majority of GWAS mutations are in noncoding regions, making it challenging to pinpoint the true disease-causal mutations in a complex linkage disequilibrium (LD) block and quantify their causality. To address this problem, we developed a deep learning (DL) algorithm that accurately predicts cell type-specific enhancers and transcription factor binding sites (TFBSs) from raw DNA sequences and used it to guide the subsequent identification of phenotype-causal enhancer mutations. In a pilot study of 10 GWAS SNPs linked to brain diseases, whose causality had been previously validated experimentally, we precisely identified 8 out of 10 SNPs as causative within their corresponding LD blocks. Among 27,488 GWAS LD SNPs associated with schizophrenia, autism, and several other central nervous system (CNS) diseases, 3% reside in predicted TF binding sites active in the fetal brain, and out of these 3%, 11 SNPs are predicted as candidate causal. These results suggest that our algorithm can be applied to resolve LD blocks of other disease-associated SNPs, for which no experimental profiling has been done yet, and accurately identify disease-causative SNPs, as well as putatively affected regulatory mechanisms and pathways.

Biography

Dr. Song received his Ph.D. degree of Bioinformatics from University of North Carolina at Charlotte. After that he pursued his postdoc research on protein structure prediction and demographic structures of human populations in the new world. Currently Dr. Song is a staff scientist in National Center for Biotechnology Information, NIH. His research interests focus on the heterogeneity of tissue-specific enhancers in human genome, including identification of the primary enhancers and predicting disease causal variants using deep learning, hierarchical structure in a multi-element regulatory program and 3D chromatin contacts of the enhancer-gene networks.

Geetanjali Rathore

Associate Professor, Department of Neurosciences and Paediatrics, University of Nebraska Medical Centre, Omaha, Nebraska, United States

The role of newborn screening in the era of gene therapy for neuromuscular disorders

Neuromuscular disorders (NMD) are debilitating conditions that often lead to early morbidity and mortality due to progressive weakness. In the last decade, the treatment of NMD has undergone a remarkable advancement and we now have several therapeutic options for these patients which can be life-saving options.

This has made newborn screening more imperative then ever given that early treatment leads to best neurological outcomes, while delays can lead to irreversible loss of function. The key to early initiation of treatment is accurate early diagnosis. Newborn screening (NBS) provides an opportunity to identify children at higher risk for these debilitating diseases, so that diagnosis can be confirmed followed by prompt initiation of therapies. Herein, is a review of the common NMD and the role of NBS in changing their landscape.

Audience Take Away Notes

- Audience will be able to get an update on Newborn screening and early diagnosis of neuromuscular disorders
- Audience will learn about new therapies including gene and enzyme modulation therapies for these disorders
- Audience will get to review evidence of how newborn screening in conjunction with advanced therapies can improve outcomes in these potentially life-threatening disorders
- If not available in their region, this may provide them with information and an incentive to advocate for early diagnosis and treatment of NMD in newborns

Biography

Dr. Geetanjali Rathore is an Associate Professor of Neurosciences and Pediatrics at UNMC, Omaha, Nebraska, USA. Dr. Rathore is the Medical Director of the Pediatric Neuromuscular Clinic at Children's Hospital, Omaha and also a board member of the Newborns Screening Advisory Committee for the Nebraska Department of Health. Dr. Rathore has published many papers, book chapters and podcasts. She has also been invited to many international talks and scientific meeting panels. Dr. Rathore is currently the Associate Editor for the Journal of Child Neurology and on the editorial board of several International Journals.

Mohit Saxena
Department of Radiology, Northwestern Medicine, Chicago, Illinois, USA

Recent advances in brain imaging

The talk will elucidate how certain imaging techniques that are currently used in research can be applied to clinical diagnoses and help the surgeons, neurologists and epileptologists make better decisions.

Recent Advances in functional MRI, DTI tractography and Spectroscopy can be used in diagnostic support towards better patient management. This helps in getting more details to the size and vascular supply and classification of different tumor types

3D post-processing of MRI, PET and CT, are a recent demand in the medical industry.

The DBS electrode localization and optimal stimulation can help the Neurologists better manage the DBS implanted patients. It has been observed that DBS implanted patients develop speech dysarthria, which results from the stimulation of undesired areas. Using fMRI and tractography, the exact stimulation volume can be determined and a more accurate DBS implant and its stimulation parameters can be achieved. This can be done prior to lead placement and so helps the surgeons to place the electrodes more accurately.

Biography

Dr. Saxena completed Ph.D. in Neurology from All India Institute of Medical Sciences, (AIIMS) New Delhi, India. His research on Parkinsonism was aimed towards understanding of the basic principles of MRI physics and how those MR image acquisition parameters can be modified towards better diagnostic imaging. Currently, as an Advanced Imaging Neuroscientist at Northwestern Medicine Hospital, Chicago Dr. Saxena is helps Neurosurgeons, Eppileptologists and Neurologists in not only viewing multimodal medical images i.e., MRI, CT, PET, SPECT, MEG, EEG etc. but also preserving the functional brain areas.

Lloyd L Tran
Biomed Industries, Inc., USA

Neurogenesis hypothesis with a case study- phase 2A clinical trials of NA-831 for the treatment of Alzheimer's disease

In the hippocampus, new neurons are generated throughout life via a process called adult hippocampal neurogenesis (AHN). In mild cognitive impairment (MCI) and mild to moderate AD (early AD), AHN is reduced suggesting that AHN impairment compromises hippocampal function. Accordingly, augmenting AHN could help prevent or slow cognitive decline in MCI and early AD.

NA-831 is a small drug molecule, which activates synaptic AMPA receptors, and increases the expression of BDNF (brain derived neurotrophic factor). BDNF is crucial in synaptic plasticity, learning and memory formation in the hippocampus. NA-831 restores neurogenesis by increasing the number of DCX+PCNA+ neuroblast cells.

A randomized clinical trial of NA-831 was conducted in 32 patients with MCI, who received 10 mg of NA-831 or placebo orally per day; and 24 patients with mild and moderate AD, who received 30 mg of NA-831 or placebo orally per day for 24 weeks.

RESULTS: NA-831 provided a significant delay in cognitive decline in MCI as measured by ADAS-Cog-13, an average score difference of 3.4 compared to placebo (p = 0.01; ITT) after 24 weeks of treatment.

Similarly, NA-831 delayed cognitive decline in early AD, an average score difference of 4.1 com-pared to placebo (p = 0.001; ITT). CIBIC-Plus showed 78 % of the study participants receiving NA-831 improved (p = 0.01; ITT).

NA-831 was well-tolerated at 30 mg/day for 24 weeks, and no serious adverse events were observed.

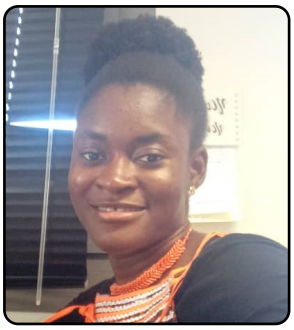
CONCLUSION: The Phase 2A results of NA-831 support the viability of Neurogenesis Hypothesis as an alternative approach to the Amyloid Hypothesis. Details of these Phase 2A clinical trials and the Neurogenesis Hypothesis will be presented and discussed.

Audience Take Away Notes

- Introduce the Neurogenesis Hypothesis as an alternative approach to the Amyloid Hypothesis
- Show the Phase 2A clinical results of a new drug NA-831 for the treatment of Alzheimer’s disease
- The Phase 2A of NA-831 supports the viability of the Neurogenesis Hypothesis
- The presentation can inspire researchers to look into Neurogenesis Hypothesis that can possibly replace the Amyloid Hypothesis as the guidance for drug development.

Biography

Lloyd is a scientist with 25-year experience in drug development and clinical trials management. He is an inventor with a number of patents in drug therapeutics in the treatment of neurological and infectious diseases.
Lloyd serves as the chairman and Chief Scientific Officer of Biomed Industries, Inc. that is conducting phase 2/3 of NA-831 for the prevention and treatment of Alzheimer’s Disease. In his early career, he was employed as a research scientist at G.D. Searle, (a subsidiary of Pfizer), and was the director of R&D at Biomed Pharmaceuticals.
Lloyd graduated with a BSc (Honours) and completed a PhD in medicinal chemistry at University of Otago and Wellington University of New Zealand.



Okechukwu N.G*, Klein C., Meyer L., Patte-Mensah C., Maitre M., Mensah–Nyagan A.G

Biopathologie de la Myéline, Neuroprotection et Stratégies Thérapeutiques, INSERM U1119, Université de Strasbourg

Evidence of the protective effect of allopregnanolone analogues in alzheimer's disease experimental models

Alzheimer's Disease (AD) is a major cause of cognitive/memory impairment and dementia. Amyloid beta (Aβ) is an extracellular 39-43 residue long peptide whose aggregation is associated with Alzheimer's Disease (AD) and it's accumulation represents one of the key neuropathological features of AD with the other been hyperphosphorylation of Tau protein which aggregates to form Neurofibrillary Tangles (NFT). The shift in the Aβ cascade hypothesis from all Aβ to small soluble oligomeric intermediates is directing the search for therapeutics towards the toxic mediators of the disease. Monomeric form of Aβ have been isolated from the brain of AD patients and have in facts been shown to be devoid of neurotoxicity but rather have been suggested to be neuroprotective. The transition of monomers to oligomers serves both as the first event of aggregation and the key event determining the transformation of benign protein to neurotoxic one. Since the pathophysiological mechanisms of AD begin several years before the onset of Mild Cognitive Impairment (MCI) or clinical symptoms, targeting the most toxic oligomers may prove to be an effective treatment by preventing their spread, however for any significant progress to be made in AD therapy, there is urgent need to identify therapeutic agents capable of delaying the onset or slowing down the progression of AD by either destabilizing the oligomeric Aβ forms, preventing the self-assembly of the monomeric Aβ into oligomers and ultimately protecting against the toxic effect of these small soluble Aβ forms. The 5XFAD mice model shows relatively early and aggressive presentation of AD-related phenotype with high intraneuronal Aβ 42 at 1.5 months, memory decline at 1 month and extracellular amyloid deposition at 2 months served as useful tools employed to study the early myelin alteration in AD, how these alterations influence learning and memory and the efficacy of a potential novel therapy in protecting against these early changes. Several methods where combined to evaluate the BR351 (a novel analogue of allopregnanolone) effect on the learning deficit, memory deficit and the capacity of BR351 to protect against Aβ. Single daily dose (10mg/kg) of BR351 prevented memory and learning deficit assessed with Morris water maze, and counteracted the AD induced demylination by preserving Myelin Basic Protein (MBP) and Proteolipid protein expression in the regions of the hippocampus and corpus callosum. Furthermore, BR351 treatment also prevented AD-evoked decreased neurofilament-200 expression in the regions of the hippocampus and corpus callosum. Thus, these novel analogs may be useful for translational investigations aiming to tackle the early stage of Alzheimer's.

Audience Take Away Notes

- This research portrays the therapeutic role of neurosteriods neurodegeneration. Furthermore, it provides pratical and novel analogs that may be useful for translational investigations aiming to tackle the early stage of Alzheimer's. Additionally, it proposes possible presymptomatic marker for Alzheimer's disease diagnosis.

Biography

Mrs. Okechukwu studied Anatomy at Ebonyi State University, Abakalili and graduated in 1999with a Master's degree, before joining the research group of Prof. Mensah–Nyagan at the INSERM U1119, Université de Strasbourg, CRBS, France as a doctorate student. She is currently in her 3rd year of her doctoral studies.

magnus Group

16-17 JUNE

DAY 02

VIRTUAL ROOM 01

POSTERS

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

Zahra Khan[1*], Avinash N. Mukkala[1], Mirjana Jerkic[1], Fawad Ashraty[1], Haejin Kwak[2], Emma Noble[1], Michael Gryciuk[1], Sandy Trpcic[1], Menachem Ailenberg[1], Ana Andreazza[2], Andrew Beckett[1], Shawn G. Rhind[3], Ori D. Rotstein[1]

[1]Department of Surgery, the Keenan Research Centre for Biomedical Science, Unity Health Toronto - St. Michael's Hospital and University of Toronto, Toronto, Ontario, Canada

[2]The Departments of Pharmacology & Toxicology and Psychiatry, University of Toronto, Toronto, Ontario, Canada

[3]Defence Research and Development Canada, Toronto Research Centre, Toronto, Ontario, Canada

Remote ischemic conditioning as an adjunct intervention in ICU traumatic brain injury patients: Preliminary findings on injury biomarkers from a randomized controlled trial

Introduction: Management of Traumatic Brain Injury (TBI) is currently aimed at secondary brain injury prevention that results from brain ischemia. Remote ischemic conditioning (RIC) is a non-invasive intervention that has been shown to lessen ischemia/reperfusion injury and potentially reduce tissue/organ injury damage. RIC has also been shown to have neuroprotective effects via anti-inflammatory action, stabilization of mitochondria, and increased cerebral blood flow in experimental animal models. In a prior nonrandomized trial, TBI patients undergoing RIC showed amelioration of neuronal injury specific markers, neuron-specific enolase and S-100 calcium-binding protein B (S100B), compared to an untreated control group. We performed a randomized double-blinded control trial in TBI patients to test the hypothesis that RIC would exert beneficial effects on secondary injury as reflected by blood-based neurological injury biomarkers.

Methods: A randomized double-blinded controlled clinical trial with two arms (sham and RIC) is in progress in TBI ICU patients at St. Michael's Hospital, Toronto, Canada. Thirteen plasma biomarkers measured at 0h (pre-intervention) and 4-6, 24, 48 and 72h (post-intervention) are primary outcome measures. Eligibility criteria include presence of intracranial hematoma on CT scan, Glasgow Coma Scale score ≤ 12. RIC involves 4 cycles of 5-min occlusion at 30mmHg>systolic blood pressure, followed by 5-min of deflation on an arm (using pneumatic torniquet) within 48h of injury. Secondary outcome measures will include clinical data and neurocognitive assessments. Currently, the trial has enrolled 43 out of 45 patients. Preliminary plasma biomarker measurements (pg/mL or copies/uL), performed by the immunoassay, commercial kits, or qPCR were conducted on 13/RIC and 15/Sham patients and levels were adjusted to baseline (0h).

Results: Five of the 13 biomarkers yielded significant and marginally significant differences between Sham and RIC groups at specific time points: RIC significantly lowered levels of neural injury marker S100B (-399 [95% CI: 64 to 734] pg/mL; ANCOVA P = 0.02164) and repair myokine Irisin (-1550 [95% CI: 740 to 2361] pg/mL; ANCOVA P = 0.00058) at 4-6h, and mitochondrial dysfunction marker lactate (-1039 [95% CI: 21 to 2058] pg/mL; ANCOVA P = 0.046) at 72h. RIC significantly increased mitochondrial dysfunction marker ccf-mtDNA (12111 [95% CI: -23889 to -333] pg/mL; ANCOVA P = 0.044) at 24h while levels of inflammatory marker HMGB1 (-26569 [95% CI: -2887 to 56025] pg/mL; ANCOVA P = 0.074) were decreased with marginal significance (P ≤ 0.1) at 48h.

Conclusions: We report that RIC demonstrates promising preliminary effects in limiting post-injury elevations in S100B, Irisin, and lactate levels observed in TBI patients. Completed results of the ongoing trial will further elucidate the mechanisms and outcome of RIC treatment as a potential novel adjunctive therapy for moderate-to-severe TBI.

Audience Take Away Notes

- They will become more aware of current research in the field of TBI
- List all other benefits
- It will help individuals gain awareness of current research in the field of TBI
- Individuals will learn about a new therapeutic intervention
- Researchers can gain an understanding of our study protocol and implement it or enhance their own
- It provides new information in assessing the issue of secondary brain injury management

Biography

Zahra Khan completed her Bachelor of Science in Human Biology and Mental Health from the University of Toronto. She is currently pursuing her Master of Science in Neuroscience at the same university, where she is researching a new intervention for patients with traumatic brain injury in the intensive care unit. Her interests lie in neuroscience and mental health research, particularly in clinical settings. With her strong academic background and passion for the field, Zahra hopes to make a significant contribution to the field of neuroscience in the coming years.

Comas Guerrero Betiana[1*], Francisco Astorino[2]

[1]Department of Pediatric Neurology, Hospital de La Baxada Dr. Teresa Ratto, Parana, Entre Rios, Argentina

[2]Department of Pediatric Neurology, Hospital Orlando Alassia, Santa Fe

Pediatric febrile status epilepticus. Case series of children admitted at Hospital de Niños Dr. O. Alassia - Santa Fe, Argentina, period 2015 until 2018

Introduction: The status epilepticus is a neurological emergency that needs intensive measures and also has high mortality. The International Leage against Epilepsy (ILAE) in 2015 sets a new

Definition: Is a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms, which lead to abnormally, prolonged seizures (after time point t1). It is a condition, which can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures. Fever the most frequent etiology in the pediatric age

Principal Aim: To describe epidemiological aspects of febril status epilepticus suffered by children admitted from 01.01.2015 to 14.04.2018.

Secondary Aims: To stablish etiology and predisposing factors, to analyze electrencephalographical patterns and to revise treatment protocols and antiepileptic drugs schemes used

Material and Method: 53 children admitted into Hospital de Ninos Dr. O. Alassia with SE febril diagnosis. Period of time: from 01.01.2015 to 14.04.2018. Analysis of clinical histories and complementary studies, patient evaluation, and review of different treatments.

Results: 53 patients, 31 males and 22 women, medium age 1, 65 years. Electroencephalograms were practiced to 36 patients, 6 with paroxysmal focal activity and 2 with generalized activity. 34 patients fulfilled neuro-images, 8 were pathological. Clinical presentation as first event 41 patients, 7 of them had pathological precedents. Etiology of feverish area: 5 encephalitis, 6 pneumonias, 4 gastroenteritis, 1 sepsis, 1 postvaccinal, 36 upper-airway infection respiratory. Treatment: 50 needed treatment of the first line with benzodiazepines, 11 of them had pre hospital treatment, only 4 needed anesthetics.

Conclusions: Median of age of presentation 1, 65 years. Etiology most frequent was respiratory. 36 electroencephalograms were practiced, only 8 of them were pathological. Only 11 received prehospital treatment, only 4 needed drugs anesthetics. 41 patients had clinical presentation as first event, 7 of them had pathological precedents.

Audience Take Away Notes

- The audience will learn about the realities of other countries
- This research will help the audience to design prehospital treatment guides
- This research shows the importance of teaching the status treatment guide in medical emergencies

Biography

Dr. Betiana Comas is from Argentina. She studied Medicine at Rosario National University and graduated in 2001. She completed her pediatric residency at San Roque Hospital and her pediatric neurology residency at Alassia Hospital. After a one-year fellowship in epilepsy at Italiano Hospital, she graduated from epileptology in 2012. She worked as an Associate Doctor in the Epilepsy Department at Italiano Hospital until 2016, when she became chief of the Neurology Department at Hospital de La Baxada Teresa Ratto.

Cristian Ravariu[1, 2*], Catalin Parvulescu[3], Alina Popescu[3]

[1]Department of Electronic Devices Circuits and Architectures, Polytechnic University of Bucharest, Splaiul Independent 313, 060042, Bucharest, Romania
[2]SME EduSciArt SRL, Bucharest, Romania
[3]Clean Room Division, National Institute for Research and Development in Micro technologies 077190, Voluntary, Ilfov

Biosensor with acetyl-choline-esterase enzymatic receptor and si-nanoporous layer as entrapping element - Extrapolated to para-sympathomimetic pesticides

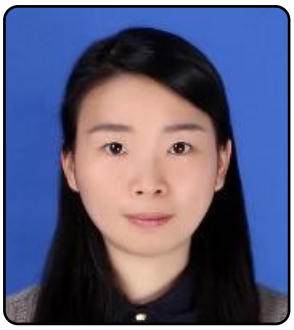
The Acetyl-cholinesterase enzyme, also noted AcHE, has the code - EC 3.1.1.7, being an enzyme that degrades by hydrolysis the acetylcholine (AcH) neurotransmitter in choline and acetate. In vivo, this enzyme is found at neuromuscular junction or cholinergic synapses from the central nervous system. The AcHE has a high catalytic activity — each molecule of AcHE can degrade about 25000 molecules of acetylcholine per second, [1]. This operation principle was extrapolated to the para-sympathomimetic pesticides. They work through the inhibition of AcHE, permitting acetylcholine to transfer nerve impulses indefinitely and causing a variety of symptoms such as weakness or paralysis. They operate by disrupting the sodium/potassium balance of the nerve fiber, forcing to a continuously transmission through the nerve. Para Oxon is a novel generation of para-sympathomimetic pesticide, acting as an inhibitor for AcHE. Chemicals from this group can act on two directions: (i) directly - stimulating the nicotinic or muscarinic receptors or (ii) indirectly - by the cholinesterase inhibition and supporting the acetylcholine releasing. In this way, Para Oxon prevents the acetylcholine breaking, favoring the time action of this neurotransmitter, so that the entire control of the parasympathetic nervous system can be transferred against the pests, [2]. Using some monitoring tools, like electronic biosensors, the pesticides can be put under control. Microelectronics offers some convenient transducers. Borrowing micro-technological processes, the enzymatic biosensors can be integrated onto the Silicon wafers. This work depicts in details the key-technological processes from a Clean Microelectronic Room to produce a Para Oxon biosensor. The key processes must be compatible both with the enzymatic membrane deposition and the Si-technology. In this way, a high series production of biosensors at lower cost is possible. A good entrapping occurs if the enzyme is captured on a nano-porous thin layer by adsorption combined with other cross-link methods. A main advantage of the Si-nano-porous layer directly grown on a wafer consists in strong anchoring to the Si-substrate. Converting p-type Si in Si-porous by anodization is a solution. Then some technological steps, with tests and microscopy analysis are presented. Finally, the preliminary tests of the developed biosensor with the AcHE enzyme immobilized onto the Si-nano-porous layer are discussed.

Audience Take Away Notes

- How a biosensor of pesticides using AcHE enzyme and Si-substrate can be co-integrated
- Si nano-porous structure action is presented
- The presented technique can be used to expand the research field of neurotransmitters biosensors
- The issue provides a practical solution to co-integrate biomaterials near electronic devices
- The presented solutions could improve the accuracy of a biosensor design

Biography

Prof. C. Ravariu graduated in 1993 in Microelectronics at Polytechnic University of Bucharest, Romania. He worked as scientific researcher at Institute of Micro technology, Bucharest, then joined the Polytechnic University of Bucharest, where he became Full Professor, 2013. After participation in foreign stages in Bioelectronics (Patras, Greece), Nano-devices (EPFL, Switzerland), Organic Electronics (LAAS, France), he received Postdoc degree in Bio-Nano-Electronics, 2012. He published more than 250 research articles. Head/participant in Bioengineering projects, C. Ravariu deals with BioFET transistors for customized bio analyses, Technology for functional characterization of the beta-pancreatic mass, differential diagnosis of meningitis by determining the cytokine profile by fast devices.



Qiao-yun Ren[1*], An Liu[1], Susu Wang[1], Wei Xie[1], Zheng-ping Jia[2]

[1]The Key Laboratory of Developmental Genes and Human Disease, Ministry of Education, School of Life Science and Technology, Southeast University, Nanjing, China

[2]Neurosciences & Mental Health, The Hospital for Sick Children, Toronto, Ontario, Canada

C-terminal domains of GluA2 regulate amyloid-beta induced synaptic and cognitive impairments

Objective: Synaptic dysfunction is one of the early symptoms of AD, which occurs much earlier than the formation of amyloid plaques and neurofibrillary tangles. Synaptic function is closely related to the number and synaptic localization of AMPA receptors (AMPARs), and this process is directly regulated by the interaction of AMPAR carboxy-terminal domains (CTDs) and their intracellular proteins. It has been found that changes in AMPAR number and synaptic localization occur in the early stages of AD, but it is still unknown whether AMPAR-CTDs are directly involved in regulating synaptic dysfunction in early AD and whether interfering with the interaction of AMPARs and intracellular proteins can rescue synaptic and cognitive dysfunction.

Methods: In this study, we applied A2C1KI mice, which have the intracellular domain of GluA2 replaced with that of GluA1 (altering the interaction between AMPAR and intracellular proteins), and treated them with amyloid-beta oligomers (Aβo), or crossed them with AD4T transgenic mice that overproduced Aβ to generate double hybrid mice. We also designed differently interfering peptides and viruses to find the key sites of CTDs further and intervene in the interaction of AMPARs with intracellular proteins. Electrophysiological and behavior tests were used to explore the synaptic function and cognitive function of different treatment groups.

Results: Aβo did not impair synaptic function and cognitive impairment in A2C1KI mice, and when we further interfered with the interaction between GluA2 and different intracellular proteins in WT mice, the toxic effects of Aα oligomers on the central nervous system could be blocked. In AD4T mice with A2C1KI or interference peptide, synaptic function, and cognitive performance were greatly improved.

Conclusion: This study reveals that the GluA2-CTDs and its intracellular proteins play an important role in early synaptic damage and cognitive impairments in AD and interfering with its interaction can significantly rescue AD-like performance at different levels.

Audience Take Away Notes

- This research presents kinds of valuable methods to model early Alzheimer's disease with synaptic failure
- This research originally explains the role and mechanism of specific domains of key synaptic molecules in the pathogenesis of Alzheimer's disease
- This research introduces an effective intervention in the prevention and treatment of early Alzheimer's disease

Biography

Dr. Ren studied Biological Science at Anhui Normal University and graduated in 2016. After that, she has been studying Neuroscience at the Key Laboratory of Developmental Genes and Human Disease, Ministry of Education, School of Life Science and Technology, Southeast University until now. She participated in the National Natural Science Foundation Youth Project and has published 2 research articles in SCI (E) journals.

Dalya Sezercan*, Nida Yildiz
Hisar School, Istanbul, Turkey

Neuroscience of addiction

Addiction can be defined as an irresistible desire for an object, individual or entity although it is known to be harmful to the person physically or psychologically. In addiction physical dependence is explained as changes in the brain structure. In addition to causing addiction in a person, the substances used also cause differences in the brain's reward system, decision-making and learning mechanisms (1). The positive reinforcement feature's most important indicator is that the person starts to search for the substance he/she uses so that person starts to crave the substance. The reward system plays a big role in becoming addicted. The reward system gets affected by addiction because addiction causing substances increase the dopamine release in the ventral striatum. Mesocorticolimbic system has an important role in the reward system because it contains the dopaminergic pathways. The mesolimbic pathway, the mesocortical pathway, the nigrostriatal pathway, and the tuberoinfundibular pathway are the four dopaminergic pathways. Dopamine is transported from the VTA (ventral tegmental area) to the nucleus accumbens and amygdala via the mesolimbic pathway. Depression has been linked to dysfunction in this system. The ventral tegmentum is linked to the prefrontal cortex via the mesocortical pathway. It is strongly linked to the frontal lobes. It is thought to be involved in a variety of cognitive processes, including motivation, emotion, and executive functions. The primary role of the nigrostriatal pathway is to affect voluntary movement, feeding behavior, and movement via basal ganglia motor loops. The nigrostriatal dopaminergic pathway influences cognition, reward, and addiction. Parkinson's disease is caused by a deficiency in this. Finally, the tuberoinfundibular pathway, which is one of the major neuronal networks in the brain that uses dopamine as its primary neurotransmitter. Dopamine release in this route modulates pituitary gland prolactin output.

Keywords: Reward system, Dopaminergic pathways, Addiction, Mesocorticolimbic system.

Audience Take Away Notes

- Addiction and its effects on brain functions
- The process of addiction
- The dopaminergic pathways and their functions
- The reward system

Biography

Dalya Sezercan was born in 2006 in Istanbul, Turkey. She will graduate from Hisar School in 2024. She was interested in neuroscience of addiction after she observed how substance use in her country affected teenagers and how teenagers were being controled by these addictive substances. She wondered why people were becoming addicted this easily even though they knew these substances were bad for them. She then decided to research what brain functions go through while someone is becoming addicted.

Mona Ramezani
Semnan University of Medical Sciences, Semnan, Iran

The effect of pelvic floor muscle training alone or in combination with multi-session anodal transcranial direct current stimulation in the treatment of sexual dysfunction in female patients with multiple sclerosis

Introduction: Sexual dysfunction (SD) following dysfunction of the pelvic floor muscles (PFM) is perceived to be more common in women with multiple sclerosis (MS) than in the general population. Studies report a prevalence of up to 80% in women. SD is a painful but still underreported and underdiagnosed complication of MS. It seems that concurrent M1 anodal transcranial direct current stimulation (a-tDCS) may prime the effects of pelvic floor muscle training (PFMT) in patients with MS. This study aimed to investigate the effects of M1 a-tDCS on the effectiveness of PFMT in treating women with MS suffering sexual dysfunction.

Methods: Thirty 18–45 years old females with MS (EDSS less than 6.5) were randomly divided into two groups of concurrent multiple session active M1 a-tDCS and PFMT (intervention) and concurrent multiple session sham M1 a-tDCS and PFMT (control) in the randomized double-blinded control trial study. These patients received 20-minute interventions three times a week for eight weeks. Sexual dysfunction was measured by Female Sexual Function Index (FSFI) pre and post-intervention.

Results: The results in the intervention group showed that the pain after intervention was significantly decreased as compared to before intervention ($P < 0.05$). In addition, the rate of satisfaction and total score of sexual function was significantly increased after active M1 a-tDCS and PFMT in the intervention group ($P < 0.05$). Moreover, the rate of satisfaction and total score of sexual function were significantly improved after the intervention in intervention as compared to control group ($P < 0.05$).

Conclusions: This study demonstrates the efficacy of both tDCS and PFMT for improving sexual function in female MS patients, but did not show superiority of any particular method. Further studies are required to investigate the differences between these two methods.

Keywords: Multiple sclerosis, Transcranial direct current stimulation, Pelvic floor muscle, Training, Sexual dysfunction, Sexual Function Index

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NEUROLOGY AND DEMENTIA

Deep learning-based risk assessment of cognitive impairment using health examination data

For effective treatment and prevention of dementia, it is important to develop an objective, accurate, and inexpensive screening test for early diagnosis of cognitive impairment. To resolve these issues, we have developed a deep neural network (DNN) model that can estimate risk of cognitive impairment based on basic blood test data that do not contain dementia-specific biomarkers. The model was based on the relationship between cognitive function and systemic metabolic disorders in the elderly. That is, lifestyle-related diseases can result in vascular cognitive impairment (VCI) due to atherosclerosis. VCI plays an important role not only in vascular dementia, but also in the development of dementia in the elderly with Alzheimer's disease (AD). In addition, malnutrition, anemia, diabetes mellitus, liver dysfunction, and renal dysfunction can cause cognitive impairment and increase the risk of dementia. Importantly, these systemic metabolic disorders, including lifestyle-related disorders, can be detected by basic blood tests for health examinations that do not contain dementia-specific biomarkers. The estimation accuracy of the DNN model was validated in subjects who were not included in the training of the DNN model ($r = 0.66$, $p < 0.001$). The binary classification based on MMSE scores (cut-off value of 23/24) showed a high estimation accuracy (75%) and specificity (87%). In addition, we evaluated whether the DNN model makes it possible to estimate cerebral atrophy based on basic blood test data. Like the DNN model for estimating cognitive function, the input data used were subject age and basic blood test data. As the output of the DNN model, we used the MRI-based brain health quotient (BHQ), which measures the amount of gray matter (GM-BHQ) and the proportion of white matter anisotropy, which was used as an indicator of cerebral atrophy in this study. The DNN model was trained using brain docking data. We found a high estimation accuracy of the DNN model using repeated 5-Fold cross-validation. Moreover, even if only blood data were input without including age in the input data, the estimation accuracy was high. These results suggest that the DNN model allows us to estimate cognitive dysfunction and cerebral atrophy with high accuracy using basic blood test data, which does not include dementia-related biomarkers such as amyloid β.

Audience Take Away Notes

- The audience will pay more attention to the systemic metabolic status of dementia patients by understanding the close relationship between systemic metabolic disorders and cognitive function and brain atrophy
- In order to reduce risk factor for dementia onset, the audience will put more effort into improving systemic metabolic disorders such as lifestyle-related diseases

Kaoru Sakatani

Department of Human and Engineered Environmental Studies, Graduate School of Frontier Sciences, The University of Tokyo, Kashiwa City, Chiba Prefecture, Japan

Biography

Kaoru Sakatani received MD in 1981, D.Med.Sci. in 1987, and Dr.Eng in 1998. He is a Research Fellow (ex-professor) in Graduate school of Frontier Sciences of the University of Tokyo. He is a Board-certified Neurosurgeon in Japan, and his research interests include prevention medicine, biomedical engineering, and data sciences. He is a recipient of the Medical Research Encouragement Prize of The Japan Medical Association (2010). He is the president of International Society on Oxygen Transport to Tissue (ISOTT), the vice president of Japan Optical Brain Functional Imaging, and executive members of Japanese Society of Integrative Medicine.

- I believe that the application of deep learning to clinical dementia will progress
- This research will become a new mass screening method for dementia and contribute to the early detection and prevention of dementia
- It will be possible to select high-risk dementia patients more accurately and efficiently than the interview-type tests (such as MMSE) currently used for dementia screening
- List all other benefits
- It uses only data of health checkup including blood data, thus, no need of blood sampling
- It does not use special equipment for measurering dementia-specific biomarkers, thus, not expensive
- It allows using smartphone for personal assessment dementia risk personally
- Individual risk of dementia can be known from blood data findings, making it possible to provide personalized dietary guidance

Evaluation of the neuroprotective potential of Indicaxanthin from *Opuntia ficus indica* fruit against dysmetabolism-related neurodegeneration in high-fat-diet-fed mice.

Overnutrition and modern diets containing high proportions of saturated fat are amongst the major factors responsible for the development of low-grade, systemic, chronic inflammation, dyslipidaemia and hyperglycaemia. Interestingly enough, recent studies started to recognize oxidative stress-related glucose dysmetabolism as a risk factor also for neurodegenerative conditions such as Alzheimer's disease (AD) and other cognitive disorders. Coherently, over 80% of AD patients have T2DM or abnormal serum glucose levels, suggesting that the pathogenic mechanisms of T2D and AD might well overlap in a self-feeding, vicious cycle between chronic neuro-inflammation, mitochondrial dysfunction and oxidative stress. In these scenarios, the molecular interconnections between neuro-inflammation and glucose dysmetabolism could represent a potential therapeutic target to prevent or ameliorate neurodegeneration and cognitive impairment.

Indicaxanthin (Ind) is a bioavailable, betalain pigment from Opuntia ficus indica fruit, able to cross the brain-blood-barrier. Relevantly, the phytochemical has been demonstrated to interfere with redox-dependent signalling pathways, exerting significant anti-oxidative and anti-inflammatory effects both in vivo and in vitro. Recent evidences also demonstrated that a nutritionally-relevant dose of Ind ameliorates glucose dysmetabolism and counteracts IR in a murine model of metabolic syndrome. Along these lines and taking into account the strict interconnections between neurodegeneration, oxidative stress and glucose dysmetabolism, this work has explored whether and how Ind, extracted from Opuntia ficus indica fruit, exerts protective effects in an in vivo model of dysmetabolism-related neurodegeneration.

To this end, Ind was purified as detailed in the Italian Patent Application No. 102021000015167 filed on 10.06.2021. C57BL/6J mice (n=24) were grouped as follows: 1. a negative control group was fed with a standard diet for 14 weeks; 2. a positive control group was fed with a high fat diet (HFD) for 14 weeks; 3. an Ind-group was fed with HFD for 10 weeks and subsequently received Ind per os at a nutritionally relevant dose of 0.86 mg/kg/day for 4 weeks within a HFD regimen. At the end of the treatment, mice were sacrificed, brains rapidly dissected for hippocampus and cortex and neuro-apoptosis, neuro-inflammation and oxidative stress evaluated.

Our results clearly show that Ind treatment significantly counteracted the HFD- induced brain weight reduction and apoptotic nuclei formation (evaluated by TUNEL assay). Moreover, the phytochemical administration also inhibited neuro apoptosis by reducing Fas- L, Bim, and P27 mRNA levels while increasing the Bcl-2 and BDNF ones (assessed by qRT-PCR).

Mario Allegra*; Simona Terzo; Ilenia C. Giardina; Antonella Amato; Alessandro Massaro; Pasquale Calvi; Alessandro Attanzio; Flavia Mulè; Antonella D'Anneo; Ignazio Restivo; Luisa Tesoriere

Department of Biological, Chemical and Pharmaceutical Sciences and Technologies, University of Palermo, Palermo, Italy

Biography

Prof. Mario Allegra studied Chemistry and Pharmaceutical Technologies at the University of Palermo, Italy and graduated as PharmD in 1997. He then joined the research group of Prof. Perretti at the Department of Biochemical Pharmacology, Queen Mary's University of London. Since 2000 he has been working at the University of Palermo where he received his PhD in Molecular Medicine and now holds a position of Associate Professor of Biochemistry. His research interests cover the role of phytochemicals in oxidative stress-dependent inflammatory conditions. He has published more than 70 research articles in SCI(E) journals, with 4545 citations and an h-index of 30.

From a mechanistic perspective, Ind-mediated effects on HFD-induced neurodegeneration were associated with a reduction of brain oxidative stress and neuro-inflammation. Indeed, Ind treatment effectively counteracted the HFD-induced RONS, malondialdehyde and NO production, evaluated by spectrofluorimetric and colorimetric assays. Coherently, an increase of the expression levels of Nrf-2 and its downstream activation products HO-1 and SOD-2 was also observed in the Ind-group. Along these evidences, Ind administration also ameliorated neuro-inflammation assessed as expression levels of NF-□B and its downstream activation products iNOS and COX-2 by western blotting.

As a whole, our present results indicate that Ind treatment is able to counteract neurodegeneration in an in vivo model of metabolic syndrome, at a nutritionally relevant dose. Modulation of the expression of crucial genes and proteins involved in the oxidative stress-dependent inflammatory reaction underlying the obesity-related neurodegeneration emerges as a possible mechanism for the observed neuroprotective effects. In perspective, our data suggest a potential employment of Ind, alone or in combo-therapy, to prevent and/or treat neurodegenerative conditions associated to glucose dysmetabolism.

Audience Take Away Notes

- The neuroprotective effects and mechanisms of Indicaxanthin, a novel, combo- therapeutic molecule
- Relevant information for biochemists, neurologists, nutritionists, pharmacologists

Personalized and precision medicine (PPM) as a unique healthcare model to secure the human healthcare, wellness and biosafety through the view of public health

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. In this regard, an upgraded model of healthcare service, which includes the philosophy, principles and armamentarium of PPM and aimed at identifying the disorder at its early (subclinical) stage, is being created and set up. PPM focuses on predictive and preventive measures that contribute to the development of individualized strategies for managing a healthy lifestyle that stabilize morbidity rates and can help to improve the working capacity of the population. 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 with clinical medicine, followed by the introduction and promotion of new generation's translational applications. Opportunities exist at every stage of disease initiation and progression to develop a Personalized Health Plan (PHP) addressing lifestyle, risk modification and disease management, and later, Personalized Health Management & Wellness Program (PHM&WP). So, a combination of genomic and phenome-related biomarkers is becoming of great significance to be applied in PPM and need to be translated into the daily practice to predict risks of the disease chronification and thus of disabling. PPM has drastically changed and is keeping on changing the landscape of healthcare. In reality, PPM is the new revolution in medicine which is dramatically modifying the traditional paradigm in medicine with huge consequences for health care systems. And putting PPM-tools in a public health perspective requires an apprehension of the current and future public health challenges. Due to our experience, a symbiotic relationship between public health and PPM may exist. And this approach will be possible only with the integration of data across levels of influence and analytic wisdom in using those data toward better identification of disease and lifestyle risks. In this sense, all healthcare professionals of the future should be educated to deliver patient-centric care as members of interdisciplinary teams, emphasizing evidence-based practice, quality improvement approaches and bioinformatics. And thus greater collaboration between clinician and patient (or person-at-risk) would replace the traditional clinician-dominated dialogue with more effective patient-clinician partnerships. So, the current model physician-patient would have to be gradually displaced by a medical advisor-healthy persons-at-risk model. And biological and medical data

William Thilly[6,7], Shawn Murphy[8], Daniel Scherman[16,17], Andrew D. Miller[12-15], Elena Antonova[1,23], Hiroyuki Abe[9,10], Daryl Pritchard5, Alan Wu[19,20], Carlos Malpica[21,22], Abner Notkins[11], Steven Dooley[17,18], Sergey Suchkov[1-5*]

[1]Institute for Global Health of MGUPP, Russia

[2]A.I.Evdokimov Moscow State University of Medicine & Dentistry, Moscow, Russia

[3]European Powder Metallurgy Association, EPMA, Brussels, European Union

[4]ISPM, Tokyo, Japan

[5]PMC, Washington, DC,United States of America

[6]MIT Center for Environmental Health Sciences

[7]Dept of Biological Engineering, MIT, Cambridge, MA, United States of America

[8]Center for Bioinformatics of the Harvard Medical School, MGH and Partners Healthcare, Boston, MA, United States of America

[9]International Society for Personalized Medicine (ISPM)

[10]Abe Cancer Clinic, Tokyo, Japan

[11]National Institutes of Health (NIH), Bethesda, MD, United States of America

from individuals can be analyzed with environmental data to determine the drivers of health and well-being. This approach mentioned should be based on postulates which will change the incarnate culture and social mentality!. The core functions of national and local government need to be strengthened, especially in relation to comprehensive understanding of the causes of the causes of inequities in health, participatory governance that engages and empowers individuals and communities and that adopt new roles in creating the conditions in which power is shared and health and well-being are co-produced with citizens and communities. It means concerted action by the range of individuals, agencies and all levels of government that can affect the social determinants of health, fostering whole-system approaches to addressing inequities in health. Thus, the main task of PPM is to extend healthy life and increase the size of working-age population, with simultaneous and timely detection of pathological changes in the body, and targeted measures aimed at preventing diseases. Implementation of PPM requires a lot before the current model physician-patient could be gradually displaced by a new model medical advisor-healthy person-at-risk. This is the reason for developing global scientific, clinical, social, and educational projects in the area of PPM to elicit the content of the new branch. So, to fully harvest the unique potential of PPM and PPM-based Public Health, 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 healthcare system. In this connection, the healthcare providers, public policy sector, and consumer industries will be required to develop new and creative models and products. And, no doubt, next generations will speak about the XXI century as a time, when medicine became preventive and personalized, and its outcomes – predictive and guarantied.

[12]Project FIT

[13]Veterinary Research Institute, Brno, Czech Republic

[14]Department of Chemistry and Biochemistry, Mendel University, Brno, Czech Republic

[15]School of Pharmacy, Shanghai Jiao Tong University, Shanghai, PRC

[16] Scientific Research National Center (CNRS), Paris, France

[17]National Scientific Research Center, Paris, France

[18]Medical Faculty Mannheim of the University of Heidelberg and 18University Hospital Mannheim II, Germany

[19]Clinical Chemistry Lab, SFGH

[20]Clinical Pharmacogenomics Lab, UCSF, San Francisco, CA, United States of America

[21]MLP Vision Biotech S.L, Madrid, Spain

[22]Valdia Health, Madrid-Spain, Doha-Qatar

[23]N.F. Filatov Moscow Municipal Clinical Pediatric Hospital, Moscow, Russia

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 Chair, Dept for Personalized Medicine, Precision Nutriciology and Biodesign of the Institute for Global Health and Biotech of MGUPP, and Professor of the Dept for Clinical Allergology & Immunology of A.I. Evdokimov MGMSU, Russia. He 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.

Neural stem cell-derived exosomal miR-9a-5p+ overexpression improving neurogenesis in ischemic stroke

Background: Ischemic stroke is a serious disease which can result in physical disability and death. The protective effect of miRNA up-regulated exosomes derived from neural stem cells (NSCs) on ischemic stroke and its molecular mechanism were investigated, aiming to reveal the therapeutic target of exosome and provide new ideas for ischemic stroke.

Methods: The miRNAs differentially expressed in exosomes derived from NSC at different differentiation periods were detected by miRNA high-throughput sequencing technology. The effects of up-regulating miR-9a-5p were assessed on proliferation and differentiation of NSCs. The different effects between exosomes derived from normal and miR-9a-5p up-regulated NSCs on cell survival, proliferation, differentiation and AMPK activation were investigated in vitro and in vivo.

Results: RNA high-throughput sequencing analysis showed that miR-9a-5p was differentially expressed of NSC-derived exosomes of at different stages. Up-regulation of miR-9a-5p exosomes promotes cell proliferation and differentiation of NSCs after OGD/R, and the apoptosis was reduced significantly. The vivo study revealed that miR-9a-5p exosomes decrease the infarct volume and BBB permeability of ischemic stroke. The apoptosis of neuron, astrocytes and Oligodendrocyte were improved, which suggested that miR-9a-5p have the ability to regulate the differentiation of endogenic NSCs. AMPK activation was increased in MACO/R rat with miR-9a-5p exosomes.

Conclusion: miR-9a-5p exosomes promote AMPK phosphorylation and HIF-1α, so as to promote NSC survival, proliferation, migration and differentiation, which have ability to protect ischemic stroke. MiR-9a-5p is a potential therapeutic target for ischemic stroke. Exosomes are a potential drug carrier, and it is feasible to artificially modify the components of exosomes.

Lukui Chen

Southern Medical University, China

Biography

Lukui Chen had completed his PhD at the age of 35 years from Central South University, China. He is the director and professor of Neuroscience Center in Integrated Hospital of Traditional Chinese Medicine, Southern Medical University, China, since 2019. He has over 40 publications that have been cited over 1000 times. He has been serving as Guest Editor of Frontiers Journals.

Development of imaging based biomarkers for neurovascular abnormalities in neurodegenerative diseases

Neurovascular abnormalities play a critical role in the pathogenesis of neurodegenerative diseases. Impaired small blood vessels and CSF clearance through cerebral lymphatic vessels are often observed in such diseases. Noninvasive imaging biomarkers can provide a powerful tool for diagnosing the diseases, monitoring the progression, and evaluating potential therapeutics. In this talk, I will first describe recent development of MRI techniques for imaging small blood and lymphatic vessels in the brain. I will then discuss the application of these MRI techniques in several brain diseases to develop imaging based biomarkers associated neurovascular abnormalities.

Audience Take Away Notes

- Advanced MRI techniques for imaging small vessels in the brain, Small blood vessel abnormalities in brain diseases, Impaired cerebral lymphatic vessels in brain diseases
- The techniques are available on mainstream clinical MRI systems that can be shared worldwide
- Abnormalities in small blood and lymphatic vessels are commonly seen in brain disorders. Noninvasive imaging techniques can provide sensitive and specific information about such abnormalities which can be used as potential biomarkers for tracking disease progression as well as potential treatment targets for the development of novel therapeutic interventions

Jun Hua

Neurosection, Division of MRI Research, Russell H. Morgan Department of Radiology and Radiological Science, Johns Hopkins University School of Medicine, Baltimore, Maryland, United States

F.M. Kirby Research Center for Functional Brain Imaging, Kennedy Krieger Institute, Baltimore, Maryland, United States

Neurosection, Division of MRI Research, Russell H. Morgan Department of Radiology and Radiological Science, Johns Hopkins University School of Medicine, Baltimore, Maryland, United States

F.M. Kirby Research Center for Functional Brain Imaging, Kennedy Krieger Institute, Baltimore, Maryland, United States

Biography

Dr. Hua's research has centered on the development and application of novel MRI technologies for in vivo functional and physiological imaging in the brain. These include the development of human and animal MRI methods to measure functional brain activities, cerebral perfusion and oxygen metabolism at high (3 Tesla) and ultra-high (7 Tesla and above) magnetic fields. He is particularly interested in novel MRI approaches to image small blood and lymphatic vessels in the brain. Collaborating with clinical investigators, these techniques have been applied to detect functional, vascular and metabolic abnormalities in the brain in neurodegenerative diseases.

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DAY 02

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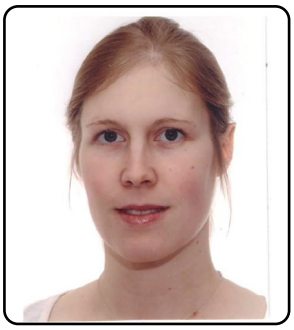
SPEAKERS

JOINT EVENT ON

NEUROLOGY AND DEMENTIA



Anne Dorothee Rosch, PhD

School of Philosophy, Psychology and Language Sciences, University of Edinbuigh, UK

Examining bilateral cortical activity in patients with Parkinson's disease and healthy controls during speech production: A FNRIS study

Background: According to the Hemispheric Asymmetry Reduction in older Adults model (HAROLD; Cabeza, 2002) healthy elderly (HE) may show bilateral cortical activity patterns compared to younger subjects showing more left lateralized cortical activity. The HAROLD model has been discussed as a compensatory process allowing HE to activate more resources to respond to cognitively demanding tasks. Parkinson´s disease (PD) is a progressive neurodegenerative disease affecting different motor and non-motor abilities. Regarding Parkinsonian speech, patients may often suffer from dysarthria, a condition summarizing an array of speech deficits related to the breakdown of speech motor control due to PD (Darley et al., 1969 a, b). Furthermore, with PD progression, further cognitive detriment ranging from mild cognitive impairment (MCI) to dementia (Aarsland et al., 2005; Williams-Gray et al., 2007) may occur. Taken together, it may be hypothesized that PD alters or inhibits bilateral cortical activity. Thus, this study shall address whether PD patients may resort to similar bilateral activity patterns as observed in HE peers or not.

Objective: This study examined cortical activity (e.g. hemodynamic changes) employing functional near-infrared spectroscopy (fNIRS) during different spontaneous and elicited speech production tasks in people aging with and without PD.

Methods: Functional NIRS was measured in 19 non-demented PD patients (Age Mn 66.5 yrs. [54-75]; 6.2) having no deep brain stimulation (non-DBS) and 18 HE (Age Mn 62.8 yrs. [52-77]; 7.8) during three different speech production tasks: (i) sentence repetition, (ii) reading and (iii) spontaneous speech (short interview). The speech tasks are outtakes from the Bogenhausener Dysarthrieskalen (Body's; Ziegler et al., 2018) being a standardized German measurement for dysarthria. The 8x8 fNIRS system integrated short channels and covered the Broca´s area, the Motor Cortex and the Wernicke´s area, as specified by the fold brain atlas (Morais et al., 2018). For calculations, fNIRS signals during speech task blocks were averaged for every task and group, respectively. Statistical analysis was conducted with SPSS (V.28) and results were seen as significant if $p < .05$ or less.

Results: Paired samples t-tests for PD patients showed only one significant difference within the spontaneous speech task in the Motor Cortex ($p = .04$), with higher fNIRS signals in the left compared to the respective right hemispheric area. In the HE group, significant differences between the hemispheres were found in the Motor Cortex across all tasks (i.e. reading, spontaneous speech and sentence repetition) ($p < .03$). There, higher fNIRS signals were always found in the left compared to the right cortical structures. Interestingly -across groups- there were no significant differences between hemispheres within the sentence repetition task. Finally, fNIRS signals in PD patients were lower than those observed in HE controls ($p < .01$) across all conditions.

Conclusions: Results suggest that hemodynamic changes were lower in PD patients compared to HE controls, and that these were mostly bilateral. Interestingly, in HE left lateral activation remained within the Motor Cortex. In contrast, PD showed bilateral activation patterns possibly indicating a compensatory mechanism kicking in once neuronal deficiencies inhibit normal processes.

Audience Take Away Notes

- Professionals are introduced to the theories of the HAROLD model, and how recent results may be linked to it: Results from the bilateral activity, which is -surprisingly- more pronounced in patients suffering from neurodegeneration than age-peers with normal age-related degeneration, may contribute to the current knowledge of natural compensatory mechanisms, which have so far received less attention in research, teaching and clinical practice
- In research, cortical bilateral activity should not be overlooked or mislabeled as artifact (for instance), as different cortices may have or take over more functions and may thus prove to have more neuroplasticity in age than formerly expected
- Natural compensatory mechanisms can be used in clinical practice and thus shape new approaches and therapies
- In teaching, recent findings contribute to evidence-based practice, critical thinking and allow novel designs

Biography

Anne D. Rosch is a speech-language therapist with expertise in neurocognitive and linguistic research in neurodegenerative disease such as Parkinson's disease. She currently holds a position as visiting academic international researcher at the University of Edinburgh (UK). Her latest research project (data collection in 2020) investigated hemodynamic cortical responses using near-infrared spectroscopy in patients with Parkinson's disease (PD) and in healthy elderly before, during and after rhythmic interventions.

Ji-Hoon Jeong[1] and Keun-Tae Kim[2*]

[1]Department of Computer Science, Chungbuk National University, Chungju, Republic of Korea
[2]Bionics Research Center, Biomedical Research Division, Korea Institute of Science and Technology, Seoul 02792, Republic of Korea

Brain-machine teaming: Recent advances for neural language decoding from EEG signals

Brain-machine teaming is a collaborative interfacing system between the human brain and external machines. It is a bidirectional communication concept based on the brain-machine interface (BMI). Especially, electroencephalogram (EEG)-based BMI has been utilized to help patients regain motor function and has recently been validated for its use in healthy people because of its ability to directly decipher human intentions. In particular, neurolinguistic research using EEGs has been investigated as an intuitive and naturalistic communication tool between humans and machines. In this study, we have focused on the EEG signals with respect to speech imagery tasks, and the proposed deep neurolinguistic learning architecture could be decoded neural languages. Five subjects participated in the experiment and we evaluated whether BMI-based cooperative tasks between multiple users could be accomplished using a variety of neural languages. We successfully demonstrated brain-machine teaming that allows a variety of scenarios, such as essential activity (e.g., drinking juice by the user himself or herself, or providing juice to the partner), collaborative play (e.g., drinking juice with the partner's help and delivering a phone with a box to the partner), and emotional interaction (e.g., expressing the user's emotions to the partner). Consequently, a novel BMI frontier can be presented from these outcomes that could extend the boundaries of the bidirectional interaction between the brain and machine team playing.

Audience Take Away Notes

- How to decode the EEG signals using machine and deep learning techniques in real-time
- How to detect user speech intention (i.e., neural language) as sentence-level
- How to collaborative play between multiple users with neuroprosthetic arm
- What are the frontiers of the brain-machine interfacing system

Rui-Yuan Pan

The Brian Science Center, Beijing Institute of Basic Medical Sciences, Beijing, China

Intermittent fasting protects against Alzheimer's pathology via the gut-microbiota-metabolites-brain axis

Alzheimer's disease (AD) is the most common form of dementia without effective clinical treatment. Here, we show that intermittent fasting (IF) improves cognitive functions and AD-like pathology in a transgenic AD mouse model (5XFAD). IF alters gut microbial composition with a significant enrichment in probiotics such as Lactobacillus. The changes in the composition of the gut microbiota affect metabolic activities and metabolite production. Metabolomic profiling analysis of cecal contents revealed IF leads to a decreased carbohydrate metabolism (e.g., glucose) and an increased abundance in amino acids (e.g., sarcosine and dimethylglycine). Interestingly, we found that the administration of IF-elevated sarcosine or dimethylglycine mimics the protective effects of IF in 5XFAD mice, including the amelioration of cognitive decline, amyloid-□ (A□) burden, and glial over-activation. Our findings thus demonstrate an IF regimen is a potential approach to prevent AD progression at least through gut-microbiota-metabolites-brain axis, and constitutes an innovative AD therapeutic avenue.

Audience Take Away Notes

- Intermittent fasting (IF) regimen improves cognitive functions and AD-like pathology in 5XFAD mice
- Gut microbiota is required for the beneficial effects of IF against AD
- IF-enriched metabolites sarcosine and dimethylglycine prevent AD progression

Biography

Dr. Rui-Yuan Pan studied Biological Science at the Minzu University of China and received his Ph.D. degree in Neuroscience at Institute of Biophysics, Chinese Academy of Sciences, and Beijing, China in 2020. Dr. Pan is now training as a postdoctoral researcher in the Brian Science Center, Beijing Institute of Basic Medical Sciences, Beijing, China. His researches focus on the pathogenesis of AD, especially the metabolic regulation of glial functions in neurodegenerative diseases, aiming to identify targets and develop candidate drugs for the diseases. His works have been published in Cell Metabolism, Nature Aging, and Science Advances etc.

Kai Wen He

Interdisciplinary Research Center on Biology and Chemistry (IRCBC) Chinese Academy of Science, China

Brain region-specific tau hyperphosphorylation mediates sleep disruption associated with early stage of Alzheimer's diseases

Sleep disorders are commonly found in Alzheimer's disease (AD) patients, which can occur at very early stage of AD and are proposed as potent risk factors for disease onset and progression. What may cause these early-onset sleep deficits remains unexplored. Here by thoroughly characterizing the sleep/wake pattern of 2-month-old 5xFAD mice, we first confirmed that these AD transgenic mice show abnormal sleep at young age similar to human patients. Specifically, they show shortened sleep and prolonged wake duration. Interestingly, we found that these AD mice develop brain region-specific tau hyperphosphorylation (p-tau) in Locus Coeruleus (LC). Importantly, selective elevation of p-tau in LC neurons is sufficient to trigger AD-like sleep phenotype in wildtype mice, supporting a critical causal role of LC p-tau in AD-associated early sleep disruption. We at last revealed that p-tau alters LC neural excitability and pharmacologically reducing p-tau rescues LC neuron hyperexcitability both in wildtype and young 5xFAD mice. Therefore, our study unveil a novel pathomechanism associated with early stage of AD.

Biography

Dr. Kai-Wen He graduated from Tsinghua Univ with a bachelor degree in biological science and then received her PhD in neuroscience from Univ of Maryland College Park. After finishing her postdoctoral training in the Johns Hopkins University, Dr, He establishes her independent laboratory in Interdisciplinary Research Center on Biology and Chemistry (IRCBC) of Chinese Academy of Science (CAS). Her lab is mainly interested in understanding how neural and brain function is regulated in health and diseases especially the early stage of neurodegeneration.

Sergey Suchkov[1-3*], Veromika Medvedeva[1,6], Noel Rose[4,7], Aleks Gabibov[5]

[1]Institute for Global Health & Biotech of MGUPP, Russia

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

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

[4]Johns Hopkins Center for Autoimmune Disease Research and PAHO/WHO Collaborating Center for Autoimmune Disorders, Johns Hopkins Medical Institutions, Baltimore, MD, USA

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

[6]Sechenov University, Moscow, Russia

[7]Harvard Medical School, Boston, MA, USA

Antibody-proteases as the upgraded translational tools of the next-step generation in personalized and precision Neurology practice

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 Bio-design.

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! Anyway, further studies on targeted Ab-mediated proteolysis may provide a translational tool for predicting demyelination and thus the disability of the MS patients.

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 Chair, Dept for Personalized Medicine, Precision Nutriciology and Biodesign of the Institute for Global Health and Biotech of MGUPP, and Professor of the Dept for Clinical Allergology & Immunology of A.I. Evdokimov MGMSU, Russia. He 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.

Esraa Askar MD[1*], Neeraj Singh MD[1, 2]

[1]Department of Medicine, Northwell Long Island Jewish - Forest Hills Hospital, Forest Hills, New York

[2]Department of Neurosciences, Zucker School of Medicine at Hofstra/Northwell

Psychosocial considerations in management of corticobasal degeneration

Background: Corticobasal degeneration (CBD) is a rare neurodegenerative tauopathy primarily affecting the cerebral cortex and basal ganglia, often featuring cognitive decline and Parkinsonism. With no proven treatments, management goals include improving patients' quality of life. We evaluate here the varying presentations of symptoms and psychosocial factors affecting a series of women of Asian Indian descent with CBD.

Case Descriptions: Patient 1 was a 77-year-old woman with short-term memory loss, who then had left anterior cerebral artery territory ischemic stroke with right-sided hemiparesis and neurogenic pain. MRI Brain confirmed the stroke's location but did not account for cognitive decline. Patient 2 was a 75-year-old woman with dysphagia, who then developed right-sided hemiparesis, right neck spasms, unstable gait, and short-term memory loss. MRI Brain and Cervical Spine revealed only showed mild chronic microvascular changes and multilevel cervical spondylosis. Patient 3 was a 49-year-old woman with left hand and leg tremors, treated with carbidopa/levodopa, who then developed weakness and paresthesias in all four extremities. DaTScan revealed bilateral caudate nuclei and putamina dysfunction, explaining the parkinsonian tremors but not the other symptoms. Patient 4 was an 82-year-old woman with left foot weakness impairing gait and causing multiple falls, who then developed slurred speech. MRI Brain showed chronic microvascular changes, but no stroke or other abnormality to explain focal neurological symptoms.

Results: PET-FDG Brain studies were performed for all patients. Patient 1's study revealed severe hypometabolism in bilateral basal ganglia, bilateral thalami, and left supplementary motor cortex, out of proportion to the known stroke. Patient 2's study revealed mild hypometabolism at the left frontal and parietal lobes, sparing the precuneus and posterior cingulate, which explained right-sided hemiparesis. Patient 3's study revealed mild hypometabolism at the left sensorimotor cortex, basal ganglia, and thalamus, explaining weakness and paresthesias on the right side of the body. These three patients had findings consistent with corticobasal syndrome phenotype of CBD. Patient 4's study revealed hypometabolism in bilateral premotor cortices, supplementary motor areas, and anterior cingulate gyri, consistent with progressive supranuclear palsy phenotype of CBD.

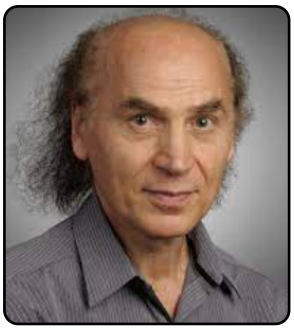
Conclusions: Various psychosocial factors were considered during evaluation and management for all patients. They had limited English proficiency, so cognitive testing was performed with medical interpreter assistance to ensure accurate results. Symptomatic treatment was tailored for each patient: Patients 1 and 3 received carbidopa/levodopa for parkinsonian tremors, while Patient 2 received cyclobenzaprine for intermittent muscle spasms. All patients had multigenerational households, and results and prognoses were discussed with multiple relatives so they could coordinate care responsibilities. All families were reluctant to introduce home health aides, expressing cultural beliefs that families should take care of their relatives on their own. As we discussed these beliefs over time, and explained the anticipated symptom progression over time, all families became more accepting of introducing home health aides, seeing them as a supplement, and not a replacement, for the family's role. Additional research is needed to explore the etiology and psychosocial impacts of CBD, to help develop appropriate treatments and conservative measures specific for this neurodegenerative condition.

Audience Take Away Notes

- Describe the definition and various presenting symptoms of Corticobasal degeneration
- Identify typical neuroimaging findings associated with Corticobasal degeneration
- Outline various psychosocial factors affecting management of patients with Corticobasal degeneration
- Recognize the importance of further research on Corticobasal degeneration

Biography

Dr. Askar graduated medical school at Faculty of Medicine, Alexandria University, Egypt in 2016. She then worked as a teaching assistant in the Microbiology and Immunology Department of the Faculty of Medicine, Alexandria University for 2 years, during which she finished her USMLE exams and then joined the Internal Medicine Residency training program at Department of Internal Medicine, Northwell/Hofstra Zucker School of Medicine, and Long Island Jewish Forest Hills, NY, USA. She was honored to be the intern of the year during her first year of residency 2020-2021. She then was chosen by her program to be the associate chief resident during her third year of residency during 2022-2023.



Suren A. Tatulian
Department of Physics, University of Central Florida, Orlando, FL, USA

Peptide-based inhibitors of amyloid beta aggregation and toxicity as alzheimer's drug candidates

There are around 35 million individuals affected by Alzheimer's disease (AD) dementia worldwide, and currently there are no effective disease-modifying medicines for AD. During the last 2.5 decades, four FDA-approved AD drugs, i.e., donepezil, rivastigmine, galantamine, and memantine, have been available. These are symptomatic drugs and do not modulate the underlying disease process. The amyloid beta (Aβ) peptide and the tau protein, especially its hyperphosphorylated form, have been the major therapeutic targets of AD drug development. Various strategies have been employed, i.e., inhibition of Aβ production by β- and γ-secretases, inhibition of Ab aggregation, clearance by monoclonal antibodies (mAbs), immunization, inhibition of tau phosphorylation etc. Two anti-Aβ mAbs, aducanumab and lecanemab, have been recently approved by FDA (June 2021 and January 2023, respectively), but their disease-modifying potency remains questionable. Unfortunately, most of AD drug development efforts have failed due to various reasons, e.g., lack of efficacy of adverse events such as edema or cerebral hemorrhage.

Despite the evidence that peptides and peptidomimetics have shown strong potential in inhibiting Ab aggregation and cytotoxicity, peptide-based compounds are not recruited as potential AD drugs. This presentation will address this gap by identifying peptide sequences with superior capabilities of inhibiting Ab aggregation and cytotoxicity that may become effective AD drug candidates.

Peptides unrelated to Aβ (e.g., YAibWF, RYYAAFFARR, NAVRWSLMRPF) or corresponding to defined stretches of Aβ, e.g., Aβ16-22 (KLVFFAE), strongly inhibited Aβ aggregation and toxicity. Our research group has recently analyzed the influence of overlapping 10- to 12-residue fragments of Aβ1-42 on the aggregation of the parent peptide (Abedin F, Kandel N, Tatulian SA. Sci Rep (2021) 11:19262). Aβ11-20 (P3) and Aβ26-36 (P6) inhibited the aggregation of Aβ1-42 by 80-90%. Moreover, P3 and P6 totally impeded Aβ1-42 toxicity to PC-12 cells at 2-fold molar excess, identifying these peptides as potential AD drug candidates.

Obstacles in delivery of peptides to the brain parenchyma, such as the blood-brain barrier (BBB) or hydrolysis by gastrointestinal or plasma enzymes, can be mitigated by methods like cyclization or N- and C- terminal capping, mutations that remove the cleavage site(s), intracerebroventricular infusion, nasal administration, or permeabilization of the BBB by focused ultrasound. Thus, short peptides emerge as potent inhibitors of Ab aggregation and cytotoxicity, with strong potential for becoming viable AD drugs.

Audience Take Away Notes

- Researchers may use the information to explore other peptides to identify strong inhibitors of Aβ aggregation and cytotoxicity
- The material presented here can be used by other scholars to expand their research or teaching by involving peptides as modulators of Ab in both their research and teaching
- Using peptides that are modified by capping or cyclization may offer solutions to metabolic instability and cell permeation of the peptides

Biography

Dr. Tatulian received his BS in Physics from Yerevan University, Armenia, and his PhD in Biology from the Institute of Cytology, St. Petersburg, Russia, where he studied membrane transport. He joined the University of Virginia Medical School as a Research Associate in 1992 and was promoted to Assistant Professor in 1995. In 1999, he moved to the University of Kansas, Lawrence, KS, as Associate Professor. In 2001, he moved to the University of Central Florida where he was tenured and promoted to full Professor. Dr. Tatulian's research focus is on protein structure and function and molecular mechanisms of disease.

Xinyu Yang, Feixiang Zhou, Huiyu Zhou*
School of Computing and Mathematical Sciences, University of Leicester, UK

Artificial intelligence based mouse behavior recognition

The behavioural phenotype research can be of great importance as the first symptom of neurodegenerative disorders is often identifiable through subtle changes in day-to-day human behaviors. Because of the similarity and homology between humans and mice, modelling mouse behaviour provides a valuable platform to study neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Comprehensive behavioural phenotypes of transgenic mice can be used to reveal the underlying functional role of genes, and can provide new insights into the pathophysiology and treatment of neurodegenerative diseases carried by the mice.

Historically, studying mouse behaviour can be a time-consuming and difficult task as the collected data requires experts to physically engage. On the other hand, the collected data requires experts to annotate and to analyse mice behaviors manually. It is a highly labor-intensive process which is error-prone and subjective to individual interpretation. Furthermore, human experts may fail to detect behavioral events that are very quick or too slow, and some behaviour events maybe missed because of dwindling attention span. Inspired by the advances in machine learning over the last decades, our works based on computer vision and pattern recognition aim to facilitate automated analysis of complex mouse behaviours. Currently our works mainly cover two problems: mouse pose estimation and mouse behaviour recognition.

As one of the fundamental problems in mouse behaviour analysis, mouse pose estimation is defined as the problem of measuring mouse posture which denotes the geometrical configuration of body parts in images or videos. This can provide useful information for relevant behaviour analysis. However, spatial contextual information between mouse body parts is weak due to highly deformable body structures. For this problem, a novel Graphical Model based Structured Context Enhancement Network (GM-SCENet) is introduced. The proposed architecture can adaptively learn and enhance the structured contextual information of each keypoint by exploring the global and keypoint-specific contextual information. Besides, a Multi-Level Keypoint Aggregation (MLKA) algorithm is designed to integrate multi-level localisation results (https://ieeexplore.ieee.org/abstract/document/9492104).

Regarding mouse behavior recognition, we propose four solutions from different perspectives:

(1) In order to effectively use contextual information in videos to recognize mouse behaviour, novel contextual features of interest points are exploited, which potentially describe spatial location and temporal changes without using an independent tracking or detecting algorithm. These features are then encoded as spatial-temporal stacked Fisher vectors which are used as the input to the neural network for mouse behaviour recognition(https://pureadmin.qub.ac.uk/ws/files/122523899/SciTePressPure_version_ICPRAM.pdf).

(2) Many mouse actions have pairwise relationships in the temporal domain. For example, it is very unlikely to have a hang or rest action immediately after a drink action. Based on this characteristic□a novel hybrid learning architecture for mouse behavior recognition is proposed in which a Hidden Markov Model is used to model the temporal transition relation of mouse behaviours (https://ieeexplore.ieee.org/abstract/document/8488486).

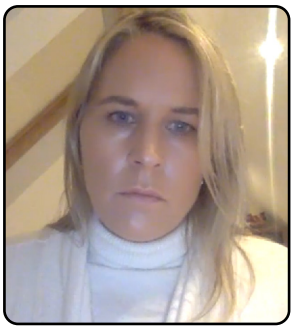
(3) Most vision-based techniques for mouse behavior recognition only rely on the single-view video recordings, which can be ambiguous when essential information of behaviours is occluded. In this work, a novel multi-view mouse behaviour recognition system based on trajectory-based motion and spatio-temporal features is introduced which aims to model: (a) the temporal relationship of image frames in each view, (b) the relationship between camera views, and (c) the correlations between the neighbouring labels (https://ieeexplore.ieee.org/abstract/document/9444134).

(4) Behavioral interaction information between mice is crucial for behavior recognition. Based on mouse skeleton key point information, a Cross-Skeleton Interaction Graph Aggregation Network (CS-IGANet) is proposed to effectively learn abundant interaction relationships between mice. Also, an auxiliary self-supervised learning strategy to enable the proposed model to focus on the similarity between pairwise nodes from different skeletons, so as to enhance the representation ability of the model (https://arxiv.org/abs/2208.03819).

Overall, these works have potential to contribute to conducting a wide range of behavioural experiments without human intervention. We believe these can provide another dimension to understand the relationship between neural activities and behavioural phenotypes in neurodegenerative diseases research.

Biography

Dr. Huiyu Zhou received a Bachelor of Engineering degree in Radio Technology from Huazhong University of Science and Technology of China, and a Master of Science degree in Biomedical Engineering from University of Dundee of United Kingdom, respectively. He was awarded a Doctor of Philosophy degree in Computer Vision from Heriot-Watt University, Edinburgh, United Kingdom. Dr. Zhou currently is a full Professor at School of Computing and Mathematical Sciences, University of Leicester, United Kingdom. He has published over 450 peer-reviewed papers in the field. He was the recipient of CVIU 2012 Most Cited Paper Award, MIUA 2020 Best Paper Award, ICPRAM 2016 Best Paper Award and was nominated for ICPRAM 2017 Best Student Paper Award and MBEC 2006 Nightingale Prize. His research work has been or is being supported by UK EPSRC, ESRC, AHRC, MRC, EU, Royal Society, Leverhulme Trust, Invest NI, Puffin Trust, Alzheimerꞌs Research UK, Invest NI and industry.



Dr Clare Cromwell[1, 2*], Mr Anthony Michael Mander[3]

[1]Dept of Obstetrics and Gynaecology, Cork University Maternity Hospital, Cork, Ireland

[2]The Menopause Clinic, Bons Secours Women's Health Centre, Bons Secours Hospital, Cork, Ireland

[3]Consultant Gynaecologist, Menopause Specialist, Nuffield Manor Hospital, Oxford, UK

Brain fog and menopause – Is the future getting clearer?

As GPs and Menopause Specialists brain fog is a frequent topic and often the most distressing and debilitating symptom expressed in consultation. It can present in a way that we don't recognise and the diagnosis is too easily missed. It can present in primary care, neurology, psychiatry and urgent care settings amongst others. It needs to be recognised and part of a differential diagnosis. But what is brain fog? It's ill defined. How do we know? Have we evidence of objective cognitive decline? Brain fog describes a lay collective term for cognitive difficulties, forgetfulness, cloudiness, cotton wool brain, fuzziness, fogginess. A collection of symptoms such as difficulty focussing, thinking and communicating, difficulty finding words and recalling numbers, forgetting events and appointments. We have a growing bank of evidence that cognitive function declines in late perimenopause and early menopause. We have some evidence that it bounces back. Studies to date have used a battery of neuropsychological tests to show these declines. These tests are done in a clinical setting by serial visits over the course of 2-4 years. Some testing is performed under observation in an fMRI scanner to show the changes in task evoked hippocampal responses and connectivity pre and post menopause. We know that cognitive decline in these transitional years can be influenced by other modalities, namely vasomotor symptoms, treatment with hormone replacement therapy, mood and sleep. There is a growing need to detect these early changes, both to research further what treatments can alter or halt their progression and to eliminate misdiagnosis of brain fog and resultant incorrect treatment and referral. I plan to discuss more convenient and accurate digital solutions to characterising and validating subjective complaints of brain fog and potentially treating brain fog with regular focussed brain activity or exercise. These tests can be done in a comfortable home setting in the patient's own time. I propose a cost effective, modern, accurate and efficient way of testing for a treatable and reversible phenomenon.

Audience Take Away Notes

- Discussing the potential link between hormonal change and cognitive decline in midlife and development of dementia in later life

Biography

Dr Clare Cromwell studied medicine in University College Dublin and graduated in 2003. She received her MICGP from the Irish College of General Practitioners and has been practicing as a GP since 2008. Her focus is on Women's Health and Menopause, with her particular interest being cognitive decline during the menopause transition. She is researching this further in order to develop convenient, cost efficient, user friendly tools to recognise the changes but also those red flags signifying progressive cognitive decline and dementia.

Marco Ruggiero
Division of Research and Development, Silver Spring Sagl, Arzo-Mendrisio, Switzerland

Negative entropy-driven self-assembly of information-containing aperiodic crystals aimed at increasing brain entropy in the context of Alzheimer's disease, dementia and cognitive decline

The degree of brain entropy (BEN) is associated with different levels of consciousness. The entropic brain hypothesis contemplates that, within upper and lower cutoff points, beyond which consciousness may be significantly reduced or absent, the entropy of spontaneous cerebral activity represents a measure of the informational richness of conscious states. Consistent with this hypothesis, BEN increases during physiological development, and decreases during states of reduced consciousness, such as aging, Alzheimer's disease, dementia, and cognitive decline. Here, a biomolecular approach aimed at increasing BEN is described; this approach is based on negative entropy-driven self-assembly of two types of information-containing aperiodic crystals. The first type consists of crystals of poly-lysine and glutamic acid; the second type consists of crystals of sulfated polysaccharides containing fucose, galactose, xylose, arabinose and rhamnose, and lysine, alanine, tyrosine and cyanocobalamin. In the first type of crystals, poly-lysine constitutes a backbone for the establishment of electrostatic bonds between the positively charged amino groups of poly-lysine and the negatively charged acidic moiety of glutamic acid. In the second type of crystals, the mixture of lysine, alanine, tyrosine and cyanocobalamin is complexed, through formation of electrostatic bonds, with the acidic backbone constituted by the sulfated polysaccharides where the negatively charged sulfate ester and carboxylic groups of the polysaccharides bind to the positively charged moieties of the amino acids and cyanocobalamin. The two types of aperiodic crystals interact in a CO2-enriched hydrophilic medium with the resulting self-assembly of complex structures composed of random-sized aperiodic crystals of [poly-lysine/glutamic acid], and [sulfated polysaccharides lysine/alanine/tyrosine/cyanocobalamin]. Although the crystals are formed through negative entropy-driven self-assembly, because of their random-size and, therefore, random distribution of electrical charges on their molecular surface, they contain an elevated degree of information according to Shannon's entropy equation. An additional level of information derives from the chemical structure of fucose, a hexose deoxy sugar that is the constituent, in repetitive sequences, of the sulfated polysaccharides taking part in the formation of the aperiodic crystals. The chemical formula of fucose, C6H12O5, highlights the presence of three delocalized pi orbital electrons among six carbon atoms forming pi resonance clouds conducive to quantum effects that are hypothesized to be at the basis of consciousness (Hameroff, 2022, Front Mol Neurosci). The aim of the approach described in this study is to increase richness of consciousness by increasing BEN. In disorders of consciousness where BEN is decreased such as aging, Alzheimer's disease, dementia, and cognitive decline, this approach restores physiological levels of consciousness as demonstrated by electroencephalography and clinical observations. In healthy individuals, it leads to higher states of consciousness that could be particularly evident, for example, during meditation or transcranial ultrasound stimulation (Sanguinetti et al., 2020, Front Hum Neurosci) as demonstrated by electroencephalography and individual reports.

Audience Take Away Notes

- The audience will be able to apply the concepts presented here in the treatment of Alzheimer's disease, dementia, and cognitive decline
- The audience will be able to use innovative approaches for the treatment of Alzheimer's disease, dementia, and cognitive decline
- Yes, it does. The approaches and the solutions presented here will provide practical solutions aimed at treating Alzheimer's disease, dementia, and cognitive decline

Biography

Marco Ruggiero, MD, PhD, worked as post-doc at Burroughs Wellcome Co. (1984) and as visiting scientist at the National Cancer Institute of the NIH (1987). He was appointed Professor of Molecular Biology at the University of Firenze, Italy, in 1992 where he worked until 2014. He published more than 240 scientific articles co-authored, among others, by scientist such as EG Lapetina, SA Aaronson, JH Pierce, PH Duesberg, HH Bauer, JJ Bradstreet, D Klinghardt. One of his articles in PNAS was sponsored by Nobel Laureate Sir John Vane. His current research interests are in immunotherapy, quantum biology and microbiome medicine.

Juliana J. Schmidt[1], Yolanda Eliza Moreira Boechat[2], Guilherme J. Schmidt[1], Eelco van Duinkerken[1], Sergio L. Schmidt[1, 3,*]

[1]Department of Neurology, Federal University of the State of Rio de Janeiro, Rio de Janeiro, Brazil
[2]Department of Geriatrics, Fluminense Federal University, Niteroi, Brazil
[3]Department of Physiological Sciences, Federal University of the State of Rio de Janeiro, Rio de Janeiro, Brazil

Discriminative power of a reaction-time test in the evaluation of cognitive impairment in elderly with high educational disparity

Objective: We aimed to analyze the discriminative power of a culture-free Go/No-Go task by comparing three groups: cognitively unimpaired healthy elderly, Mild Cognitive Impaired subjects (MCI), and early Alzheimer´s disease patients (AD). For the three groups the samples had high educational disparity.

Methods: One hundred and ten participants with a wide range of years of formal education (0–14 years) were randomly selected from a geriatric unit and divided based on their CDR scores into cognitively unimpaired (CDR = 0), MCI (CDR = 0.5), and early AD (CDR = 1). All the participants underwent a 90-second Go/No-go task which consisted of 72 (80%) targets and 18 (20%) non-targets. For each participant, reaction times and intraindividual variability of reaction times of all correct target responses, as well as the number of omission and commission errors were evaluated. For each participant, the coefficient of variability was also calculated by dividing the standard deviation of the reaction times by the correspondent mean reaction time. A MANCOVA was performed to examine between-group differences using educational level, age, and sex as covariates. Discriminate analysis was performed to find the most reliable test-variable to discriminate the three groups.

Results: Mean group differences reached significance for commission errors, intraindividual variability of reaction time, and coefficient of variability. The discriminant analysis demonstrated that coefficient of variability was the most reliable variable to predict the CDR scores.

Conclusion: The Go/No-Go task was able to discriminate people with MCI or early AD from controls independent of the educational level of the participants.

Audience Take Away Notes

- This study provides a practical solution (reaction-time test) to the problem of distinguishing MCI and early AD from healthy elderly
- The test can be applied in different cultures
- The test is easy to administer
- There is no effect of fatigue (the test is quick)
- There is no learning effect and the test can be used many times in order to measure treatment's efficacy
- The solution is affordable because the software is free

Biography

BSc in Physics (major field: biophysics); MD (neurology); Ph.D. (behavioral neurology). Killam post-doctoral fellow at the University of Albert, Canada (U of A) from 1990 to 1993. Adjunct Professor (1992-1995) at the Department of Psychology (U of A). Ambassador of the Alberta Educational System in Brazil (1995-2000). Full Professor at the State University of Rio de Janeiro (2002-2019). Titular Member of the Brazilian Academy of Neurology (2002-till .now). Full Professor at the Federal University of the State of Rio de Janeiro, Brazil (2019- till now).

Cristina Carvalho[1,2,3*], Susana Cardoso[1,2,3], Sonia Correia[1,2,3], Mariana Laranjo[1,2,4], Paula I. Moreira[1,3,5]

[1]Center for Neuroscience and Cell Biology (CNC), University of Coimbra, Portugal
[2]Institute for Interdisciplinary Research (III), University of Coimbra
[3]Center for Innovation in Biomedicine and Biotechnology (CIBB)
[4]PhD Program in Experimental Biology and Biomedicine (PDBEB), University of Coimbra, Coimbra, Portugal
[5]Institute of Physiology, Faculty of Medicine, University of Coimbra, Coimbra, Portugal

Brain mitochondrial function and oxidative stress normalized after WWOX inhibition in a mouse model of type 2 diabetes

Type 2 diabetes (T2D) is a chronic metabolic disorder that significantly increases the risk of dementia, particularly Alzheimer's disease (AD). It is of utmost importance to decipher the mechanisms underlying T2D-associated brain damage to facilitate the development of effective strategies capable of preventing or reducing dementia. It has been recently shown that WW domain-containing oxidoreductase 1 (WWOX) overexpression/over activation plays a pivotal role in mitochondrial dysfunction and development of insulin resistance, features shared by T2D and AD. In this line, this study was aimed to 1) assess the involvement of WWOX in T2D-associated brain damage and 2) evaluate the therapeutic potential of Zfra1- 31 peptide, a specific inhibitor of WWOX. High fat diet (HFD)-induced T2D mice treated or not with 2mM Zfra1-31 for 4weeks (4x; 1injection/week via tail vein) were used as experimental in vivo model. We performed a battery of behavioral and cognitive tests, and we evaluated glucose tolerance and insulin levels by ELISA and enzymatic methods. The levels of oxidative stress markers were evaluated by fluorometric techniques, and the function of brain cortical mitochondria was assessed using a XF24 Extracellular Flux Analyzer. HFD induced a clear phenotype of T2D, as demonstrated by the increase in body weight and peripheral blood glucose levels and decreased glucose tolerance. T2D mice also showed increased levels of anxiety and impaired cognitive function. Mitochondria isolated from the brain cortex of T2D mice presented defects in the respiratory chain and phosphorylative system, decreased calcium buffering capacity and increased oxidative stress levels. Interestingly, T2D mice treated with Zfra1-31 showed an amelioration of blood glucose levels as well as of the anxiety-like behavior and memory impairment, compared to untreated mice Furthermore, Zfra1-31 improved brain mitochondrial respiratory chain, phosphorylation system and calcium buffering capacity and decreased oxidative stress levels. In conclusion, these observations demonstrate that HFD induces a phenotype of T2D associated to behavioral/cognitive and mitochondrial defects. More, our study suggests the involvement of WWOX in T2D-associated brain damage since Zfra1-31 treatment improved behavior and cognition, brain mitochondria function and oxidative stress. The authors' work is supported by ERD, through COMPETE 2020—Operational Programme for Competitiveness and Healthy.

Audience Take Away Notes

- High calorie diets increase the risk of T2D and, consequently, of dementia, particularly AD
- WWOX is a new player in T2D-associated brain damage and represents a possible therapeutic target
- Zfra1-31, a specific inhibitor of WWOX, has a therapeutic potential to fight T2D-associated brain damage

Biography

Cristina Carvalho is a doctorate investigator interested in exploring the mechanisms underlying type 2 diabetes-associated (brain) complications. The ultimate goal is to identify new targets for therapeutic intervention. Her CV comprises >50 papers published in peer-reviewed journals, including a publication in Cellular and Molecular Life Sciences (2022; IF=9.2). In 2021, she won an Exploratory Research Grant from European Society for Clinical Investigation and a FCT Exploratory Research Project (Portuguese Foundation for Science and Technology.

Bitra Veera Raghavulu
University of Botswana, Botswana

mTOR signaling as a molecular target for the alleviation of Alzheimers disease pathogenesis

Mechanistic/mammalian target of rapamycin (mTOR) belongs to the phosphatidylinositol kinase-related kinase (PIKK) family. mTOR signaling is required for the commencement of essential cell functions including autophagy. mTOR primarily governs cell growth in response to favorable nutrients and other growth stimuli. However, it also influences aging and other aspects of nutrient-related physiology such as protein synthesis, ribosome biogenesis, and cell proliferation in adults with very limited growth. The major processes for survival such as synaptic plasticity, memory storage and neuronal recovery involve a significant mTOR activity. mTOR dysregulation is becoming a prevalent motif in a variety of human diseases, including cancer, neurological disorders, and other metabolic syndromes. The use of rapamycin to prolong life in different animal models may be attributable to the multiple roles played by mTOR signaling in various processes involved in ageing, protein translation, autophagy, stem cell pool turnover, inflammation, and cellular senescence. mTOR activity was found to be altered in AD brains and rodent models, supporting the notion that aberrant mTOR activity is one of the key events contributing to the onset and progression of AD hallmarks.

Key words: mTOR; Alzheimer's disease; Rapamycin; Macroautophagy; Apoptosis; Cognition.

Katarzyna Stachowicz

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

Looking for new antidepressants - interactions between COX-2 and glutamatergic receptors

Depression is one of the main mental disorders requiring hospitalization, fraught with the incidence of suicide attempts. Statistics indicate that depression is twice as common in women as in men; it affects middle-aged people, but also the elderly and children and adolescents. Recent literature shows a significant increase in the incidence of depression. It is important to search for new mechanisms of depression in order to synthesize drugs. My search has focused on interactions between cyclooxygenase-2 (COX-2) and metabotropic glutamate receptors (mGluRs). About this very research I would like to tell you. Glutamate (Glu, glutamic acid) is the main excitatory neurotransmitter in the mammalian brain, which, together with the inhibitory gamma-amino-butyric acid (GABA), is responsible for maintaining normal functional homeostasis in the brain. Dysregulation of Glu neurotransmission is observed in depression, anxiety, and cognitive dysfunction, among others. The distribution of neurons, pathways, and glutamatergic endings in the brain is very well understood and described. Glu is the main neurotransmitter of the pyramidal neurons of the new cortex, from where it forms projections to the most essential brain structures (such as the striatum, amygdala, thalamus), and to the spinal cord. Glu receptors are divided into ionotropic, and metabotropic receptors. In turn, one of the main building components of neural tissue are polyunsaturated fatty acids of the omega- 6 (□-6), and □-3 types. The main fatty acid of the □-6 pathway is arachidonic acid (AA), which is released from membrane phospholipids under the influence of phospholipase A2 (PLA2). The □-6 pathway is known as the inflammatory pathway, or the pro-inflammatory cytokine pathway. AA released from cell membranes is metabolized to prostanoids (PG) under the influence of cyclic prostaglandin hydroperoxide synthase (PGHS), called cyclooxygenase. How these two components regulate each other affecting the mood I will tell in the lecture.

Audience Take Away Notes

- The lecture will provide an understanding of the basic mechanisms of brain function and bring awareness to the importance of neurotransmission pathways such as Glu and COX-2 in mood changes
- The knowledge gained will allow the introduction of new research techniques in laboratories and will allow to establish cooperation
- Understanding what modulation of excitatory transmission involves will allow for new hypotheses to synthesize new, more effective drugs for depression

Biography

Dr. Katarzyna Stachowicz's research interests focus on the molecular mechanisms of depression, cognition and anxiety. Her doctoral dissertation at the Department of Neurobiology, Institute of Pharmacology, Polish Academy of Sciences, Krakow, Poland, focused on molecular mechanisms of anxiety involving mGluRs. Dr. was a postdoctoral fellow at the Center for Neuroscience Drug Discovery, Vanderbilt University Medical Center, Nashville, TN, USA. Dr. is an assistant professor, his postdoctoral dissertation focused on COX-2/mGluRs interactions in depression. Dr. is also a pop scientist who seeks to combine neuroscience and physics to talk about the underlying molecular mechanisms in the brain. She has published more than 50 research articles in SCI journals and fourteen popular science articles.

Esra Demir Unal
Department of Neurology, Nevşehir State Hospital, Nevşehir, Turkey

Evaluation of anterior versus posterior circulation acute ischemic stroke with clinico-topographic correlations and relationship with early mortality-based scale predictions

Background: Posterior circulation ischaemic strokes (PCIs) are a clinical syndrome associated with ischemia or embolic occlusion of posterior circulation and differ from anterior circulation ischaemic strokes (ACIs) in many aspects. In this study, ACIs and PCIs were evaluated in terms of clinico-radiological and demographic aspects, and the relevance of objective scales to early disability and mortality was investigated.

Methods: 100 acute ischemic stroke patients were included in the first 24 hours. The definition of ACIS or PCIS was classified according to the Oxfordshire Community Stroke Project (OCSP). Arrival NIH Stroke Scale/Score (NIHSS) and Glasgow Coma Scale (GCS) and Modified SOAR Score (mSOAR) were evaluated for early clinical scores and mortality-based scale. All data were compared, and mean, median (IQR) values and ROC curve analysis were determined.

Results: Hypertension was the most common disease for both groups. The second one was hyperlipidemia (82%) in the ACIs and diabetes mellitus (40%) in the PCIs. The frequency of right hemisphere ischemia was higher in ACIs (63.6%) and PCIs (48%). The mean NIHSS and GCS score (also median IQR) were higher in the right ACIs (median (IQR):9.5(13). The highest NIHSS mean was in the right PACS (median (IQR):14.5(3)). The mean NIHSS and GCS score of bilateral POCs was the highest in PCIs (median (IQR):3(17), median (IQR):15(4) respectively). The mSOAR mean was the highest in the right PACs and bilateral POCs (median (IQR):2.5(2), (median (IQR):2(2) respectively).

Conclusion: The association of PCIs with hyperlipidemia and the male gender was interpreted, and anterior infarcts were found to cause higher early clinical disability scores. The NIHSS scale was effective and reliable, especially in acute anterior strokes, but also emphasized the necessity of using the GCS assessment together in the first 24 hours to assess PCIs. MSOAR scale is a helpful predictor in estimating mortality not only in ACIs but also in PCIs, similar to GCS.

Keywords: Modified SOAR Score for Stroke, NIH Stroke Scale, Posterior Circulation Brain Infarction, Topographic Imaging.

Audience Take Away Notes

- Acute Ischemic Stroke is an acute neurological pathology that ranks first in life-threatening mortality
- Clinic-topographic degelrendrime is essential in disease prognosis and treatment selection in AIS
- PCIs differ from AISs in terms of clinical, topographical, and mortality
- NIHSS is one of the parameters that can be used safely in anterior strokes
- When evaluating PCIs, the GCS and mSOARo scales should be evaluated together with the NIHSS

Biography

Dr. Esra the DEMIR UNAL studied Medical Faculty at the Ataturk University, Turkey, and graduated as M.D in 2016. She then joined the Neurology Department at the Institute of Yıldırım Beyazıt University Medical Faculty, Turkey. She received her Neurology Specialist degree in 2021 at the same institution. She is working as the Chief Department of the Neurology Clinic at Nevşehir State Hospital. Currently, she continues her thrombolytic treatment and practices at Nevşehir State Hospital Acute Stroke Clinic. She also carries out studies on invasive interventions for Parkinson's and Movement Disorders and is currently the director of the Apomorphine Treatment and Development Strategies Clinicfor Moderate- Advanced Parkinson's Patients.

João Rafael Rocha da Silva

Physiotherapy sector coordinator, Connect Life Rehabilitation and Performance, Ubatuba, São Paulo, Brazil

Manual therapy and segmental stabilization in the treatment of chronic pain

Pain is one of the most frequent complaints of patients, being responsible for causing damage to the public health system, disability, and loss of functionality to patients.

Understanding the different levels and types of Pain and its possible musculoskeletal dysfunctions is essential for health professionals, giving them a better capacity for dysfunctional diagnosis.

The use of manual therapy techniques and segmental stabilization are resources used worldwide in the treatment of pain by physiotherapists, but the lack of specificity of the techniques as well as the technical knowledge of the professionals is essential to achieve good results in clinical practice.

What is observed is an existing barrier between the application of the technique and knowledge about pain, in this way patients may not achieve the desired results.

In my two studies published this year, it can be observed that when applied specifically, using evidence-based practice, we can obtain good results in the clinical improvement of Pain.

New studies are providing a better understanding of the physiological processes that occur in these individuals after the application of these techniques, changing the perspective of professionals to a more in-depth look at changes in the central nervous system instead of seeking only musculoskeletal tissue explanations.

This conceptual change opens up a new range for new studies and may change the clinical management of these patients.

Audience Take Away Notes

- Attendees will be able to increase their knowledge of Pain assessment and treatment
- Will improve pain management.
- It will open up the possibility of a new direction for research that addresses this topic.
- Knowing the high incidence of these patients in clinical practice, improving the management of these patients can be a differential for professionals
- Certainly
- List of other benefits

Biography

Physical Therapist João Rafael Rocha da Silva, Postgraduate Degree in Sports Rehabilitation Sports Orthopedics and Traumatology CETE Federal University of São Paulo, Improvement in Pain Assessment and Interdisciplinary Treatment Hospital das Clinicas, USP Medical School.

Published two works in 2022 Manual Therapy in the Treatment of Pain Revista Neuro Ciencias and Assessment of the Transversus Abdominal Muscle in Individuals with Pain Med Crave Neurology Journal, Scientific Reviewer.

Alex Goraltchouk[1*], Svetlana Mankovskaya[2], Kuznetsova Tatjana[2], Hladkova Zhanna[2], Francesco Luppino[1], Alexey Seregin[1]

[1]Remedium Bio, Inc., Needham, MA, USA
2Institute of Physiology, National Academy of Sciences of Belarus; 28 Academicheskaya St, Minsk

rhFGF18 and rhGDF11 for the treatment of ischemic stroke

Objective: The goal of our research was to objectively evaluate the safety and efficacy potential of two investigational treatment regimens using rhFGF18 and rhGDF11 for the treatment of ischemic stroke.

Background: Despite recent advances in prevention and treatment, stroke remains the second-leading cause of death and third-leading cause of disability worldwide. Currently, thrombolytic tissue plasminogen activator remains the only therapeutic treatment with established efficacy, despite its associated risk of haemorrhage and limited effect on survival. Recent studies using growth factor derived therapeutics, including rhGDF11 and rhFGF18 have shown promise in early preclinical models, however, their mechanisms remain poorly understood and a head-to-head comparison has never been performed.

Methods: Cerebral ischemia was induced in 250-300g m/f Wistar rats via middle cerebral artery occlusion (MCAO) (2 hours, Day 1). PBS and 100ug/kg/hr rhFGF18 were administered intravenously (0.5mL/hour, 3 hours); rhGDF11 was administered intraperitoneally (5-daily, 100ug/kg injections in 100uL PBS, starting Day 7 following MCAO). Morris water maze was used to assess motor-cognitive recovery on Days 7, 21, and 42. Tissue sections were collected on Days 21 and 42 to assess markers of neurodegeneration (Nissl stain), acetylcholinesterase (AChE) fibre density and activity, as well as metabolic parameters including succinate dehydrogenase and lactate dehydrogenase as markers of oxidative phosphorylation and glycolysis.

Results: By Day 7, the water maze times in the PBS control group, rhFGF18, and rhGDF11 increased by 38.3%, 2.1%, and 23.1% relative to pre-MCAO baseline, with rhFGF18 achieving statistical significance over PBS. Fraction of neurons with abnormal morphology decreased in all groups toward Day 42 and was lowest for rhFGF18, while gliosis improved in all test groups. AChE-positive fibre density appeared to increase over time in rhFGF18 treated animals, remained unchanged for rhGDF11, and declined in the PBS control-treated animals. Parallel changes were observed in the level of AChE enzymatic activity. Metabolic increases were greatest in rhGDF11 treated animals, as evidenced by an increase in SDH and LDH activity toward Day 42, with both rhFGF18 and rhGDF11 achieving statistically significant improvements over PBS. Finally, rhFGF18 appeared to promote a trend for reduced mortality relative to PBS, with post-MCAO mortality rates of 5.6% (95% CI [27.3%, 0.1%]) and 22.2% (95% CI [47.6%, 6.4%]) respectively.

Conclusions: Our findings suggest that early intervention with rhFGF18 appears cerebroprotective with potential benefits in survival, recovery of motor and memory function, as well as neuronal viability and metabolic activity.

Audience Take Away Notes

- The audience will learn about the efficacy potential of two currently investigational treatments for one of the largest unmet needs in neurology – ischemic stroke
- The audience will be able to expand their knowledge on the novel treatments and their potential mechanisms of activity in promoting neuroprotection and neuroregeneration
- The scientific community will be afforded a technical foundation for future mechanistic, safety, and efficacy studies of rhFGF18 and rhGDF11 in cerebral ischemia

Biography

Alex is the Chief Operating Officer of Remedium Bio, a Boston-area biotechnology company. Prior to Remedium Alex held roles of increasing responsibilities at companies including Allergan, Biogen, and Regeneron. His efforts were instrumental in bringing to market a number of global blockbuster therapies for the treatment of Multiple Sclerosis, Rheumatoid Arthritis, and Atopic Dermatitis. Alex holds a Bachelor in Materials Engineering and a Master in Chemical Engineering from the University of Toronto, MBA and Master of Science in Finance from Indiana University, and a Master in Microbiology and Cell Sciences from the University of Florida.

Jihen Souissi[2], Sourour Karmani[2] and Ridha Djemal[1]*

[1]Electronic Engineering Department ISSAT, Sousse Tunisia
[2]ATMS Laboratory, ENIS Sfax, Tunisia

Enhancing epilepsy diagnosis using automated temporal windowing for frequencies and entropies feature extraction computing

The diagnosis of epilepsy relies on careful analysis of Electroencephalograph (EEG) records to understand the underlying mechanisms of epileptic disorders. Due to noise interference, filtering is necessary before applying feature extraction and classification. Traditional frequency analysis methods have limitations in diagnosis, thus this study proposes combining statistical and artificial intelligence techniques. A Butterworth filter of order 4 with ICA is applied to eliminate noise from eye blinking and muscle activities. Feature extraction involves Temporal Windowing (TW) for frequency features (amplitude, phase, spectral density) and statistical measures like Shannon entropy and Spectral entropy. Five parallel classification techniques (KNN, SVM, Random Forest, LDA, MLPC) are used, with an average accuracy estimated for epilepsy diagnosis as depicted in the following Figure. By identifying EEG features, this system supports physicians in the diagnostic process. Adding ICA and AR models improved accuracy by approximately 18%. This scheme, processing each channel individually, outperforms other methods with an accuracy close to 100%.

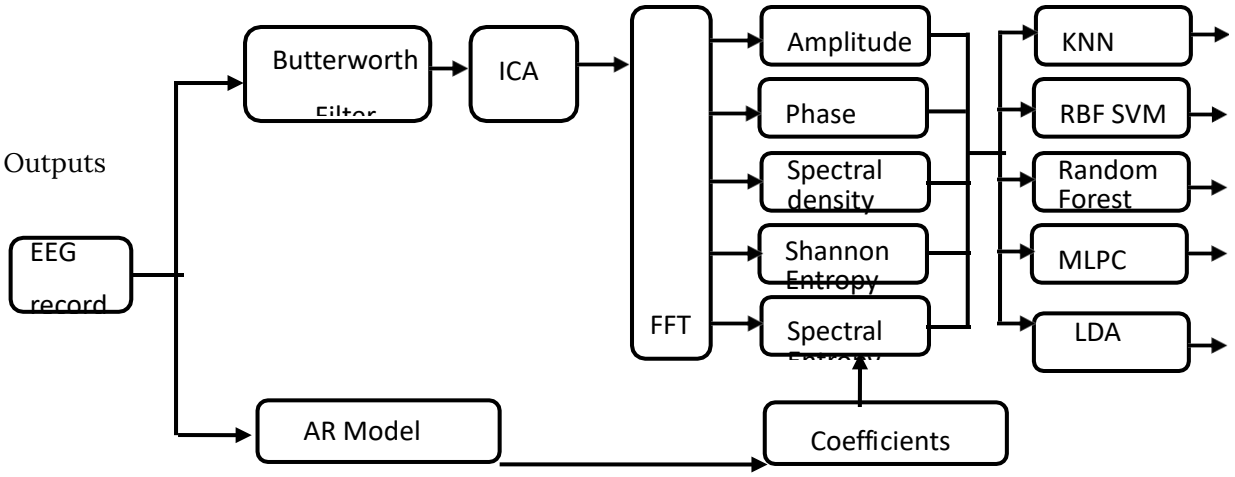
During the development of our diagnosis scheme, we implemented the following components:

1. Improved Preprocessing Technique: To eliminate unwanted artifacts such as eye blinking, muscular movement, and other interference noises, we enhanced the preprocessing step by incorporating Independent Component Analysis (ICA) into the IIR filter.

2. Temporal Windowing Approach: We applied the temporal windowing approach with an optimal window size of 0.3 seconds. This allowed us to capture relevant temporal variations, maximizing our model's performance in feature extraction.

3. Combination of Frequencies and Statistical/Nonlinear Features: By combining frequency- based features with statistical and nonlinear features, we obtained valuable insights into the complexity of EEG signals. This information proved relevant for the detection of epileptic anomalies. Additionally, we introduced the autoregressive model (AR), which captures temporal dynamics, models complexity, adapts to different orders, and facilitates prediction and anomaly detection. These advantages contributed to more accurate and informative feature extraction, significantly improving the effectiveness of epilepsy diagnosis.

4. Parallel Classification Techniques: We experimented with five classification techniques in parallel to demonstrate their similar accuracy. This outcome is a result of the optimization achieved in the preprocessing and feature extraction stages.



The following table presents accuracy estimations obtained using the Bonn Data Set, consisting of five labeled sets (SetA, SetB, SetC, SetD, and SetE). Each set comprises 100 segments of monopolar EEG data, with a duration of 23.6 seconds, recorded at a sampling rate of 173.6 Hz.

Biography

Without ICA and AR		With ICA and AR	
Classifier	Accuracy (%)	Classifier	Accuracy
Nearest Neighbors (KNN)	80.8	Nearest Neighbors (KNN)	99.3
RBF SVM	87.6	RBF SVM	100
Random Forest	88.5	Random Forest	100
MLPClassifier	87.9	MLPClassifier	99.8
LDA	84.7	LDA	99.4

Biography

Dr. Ridha Djemal, a full professor in Electronics and Embedded System Design at ISSAT in Sousse, Tunisia, earned his PhD in Electronics from INP Grenoble, France, in 1996. He is an active member of the ATMS Laboratory at ENIS, Sfax University. His research focuses on integrating artificial intelligence techniques with EEG signal analysis for Motor imagery and the diagnosis of medical pathologies such as Autism, Epilepsy, Alzheimer's, and Apnea. With active involvement in numerous research projects, Dr. Djemal's contributions advance the field of EEG signal analysis in medical diagnostics.

Lloyd L. Tran PhD, Markku Kurkinen PhD, Zung V. Tran* PhD
Biomed Industries, Inc., San Jose, California, USA

What's Leqembi got to do with Alzheimer dementia

Lecanemab is the humanized IgG1 version of the mouse monoclonal antibody mAb158, which selectively binds to large, soluble Aβ protofibrils [1].

On January 6, 2023, Lecanemab (Leqembi) was approved by the FDA under an accelerated approval pathway based on the observed reduction of amyloid beta plaque quantified using positron emission tomography (PET) imaging. FDA concluded that "the reduction in plaques is reasonably likely to result in clinical benefits"

On June 9, the FDA's Peripheral and Central Nervous System Drugs Advisory Committee of six members endorsed 6-0 the full approval of lecanemab, which the FDA is expected to announce by July 6. The purpose of this talk is to demonstrate the clinical trial data pub- lished by van Dyck et al, 2023 show no evidence for lecanemab's efficacy in slowing cog- nitive decline.

Lack of Proof of Efficacy: The primary endpoint was the change from baseline in the Clinical Dementia Rating - Sum of Boxes (CDR-SB) at 18 months of treatment. CDR-SB measures cognition and function on a 0-18 points scale, higher scores indicating worse performance. In the trial of 1795 indi- viduals, CDR-SB score increased from the 3.2 baseline to 4.41 in 18 months in the lecanemab group, a change of 1.21, and to 4.86 in the placebo group, a change of 1.65, cal- culated as "adjusted least-squares mean change". The - 0.45 difference (4.41 - 4.86) be- tween the groups is often interpreted as 27% less cognitive decline (0.45/1.65) as the clini- cal benefit of lecanemab treatment. This is misleading and an erroneous conclusion. What matters in real world at the end of the trial are the CDR-SB scores in the lecanemab and pla- cebo groups, which give lecanemab a clinical benefit of 9.3% (0.45/4.86), which is unlikely to make any difference for people living with early AD.

Safety Issues: Overall, in Study 301 Core program and open label extension (OLE) the incidence of death was 6.9/1000 person years or 16 deaths amongst 2331 participants in the clinical trials of lecanemab.

The main safety problem associated with lecanemab, is amyloid related imaging abnormali- ties (ARIA), cerebral hemorrhage, and infusion-related reactions and hypersensitivity.

ARIA is classified as ARIA with edema (ARIA-E) and ARIA with hemosiderin deposition (ARIA-H), which includes microhemorrhage and superficial siderosis. ARIA-H generally occurs in association with an occurrence of ARIA-E. The presence of the ApoE E ε4 allele increases the risk of ARIA, with greater risk observed in homozygotes than heterozygotes.

The use of antithrombotic medication, particularly, anticoagulation therapy, may increase the risk for cerebral hemorrhage in patients taking lecanemab.

These risks need to be considered in the benefit-risk discussion between prescribers and pa- tients/ caregivers when making the decision to initiate therapy.

Costs and Reimbursement: On June 1, 2023 Centers for Medicare & Medicaid Services (Medicare) outlined plans to broadly cover a new class of Alzheimer's drugs indicating the drugs must have traditional approval

from the Food and Drug Administration. In addition, physicians must participate in registries designed to collect information on how the drugs work in the real world, as op- posed to the tightly controlled environment of clinical trials. We believe patient registries would not hinder the accessibility of the drug and support the approach from CMS. The col- lection of information and bioanalytic data will benefit CMS, the drug companies, and other stakeholders to validate the efficacy and safety of lecanemab post marketing approval.

According to Kaiser Family Foundation, if 5% of the 6.7 million older adults in the US with Alzheimer's disease take Leqembi, at the annual list price of $26,500, this would add $8.9 billion to Medicare Part B spending annually (assuming all are enrolled in Part B). If just 10 percent of the 6.7 million older adults with Alzheimer's disease take Leqembi, the has esti- mated that it would cost $17.8 billion—or nearly half of what Medicare Part B spent on all drugs in 2021.

Conclusion: The development of aducanumab and lecanemab was guided by the amyloid hypothesis, which proposes Alzheimer's disease begins in the brain with the accumulation, aggregation, and amyloid formation. For 30 years, the amyloid hypothesis has almost singularly guided biomedical research, drug discovery, and clinical development in Alzheimer's disease. The biomarker data of aducanumab showed reduction of amyloid beta plaque, but no evidence was presented to correlate biomarker changes to cognitive benefits.

The FDA approval of aducanumab and lecanemab is not a proof of the validity of the amy- loid hypothesis. The amyloid hypothesis is misleading and too good to be true.

The accelerated approval of lecanemab was based on the observed reduction of amyloid beta plaque. Dr. Alois Alzheimer never suggested plaques and tangles were the cause of de- mentia. Based on the clinical data of lecanemab submitted to the FDA, lecanemab has not demonstrated a proof of efficacy and safety to warrant the full FDA approval.

We believe that people with Alzheimer's disease deserve a drug that is safe, effective, and affordable.

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POSTERS

JOINT EVENT ON

NEUROLOGY AND DEMENTIA

Byung Hee Han*, Min Chan Shin, Jinu Jang, Eun Ji Kim, Sung Min Kang

Kai-i Company, Seoul, South Korea

The effectiveness of a mobile application for early detection of Alzheimer's disease using the Korean dementia screening questionnaire

Background: Early detection of dementia is very important for improving the quality of life of patients and their families and providing appropriate medical interventions. The Korean Dementia Screening Questionnaire (KDSQ) was developed by Dong Won Yang et al. in 2002 and is used to screen for dementia and mild cognitive impairment in the elderly. KDSQ is a self-assessment questionnaire consisting of 25 items in 5 parts, each evaluating memory impairment, behavioral impairment, daily living impairment, vascular impairment, and depressive impairment. KDSQ showed excellent performance with a sensitivity of 79% and a specificity of 80% in a study of Koreans. In this study, we selected and investigated items related to Alzheimer's disease (AD) from the KDSQ questionnaire, and we developed a mobile application that allows users to self-assess AD more easily and quickly.

Methods: A mobile application was developed using a simple KDSQ that consists of 10 items related to Alzheimer's disease (AD) from the KDSQ. The application is embedded with a deep neural network (DNN) classifier that classifies users into normal and suspect groups and recommends hospital visits for suspects. To evaluate the effectiveness of the application, AD screening was conducted using the developed application for 939 elderly people at the Daedeok Senior Welfare Center (DSWC, Daejeon, South Korea) and 973 visitors at the Gyeonggi Provincial Medical Center Icheon Hospital (GPMC-IH, Gyeonggi, South Korea).

Results: The results showed that 14.9% of the elderly people at the DSWC and 36.2% of the visitors to the GPMC-IH were classified as suspected AD groups and recommended to visit the hospital. These rates of suspected AD groups were higher than the current dementia prevalence rate of 10% for Korean elderly people over the age of 65. In the case of the welfare center, the average age was 78.9 years old, so the suspected AD group was judged to be higher due to age factors. In the case of hospital visitors, the results were higher because they were the results of a survey of elderly people who suspected dementia and responded to the survey. Those who were classified as suspected AD groups were recommended to visit the hospital for additional treatment, and those who were classified as suspected AD groups among the Icheon Hospital visitors were linked to hospital treatment. Of the suspected AD group at Icheon Hospital, 40.9% received additional treatment from the hospital.

Conclusions: The sensitivity and specificity of the application can be calculated by medically confirming the dementia of people who received early dementia screening through this application. The rate of suspected AD groups judged in the application was higher than the current dementia prevalence rate of Korean elderly people, so the AD screening sensitivity of this application is expected to be high. Therefore, it is thought that this application can be used effectively as a tool for early self-administered AD screening. In conclusion, we have developed a mobile application that can check AD early through a simple KDSQ self-questionnaire, guides hospital visits for suspected AD groups, and provides dementia management and prevention education content. This application is expected to be useful for early diagnosis of dementia. In the future, we will study additionally to calculate the sensitivity and specificity of dementia screening

using the mobile application and to evaluate its effectiveness. This study was supported by the Korea Ministry of SMEs and Startups (S3046273).

Audience Take Away Notes

- Self-AD early screening method using a mobile application
- The AI classification of dementia severe level using KDSQ items
- How to present services to users in a dementia early screening application

Biography

Dr. Byung Hee Han received his Ph.D. in Biomedical Engineering from Kyung Hee University, Korea, in 2003. From 2004 to 2011, he worked as a research professor at the same university, where he studied medical imaging systems. He worked at Electronics and Telecommunications Research Institute (ETRI) from 2012 to 2022, where he developed technologies to solve the problems of small and medium-sized enterprises. He currently works at Kai-i Company, where he conducts research on the application of artificial intelligence technology to dentistry, dementia screening, disability, and infant care.

Lori A Gray, PhD
Western Michigan University

Mindfulness-based protocol for recovery from stroke (MBRfS): A pilot study

Decades of research suggest that Mindfulness-Based Stress Reduction (MBSR) training supports a greater capacity to live with chronic medical conditions and contributes to lowering stress levels. This paper introduces a model for a Mindfulness-Based Recovery from Stroke (MBRfS) for promoting stroke recovery, informed by the lived experience of the author (a stroke survivor and certified MBSR instructor), the research literature regarding MBSR training, and the specific challenges of stroke recovery. A literature search was conducted to explore possible research and publications adapting M.B.S.R. to medical populations, including stroke survivors. There is very limited research in this area. A summary will be presented of similar protocols, such as applying M.B.S.R. in programs for cancer survivors and chronic pain patients. An autoethnography was developed as a methodology to support to the model. A theoretical case is made for the value and need for the development of a mindfulness-based program for those who have experienced and are recovering from a stroke. The proposed mindfulness-based stroke recovery intervention (MBRfS) is outlined in detail, informed by similar evidence-based programs. Case vignettes are offered in the autoethnography that emerged, drawing on the authors experiences as a stroke survivor and MBSR certified teacher. A four-component MBRfS model is offered, which consists of integration amongst a modified MBSR framework, emergent attitudinal themes, and insights from the autoethnographic vignettes. The MBRfS model offers a path for providing participants with a supportive experience within stroke recovery. Recommendations and suggestions for future studies are offered to support the development of MBRfS for stroke survivors and their caregivers, as well as contributing to healthcare providers.

Audience Take Away Notes

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

Biography

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

Dandan Lv*, An Liu, Wei Xie

The Key Laboratory of Developmental Genes and Human Disease, Ministry of Education, School of Life Science and Technology, Southeast University, Nanjing, Jiangsu, China

The mechanism of NLG1 regulating ASD-like (restricted and repetitive) behaviors

Objective Autism spectrum disorder (ASD) is a common neurodevelopmental disorder with two core symptoms, namely repetitive stereotyped behaviors and impairments in social interaction. Neuroligin1 (NLG1) is one of synaptic adhesion molecules, which is a high-risk molecule found in individuals with autism. Previous research has shown that NLG1 knockout mice shown excessive self-grooming behavior and deficits in social ability and memory. The lesions of the striatal-basal ganglia circuit are also the key targets for the pathogenesis of many diseases such as Parkinson's disease, Huntington's disease, and autism. Our project aims to investigate how NLG1 in the striatum regulates repetitive stereotyped behavior investigate, which may provide clinical diagnosis and treatment for autistic patients.

Methods The expression of NLG1 in the striatum was specifically manipulated by different types of viral tools, and behavioral tests were performed 5-6 weeks later. The activity of neurons was detected by in vivo calcium imaging and electrophysiology. Rescue experiments were carried out by optogenetic, DREADDs, and pharmacological methods.

Results (1) NLG1 decrease in striatum promotes restricted and repetitive behaviors. (2) Knocking down NLG1 in dorsal D2-MSN (medium spiny neuron) of striatum induces restricted and repetitive behaviors. (3) Adult restoration of NLG1 expression in D2-MSN of NLG1 knockout mice rescues repetitive and restricted behaviors. (4) The loss of NLG1 in D2-MSN induces an increase in neuronal excitability. (5) Dorsal D2-MSN is excessive activated in NLG1 KO mice, thus inhibiting D2-MSN activity can recue restricted and repetitive behaviors.

Conclusion The restricted and repetitive behaviors of NLG1-deficient mice are caused by excessive activation of dorsal D2-MSN.

Audience Take Away Notes

- Understand current techniques used in basic neurobiology research, including in vivo calcium, optogenetic, DREADDs, and pharmacological
- Be familiar with the current status of autism, including prevalence, risk factors, and treatment options
- Help the audience better understand the heterogeneity and particularity of striatum brain region, which is an important region of ASD patients
- Know the role of NLG1 in autism, and how to design drugs to treat behavioral deficits associated with ASD by the molecular mechanism based on NLG1

Biography

Dandan LV received the B.Eng. degree in biological engineering from the Nantong University, Nantong, China, in 2017. From 2017 to now, she is currently pursuing the MD-PhD degree in neurobiology with the School of Life Science and Technology, Southeast University, Nanjing, Jiangsu, China. Her current research interest includes repetitive and restricted behaviors and molecular mechanisms of autism, molecular mechanisms of synaptic development and synaptic plasticity.

Participants List

Akankunda Veronicah Karuhanga Golden Age Elderly Homes, Uganda	46
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Jun Hua Johns Hopkins University School of Medicine, United States	106
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We wish to meet you again at our upcoming events next year...

Questions? Contact

+1 (702) 988-2320 or
secretary@magnusconference.com