

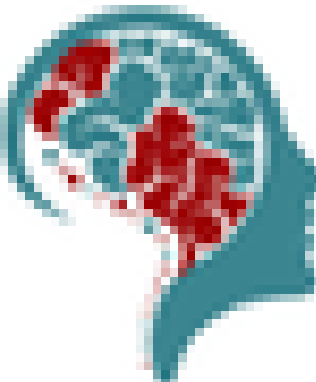


International Conference

Advancing Research, Enhancing Lives



July 09-10, 2026
Frankfurt, Germany

 **Neuro - 2026**
Frankfurt, Germany

ISBN: 978-1-917892-37-7

 **WCAG-2026**
July 09-10, 2026 | Germany

ISBN: 978-1-917892-36-0

 **Skin Summit-2026**
July 9-10, 2026 | Frankfurt, Germany

ISBN: 978-1-917892-38-4



LETTER FROM THE CEO & DIRECTOR

On behalf of United Research Forum, UK we are pleased and delighted to welcome the organization of the International Conference at Hotel NH Frankfurt Airport West, Frankfurt, Germany from 09 to 10 July, 2026.

Our goal is to providing an international forum where international scientists meet with scientists from the different fields of health care sectors to exchange ideas and information on current trends of research results and practical experiences. We are looking forward to welcoming all of you in Germany and to have with you an excellent meeting with great scientists from different countries around the world and sharing new and exciting results in the different subjects of the conference.

We hope that you will enjoy your stay in Germany and advice you to take this opportunity to visit the beautiful historical sites that you can find everywhere in the country. Finally, I would like to add my best wishes for a successful and fruitful conference and many thanks to our partners the organizers of this Conference.



Sincerely,
Dr. Vanga

Director, URF, UK

Exhibitors

Naason Science. Inc



Naason Science Inc.,

South Korea

Naason Science Inc. is a leading preclinical Contract Research Organization (CRO) based in South Korea, specializing in drug discovery and preclinical research services. Established in 2017, the company provides advanced research platforms and customized preclinical models that support pharmaceutical, biotechnology, and academic researchers in the development of innovative therapeutics.

Preclinical Modelling Platforms for Gynecologic, Endocrine, and Inflammatory Diseases are among the company's key areas of expertise, enabling researchers to evaluate novel therapeutic approaches through advanced disease-specific models.

With expertise in efficacy evaluation, disease modeling, and translational research, Naason Science offers integrated solutions that accelerate drug development from early discovery to preclinical validation. The company is committed to scientific excellence, innovation, and global collaboration in advancing healthcare and biomedical research.

Scientific Program

International Conference

Frankfurt, Germany
July 09, 2026

Day-1 (July 09, 2026)

09:00-09:30 Registrations & Opening Remarks

Plenary Forum

09:30-10:00

Inaugural Address by

Thomas Klaholz, University of Bonn, Germany



10:00-10:20 Networking Break

10:20-10:40 Group Photo

Keynote Talk

10:40-11:00

Title: Your Way to Longevity - the 7 Secrets of Healthy Aging

Thomas Klaholz, University of Bonn, Germany

Technical Session-I (10:40 - 14:00)

Session Chairs:

Dr. Barbara L. Parry,
University of California San Diego, USA

Prof. Maria Nicoleta Turliuc,
Alexandru Ioan Cuza University, Romania

11:00-11:20



Title: Technical assistance system for monitoring mobility and nutrition of older adults for preventive care and healthy aging: A pilot study
Mareike Forster, Carl von Ossietzky University of Oldenburg, Germany

11:20-11:40

Title: Better and Healthier Together? The Mediation Effect of Positive Psychological Capital on the Relationship Between Perceived Social Support and Health-Related Quality of Life Among Older Adults



Maria Nicoleta Turliuc, Alexandru Ioan Cuza University, Romania

11:40-12:00



Title: RIPK2 expression in the skin of atopic dermatitis May define Th1/Th2 cytokine profile
Elias Toubi, The holy family Hospital, Israel

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12:00-12:20

Title: Revealing Healthy Aging: AI-Derived Facial Biomarkers for Early Detection and Patient-Centered Preventive Longevity Medicine

Rainer Pooth, Clinical Research ICAAN, Germany



12:20-12:40

Title: CD4+CCR5+ T cells and CCL3+ mast cells are increased in the skin of patients with chronic spontaneous urticaria

Prof. Zahava Vadasz, Bnai-Zion Medical Center, Israel

12:40-13:00

Title: Percentiles of neuromotor functions from 6 to 80 years

Kakebeeke Tanja, University Children's Hospital Zurich, Switzerland



13:00 - 14:00 Lunch @ Restaurant

Technical Session-II (14:00 - 17:30)

14:00-14:20

Title: The prolyl oligopeptidase and α -synuclein connection: context, insights and hypotheses

Anne-Marie Lambeir, University of Antwerp, Belgium





14:20-14:40

Title: Integrated Active Aging Index (IAAI): From Individual-Level Quantification to Community-Based Active Aging Intelligence

Chanvit Tharathep, Thai Biomedical Engineering Association, Thailand

14:40-15:00

Title: Targeting alpha-synuclein with the lipoxygenase inhibitor PTC-041 as a therapeutic approach for the treatment of Parkinson's disease

Angela Minnella, PTC Therapeutics, Inc, USA



15:00-15:20

Title: POMGnT1 Deficiency Promotes Tau Hyperphosphorylation and Cognitive Impairment in Alzheimer's Disease

Xiaofeng Li, The second affiliated hospital of Chongqing Medical University, China



15:20-15:40

Title: Evaluation of resensibilization in flaps containing sensory nerves in the animal model: A systematic review of the literature

Patricia Esther Engels, University Hospitals of Geneva, Switzerland



15:40-16:00 Poster Presentations + Refreshment Break

Poster-1



Title: Acid-Base Dysregulation Links Aging Metabolism to Frailty

Wan-Hui, MacKay Memorial Hospital, Taiwan

Poster-2

Title: Postoperative Course Following Excision Surgery and Biopsy: A Retrospective Cohort Study In The Dermatology Outpatient Department of A Tertiary Hospital In The Philippines

Nicole Therese Mariano Mendiola, Jose R. Reyes Memorial Medical Center, Philippines



Poster-3



Title: Evaluation of Circadian Rhythm Disruption in the DHT-Induced Rat Model of PCOS, Exploratory Pharmacological Modulation with Niclosamide

Dr. Kamalakannan Radhakrishnan, Naason Science Inc. KBIO New Drug Development Center, South Korea

Poster-4

Title: Development of a Dichoptic Chromatic Pupillometer

Dr. Sébastien Buchwalder, Oculox Technologies, Switzerland



Poster-5



Title: The Alleged Harm of Chocolate Consumption on Teeth: A Myth or Reality

Dr. Behzod Shavkatxojayev, Central Asian University, Uzbekistan

Poster-6

Title: Jalili Syndrome: The Rare retinopathy with Amelogenesis Imperfecta, Clinical Insights, novel variant and genetic Perspectives in four patients

Dr. Nehal Hassib, National Research Centre, Egypt



Poster-7

Title: An Analysis of Frontal Sinusitis in three Ancient Populations from the Turpan Basin, Xinjiang, China

Dr. Qingxin Fu, Jilin University, China



Poster-8

Title: Revisiting the Paternal History of the Sherpas
Dr. Lobsang Dargye, Jilin University, China



Session Continues

16:00-16:20

Title: A prospective multi-site cohort study on the prevalence of frailty in patients aged over 70 years presenting after serious injury and implications for outcomes

Martie Botha, Flinders Medical Centre, Australia



16:20-16:40

Title: Microglia-Mediated Neuroinflammation Drives Neuropathology in Alzheimer's Disease during *P. gingivalis* Infection
Zhou Wu, Kyushu University, Japan



16:40-17:00

Title: Human immunomodulatory ligand B7-1 mediates synaptic remodeling via the p75 neurotrophin receptor
Victor Danelon, Weill Cornell University, USA



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17:00-17:20

**Title: Clinical Application of Low Temperature Plasma in Wound Healing: A
Prospective Observational Study**

Xinhua Zhang, Changzhou Institute of Technology, China



DAY 01 CONCLUDES

Scientific Program

International Conference

Frankfurt, Germany
July 10, 2026

Day-2 (July 10, 2026)

Technical Session-III (10:00 - 12:50)

SESSION CHAIRS:

Prof. Anne-Marie Lambeir,
University of Antwerp, Belgium

10:00-10:20

Title: Multiphoton Tomography: A New Technology for Testing Anti-Ageing Products
Aisada Koenig, JenLab GmbH, Germany



10:20-10:40

Title: A Sleep and Light Intervention that Shifts Melatonin Rhythms Earlier Improves Peri- and Post-Menopausal Depression: Preliminary Findings
Barbara L. Parry, University of California San Diego, USA



10:40-11:00

Title: Hereditary transthyretin-related amyloidosis is frequent in polyneuropathy and cardiomyopathy of no obvious aetiology
Volha Skrahina, RareGenetix GmbH, Germany



11:00 - 11:20 Networking Break

11:20-11:40

Title: Probiotic interventions to expand healthy human longevity
Roberto Ricardo Grau, Parque Científico de Madrid, Spain





11:40-12:00

Title: **Cardiometabolic multimorbidity in relation to the metabolic score for insulin resistance and creatinine-to-cystatin C ratio in a middle-aged and aged population**
Li Wang, Department of Geriatrics, The Second Affiliated Hospital of Chongqing Medical University, China

12:00-12:20

Title: **Mitophagy in Alzheimer's disease and its potential as a therapeutic target**
Jiahua Wei, Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University School of Medicine, China



12:20-12:40

Title: **The Hidden Hierarchy of Functional Aging: Neuromuscular Decline, Predictive Movement Systems, and the Overlooked Role of Motor Units in Longevity**
Tony Ruggia, Germany

12:40-13:00

Title: **Ultrasound and Its Effects on the Skin: Biophysical Mechanisms and Practical Applications for Supporting Skin Health**
Nataliia Koziukevych, NATA-LUX Beauty & Training, Germany



13:00-14:00 Lunch

Technical Session-IV



14:00-14:20

Title: **Pupillary responses as non-invasive biomarkers of cognitive decline in aging**
Manon Lenain, Univ. Lille, CNRS, Institut Pasteur de Lille, France



14:20-14:40

Title: Association between chemotherapy for surgically treated rectal cancer and second primary endometrial cancer

Du peng, The Second Hospital of Lanzhou University, China

14:40-15:00

Title: Making the Healthy Choice the Easy Choice

Maelle Poirel, Utrecht Longevity Society, The Netherlands



15:00-15:20

Title: Prevention of cognitive impairment by B-Vitamins and omega-3-fatty acids - state of knowledge

Uwe Till, The First Affiliated Hospital of Shandong First Medical University, Germany

15:20-15:50 Networking Break

15:50-16:10

Title: Constraint-State Neuroscience: A Dynamical Framework for Understanding Neural Vulnerability, Cognitive Decline, and Neurodegenerative Progression

Mohammad Dawood Rahimi, Freie Universtat Berlin, Germany



16:10-16:30

Title: Addressing Anti-Aging Bias and the Impact on Oral Health of Older Adults

Christie Hogue, Mouth Mirrors LLC, USA



16:30-16:50

Title: Examining the Effects Of Chronic Low Back Pain On Sleep Quality, Sleep Variables, Disability, And Quality Of Life

MUSA, Necmettin Erbakan University, Turkey

16:50-17:10

Title: Housing conditions, satisfaction with housing conditions and life satisfaction of older adults in Tanzania: a cross-sectional study

Stellah John Kilawe, Xi'an Jiaotong University, China



DAY 02 CONCLUDES

**Parallel Virtual
Scientific Program
July - 10, 2026
10:00AM CET Time**



Join Zoom Meeting

Meeting ID: 860 2775 9809

Passcode: Urf@2026

Day-2 Virtual Session (July 10, 2026)

09:50-10:10



Title: Association between chemotherapy for surgically treated rectal cancer and second primary endometrial cancer

Du peng, The Second Hospital of Lanzhou University, China

10:10-10:20

Title: Determination of the mechanical properties and in vivo healing assessment of a moxifloxacin hydrocolloid wound dressing

Ni Deng, Liangjiang Hospital of Choongqing Medical University, China



10:20-10:40



Title: POMGnT1 Deficiency Promotes Tau Hyperphosphorylation and Cognitive Impairment in Alzheimer's Disease

Xiaofeng Li, The second affiliated hospital of Chongqing Medical University, China

10:40-11:00

Title: A Pharmacogenetic Panel-Based Prediction of the Clinical Outcomes in Elderly Patients with Coronary Artery Disease

Tong Yin, Institute of Geriatrics, National Clinical Research Center for Geriatric Diseases, Second Medical Center of Chinese PLA General Hospital, China



11:00-11:20



Title: Cardiometabolic multimorbidity in relation to the metabolic score for insulin resistance and creatinine-to-cystatin C ratio in a middle-aged and aged population

Li Wang, Department of Geriatrics, The Second Affiliated Hospital of Chongqing Medical University, China

11:20-11:40

Title: A Buffering Role of Perceived Social Support and Resilience between Caregiver Burden and Perceived Stress among Informal Caregivers of Dementia Elderly Patients
Saba Hayat, Bahria University, School of Professional Psychology, Islamabad, Pakistan



Keynote Talk

11:40-12:10



Title: Telomerase in brain and neurodegeneration
Gabriele Saretzki, Newcastle University, UK

Keynote Talk

12:10-12:40

Title: The role of neurodiversity in older people
Marios Kyriazis, National Gerontology Centre, Cyprus



12:40-13:00

Title: Chr17q21.31 locus risk haplotype H1 susceptibility to Ferroptosis is mediated by the endolysosomal system
Eldem Sadikoglou, DZNE, Germany

13:00-13:20

Title: From awareness to action: How patient organizations are driving DRPLA research forward
Silvia Prades, Ataxia UK, CureDRPLA, UK



13:20-13:40

Title: Predicting Health Trajectories in Older Population: A Multi-Objective Analysis of Climate, Pollution, Life-style Risks
Alina Fedorova, Applied Quantitative Solutions for Complex Systems, Spain

13:40-14:00

Title: Sensorimotor Training Elicits Comparable Strength Gains to Resistance-Endurance Training While Improving Balance-Related Factors Associated with Fall Prevention in Older Women

Lubica Ziska Bohmerova, Faculty of Physical Education and SportComenius University
Bratislava, Slovakia



14:00-14:20



Title: Fulminant Sporadic Creutzfeldt-Jakob Disease Presenting as Rapidly Progressive Confusion: A Case Report

Dalia Salih Mohammed Salih, Walsall Healthcare NHS Trust, UK

14:20-14:40

Title: Lactoferrin and Altered Immune Response: Novel Insights into the Link Between Periodontitis and Alzheimer's disease

Hoda Tayebi Hillali, University of Santiago de Compostela, Spain



14:40-15:00



Title: Spatial Gaps in Access to Specialized Healthcare for the Aging Population: A County-Level Analysis in Poland

Agata Zołtaszek, University of Lodz, Faculty of Economics and Sociology, Poland

15:00-15:20

Title: Arousal Threshold and Sleep Stability Endotypes Under Controlled GABAergic Modulation: A Prospective Cohort Study

Nyree Penn, Nsomnia Sleep, USA





15:20-15:40

Title: Testosterone pellet surface area relates to effectiveness more than dose
Charles Virden, VitalTE Lifesciences, INC, USA

15:40-16:00

Title: Brain-specific microRNA-mediated regulation of metabolic homeostasis in *Drosophila melanogaster*
Pushpa Verma, Harvard Medical School, USA



16:00-16:20

Title: Enhancing Cognitive Functioning in Dementia Through Art Therapy
Jinnie Jeon, Adler University, Canada

16:20-16:40

Title: Model of environment dependent amyloid transformation
Irena Roterman, Jagiellonian university - Medical college, Krakow, Poland



World Congress on
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Presenter Dr. Martie Botha¹ and Anthony P Joseph², Bonnie M Liu^{3,4}, Elizabeth Wake⁵, James E Hardy³, Bhavik Patel⁶ and Sarah N Hilmer^{3,4}

¹ *Trauma Service, Royal Adelaide Hospital, Adelaide, Australia*

² *Discipline of Emergency Medicine, Faculty of Medicine and Health. University of Sydney NSW, Australia*

³ *Department of Aged Care, Royal North Shore Hospital, St Leonard NSW Australia*

⁴ *Kolling Institute, Northern Sydney Local Health District and The University of Sydney, Australia*

⁵ *Trauma Service, Gold Coast University Hospital, Southport, Queensland, Australia, School of Medicine and Dentistry, Griffith University, Gold Coast Campus, Australia*

⁶ *Trauma Service, Gold Coast University Hospital, Southport, Queensland, Australia*

A prospective multi-site cohort study on the prevalence of frailty in patients aged over 70 years presenting after serious injury and implications for outcomes

With an ageing population, trauma services are managing increasing numbers of older adults who sustain serious injuries, among whom frailty has emerged as a critical determinant of recovery and long-term outcomes. This study aimed to determine the prevalence of frailty in older trauma patients admitted to major trauma centres in Australia and to examine its association with adverse events and post-discharge outcomes. A prospective multi-site cohort study was conducted between 2018 and 2023 across three Major Trauma Services in Australia: Royal North Shore Hospital (NSW), Royal Adelaide Hospital (SA), and Gold Coast University Hospital (QLD). Patients aged ≥ 70 years with serious injury (Injury Severity Score >12 , trauma service or intensive care unit admission) were enrolled. Frailty was assessed on admission using the Reported Edmonton Frail Scale (REFS), and participants were followed for 12 months after discharge. Of 217 patients recruited, 32 (14.7%) were frail and 28 (12.9%) pre-frail. Frail patients were older (mean 81.8 vs 78.6 years), had more comorbidities, and were more frequently injured by falls (72% vs 52% in non-frail patients). Geriatrician review occurred in 56% of frail versus 21% of non-frail patients. Frailty was associated with increased confusion and infection during inpatient stay and increased dependency, confusion, readmission, and mortality at 3 months (OR 3.46, 95% CI 0.81–14.82) and 6 months (OR 14.95, 95% CI 1.30–172.11). Frailty assessment on admission may enhance prognostication, inform early geriatric involvement, and support development of frailty-specific pathways to improve outcomes in older trauma patients.

Keywords: Frailty; Older trauma patients; Major trauma; Outcomes; Geriatric trauma care

Biography

Dr Martie Botha MBChB FACEM is an Emergency Physician and Retrieval Medicine Specialist with nearly three decades of international experience across Australia, the UK, and South Africa. She is a Consultant with Flinders Medical Centre and SAAS MedSTAR, and former Trauma Service Consultant at the Royal Adelaide Hospital. Dr Botha has a strong clinical and academic interest in geriatric emergency and trauma care, with extensive experience managing frailty, delirium, and complex care in older patients. She is a Fellow of ACEM, a founding member of the GEMSEM Faculty, and serves on the South Australian Trauma Committee.

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Anne-Marie Lambeir*

University of Antwerp, Belgium

The prolyl oligopeptidase and α -synuclein connection: context, insights and hypotheses

More than a decade after the discovery that prolyl oligopeptidase (PREP) influences α -synuclein (aSyn) aggregation by a process that does not rely on its peptidase activity, the molecular basis of this phenomenon is still largely unknown. We revisit the connection between PREP and aSyn and review old and new experimental data in a contemporary setting. In vitro the interaction between PREP and aSyn is weak and transient, mostly promoting early phases of aggregation without affecting the rate of β -fibril formation. Moreover, this interaction is not dependent upon the C-terminal prolines of aSyn but is affected by PREP inhibitors and it inhibits PREPs peptidase activity. There is evidence for a binding site for peptides much longer than the usual PREP substrates. In cellular aggregation models PREP colocalizes with aSyn in the cell periphery and neurites but not with aggresomes and stress markers. The modular assembly of molecular machines and the observation that PREP's protein-protein interactions are tuneable by active site inhibitors, lead to the hypothesis that this binding site features in the crosstalk between autophagy and neuron-specific pathways involving vesicle transport and protein secretion.

Keywords: Prolyl oligopeptidase, α -synuclein, autophagy, peptidase inhibitor, Parkinson's Disease

Biography

Professor Anne-Marie Lambeir is Emerita Professor of Medical Biochemistry in the Department of Pharmaceutical Sciences at the University of Antwerp, where she has built a distinguished career in research and higher education. Her academic work centers on enzymology, with particular expertise in enzyme kinetics, structure–function relationships, and the biochemical mechanisms that regulate enzyme activity. Throughout her career, she has been dedicated to advancing pharmaceutical sciences while mentoring students and researchers in the fields of biochemistry and molecular medicine.

Professor Lambeir is widely recognized for her contributions to the study of enzymes as therapeutic targets, particularly in the context of neurodegenerative diseases and other disorders with significant unmet medical needs. Her research has enhanced the understanding of enzyme function and inhibition, supporting the development of novel drug discovery strategies. Through her extensive scientific publications, collaborative research, and commitment to education, she has made lasting contributions to biomedical science and pharmaceutical research.

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**Jiahua Wei^{1,2*}, Tiegang Xiao¹, Jialu Lyu^{1,2},
Yueshuang Zhao^{1,3}, Yang Zhang^{1,4}, Xiangyu Du^{1,3},
Ying He^{1,4}, Mengqing Zhao^{1,4}, Xiaoyi Yang^{1,3},
Yutong Yao^{1,4}, Jun Ruan⁵, Kaili Liu¹, Li Zhang⁴,
Jiangbo Zhao^{6*}, Jun Xu^{1*}, Bing Wang^{1*}**

¹*Shanghai Sixth People's Hospital Affiliated to Shanghai Jiao Tong University School of Medicine, Shanghai, 200233 China*

²*Shanghai Jiao Tong University School of Medicine, Shanghai, 200025 China*

³*Shaanxi University of Chinese Medicine, Xianyang, 712046 China*

⁴*Shanghai University of Traditional Chinese Medicine, Shanghai, 201203 China*

⁵*Shanghai Municipal Hospital of Traditional Chinese Medicine, Shanghai, 200071 China*

⁶*Department of Traditional Chinese Medicine, Jiangxi Provincial Children's Hospital, Nanchang, 330038 China*

Mitophagy in Alzheimer's disease and its potential as a therapeutic target

Accumulation of damaged mitochondria is a hallmark of age-related neurodegenerative disorders such as Alzheimer's disease (AD). Mitophagy, the selective autophagic degradation of damaged mitochondria, plays an essential role in maintaining neuronal health, and increasing evidence links its dysfunction to AD progression. This review elucidates the molecular mechanisms underlying mitophagy deficits and their interaction with amyloid- β (A β) and Tau pathology, which may create a vicious cycle of neuronal damage and death. Additionally, mitochondrial dysfunction exacerbates AD pathogenesis by activating the NLRP3 inflammasome, contributing to inflammation in neurons and microglia. Investigating the dual role of mitophagy in pathological ferroptosis further reveals its complex involvement in AD. By consolidating current insights, this study highlights mitophagy as a promising therapeutic target, emphasizing its potential role in developing novel, disease-modifying interventions for AD treatment and prevention.

Keywords: Alzheimer's disease; Mitophagy; Mitochondrial dysfunction; Neuroinflammation; Ferroptosis

Biography

I am a second-year Master's student at Shanghai Jiao Tong University School of Medicine, mentored by Dr. Bing Wang (Shanghai Sixth People's Hospital). My work centers on mitophagy and neuroinflammation in Alzheimer's disease. As co-first author, I published a narrative review in *Neurobiology of Disease*. At this meeting, I will share a concise framework and open questions from our synthesis, and I look forward to exchanging ideas and learning from this community.

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Tanja H. Kakebeeke^{a,b*}, Jon Caflisch^{a,b}, Oskar G. Jenni^{a,b,c}

^a*Child Development Center, University Children's Hospital Zurich, Zurich, Switzerland;*

^b*Children's Research Center, University Children's Hospital Zurich, Zurich, Switzerland;*

^c*University of Zurich, Zurich, Switzerland*

Percentiles of neuromotor functions from 6 to 80 years

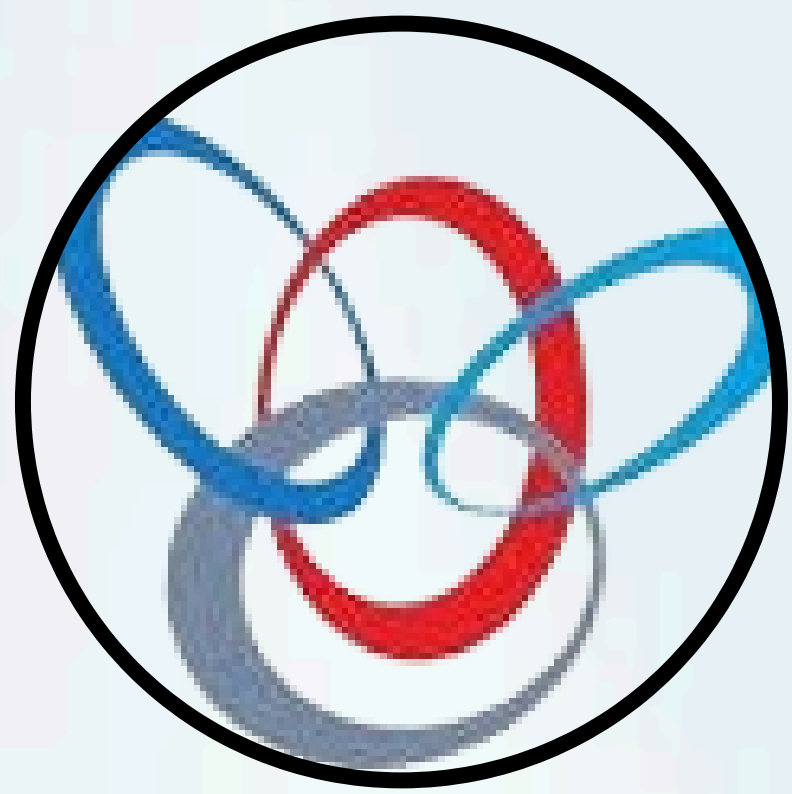
The aim of the study was to investigate the dynamics of neuromotor functions from 6 to 80 years and to provide reference curves for clinical and research use. Neuromotor data on 1620 individuals (828 females) measured with the Zurich Neuromotor Assessment between 1983 and 2023 in the region of Zurich, Switzerland were analyzed. Performance on 14 motor tasks was modeled as a function of age and sex. The age of peak performance was identified for each task. Motor performance was converted into standard deviation scores (SDSs) and combined into the 5 motor components: fine motor, pure motor, balance, gross motor, and contralateral associated movements. The effect of body mass index (BMI) was investigated. The data showed a rapid increase in motor functions during youth, particularly before age 10 years, a levelling in performance between the ages of 20 and 40 years and a decrease thereafter. The age of peak performance and the rate of decline varied greatly between the tasks: Motor functions in tasks requiring muscle force deteriorated faster than those involving isolated movements. Males reached peak performance on average one year later than females. High BMI (SDS > 1) negatively impacted balance and gross motor functions but not other neuromotor domains. Neuromotor functions undergo dynamic changes throughout the lifespan from early childhood to older adulthood, with peak performances and declines depending on type of motor task and sex. In presenting percentiles for all motor functions over the lifespan, rapid and aberrant deteriorations in degenerative diseases can be easily detected.

Keywords: motor development, aging, Zurich Neuromotor Assessment (ZNA), body mass index (BMI), lifespan

Biography

Since 1981 physiotherapist; since 1984 Bobath-instructor; since 1991 human movement scientist; since 1999 PhD in neurophysiology; since 2008 Venia Legendi; since 1984 adjunct professor in neurophysiology; currently connected to the University Children's Hospital Zurich

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Silvia Prades^{1,2*}, Andrea Compton², Julie Greenfield^{1,2} and Jeffrey B. Carroll^{2,3}

¹Ataxia UK, UK; ²CureDRPLA, USA; ³Department of Neurology, University of Washington, USA

From awareness to action: How patient organizations are driving DRPLA research forward

Dentatorubral-pallidoluysian atrophy (DRPLA) is an ultra-rare neurodegenerative disorder characterized by ataxia, cognitive decline, myoclonus, chorea, epilepsy, and psychiatric manifestations. With no disease-modifying therapies approved, coordinated efforts from patients, clinicians, and researchers are essential to accelerate translational progress. CureDRPLA, a family-led non-profit foundation, was established in 2019 to address this unmet need by driving research and enabling global collaboration. Since its inception, the organization has launched the DRPLA Research Program in partnership with Ataxia UK and supported a broad portfolio of studies, including development of human cellular and mouse models, natural history and biomarker investigations, and the creation of an international patient registry.

Collecting the lived experience from people with DRPLA and their caregivers has been a priority. We conducted qualitative interviews with caregivers to capture symptom progression, impact on daily activities and views on treatments. We also hosted, in collaboration with the National Ataxia Foundation, an Externally-Led Patient Focused Drug Development meeting to share lived experiences directly with the FDA, treatment developers, and researchers.

Another major focus of CureDRPLA is the advancement of therapeutic strategies aimed at reducing expression of ATN1, the causal gene underlying DRPLA, with the goal of targeting disease mechanisms at their origin. To this end, CureDRPLA actively supports and coordinates preclinical evaluation of multiple therapeutic modalities across international research teams. However, the rarity of DRPLA presents persistent challenges, particularly in harmonizing basic and clinical research activities across geographically dispersed sites.

Keywords: DRPLA, rare diseases, patient advocacy group

Biography

Dr Silvia Prades is a neurobiologist with over six years' experience coordinating international research and clinical programs in the rare disease space. She has established collaborations across academia, industry, and patient organizations. Combining scientific rigor with strategic leadership, she has built global research networks, designed patient registries, and strengthened the bridge between scientific innovation and real-world patient impact. In 2025 Silvia joined the Ataxia Global Initiative Steering Committee. Silvia holds a PhD in Neurobiology from University College London and is completing an MBA from Imperial.

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Gabriele Saretzki

Biosciences Institute, Newcastle University, Campus for Ageing and Vitality, Newcastle upon Tyne NE4 5PL, UK

Telomerase in brain and neurodegeneration

Telomerase is an enzyme that in its canonical function maintains telomeres, the ends of chromosomes in dividing cells that shorten their telomeres continuously. However, there are a number of telomere-independent functions known for the telomerase protein TERT (Telomerase Reverse Transcriptase). This includes the shuttling of the TERT protein from the nucleus to mitochondria where it decreases oxidative stress, apoptosis sensitivity and DNA damage. Recently, evidence has accumulated on a protective role of TERT in brain and postmitotic neurons. This function might be able to ameliorate the effects of toxic proteins such as amyloid- β , pathological tau and α -synuclein involved in neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD). Thus, these novel functions of TERT/telomerase in brain could have important implications for the treatment of neurodegenerative diseases. Mechanisms include changes in neuronal gene expression and synaptic functions together with the activation of signaling pathways as well as protein degradation pathways such as autophagy upon experimental increase of TERT levels in neurons and the brain of in vitro as well as in vivo models of neurodegenerative diseases such as Alzheimer's Disease (AD) and Parkinson's Disease (PD).

Recently, our group applied two telomerase activators (TA-65 and GRN510) on a mouse model of PD and found increased TERT expression levels, an improvement of PD-related symptoms such as gait and balance together with a significant decrease of total, phosphorylated and aggregated α -synuclein in these mice. We also showed the activation of autophagy as an important protein degradation process for toxic neuronal proteins by increasing TERT levels. These results highlight the non-canonical role of the telomerase protein TERT in brain and demonstrates its potential benefit for the amelioration of brain ageing and neurodegenerative diseases and might form the basis for the development of novel strategies and therapies against diseases such as AD and PD.

Biography

Dr. Gabriele Saretzki is a Lecturer at Newcastle University whose research focuses on the molecular biology of aging, cellular senescence, and age-related diseases. With expertise in biogerontology, she investigates the mechanisms that influence cellular lifespan, genomic stability, and the biological processes that contribute to healthy aging. Her work integrates molecular and cellular approaches to better understand the factors that drive aging and associated disorders.

Dr. Saretzki has made significant contributions to research on telomere biology, telomerase regulation, mitochondrial function, oxidative stress, and stem cell aging. Through her publications and collaborative research, she has advanced knowledge of the interplay between cellular metabolism and the aging process, with the long-term goal of identifying strategies to promote healthy longevity and develop interventions for age-related diseases.

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Hoda Tayebi Hillali¹

¹.A Medical-Surgical Dentistry Research Group (OMEQUI), Health Research Institute of Santiago de Compostela (IDIS), University of Santiago de Compostela (USC), Santiago De Compostela, Spain.

Lactoferrin and Altered Immune Response: Novel Insights into the Link Between Periodontitis and Alzheimer's disease

Periodontal disease has been associated with an increased risk of developing Alzheimer's disease. Traditionally, this relationship has been attributed to chronic systemic inflammation and/or the hematogenous dissemination of oral bacteria, both of which may potentiate neuroinflammatory processes. In this context, multiple studies have suggested that periodontal pathogens and their inflammatory mediators could contribute to the cerebral accumulation of β -amyloid plaques—a pathological hallmark of Alzheimer's disease—thereby establishing a plausible biological link between the two conditions. The identification of early biomarkers for Alzheimer's disease has become a public health priority in light of the global rise in dementia prevalence. Within this framework, salivary lactoferrin—an immunomodulatory glycoprotein primarily produced by neutrophils—has been shown to exhibit a marked reduction from the earliest stages of the disease, positioning it as a potential early diagnostic biomarker for Alzheimer's disease. Conversely, lactoferrin is overexpressed in the general population affected by periodontitis. In parallel, several hematological indices derived from routine blood counts, such as the neutrophil-to-lymphocyte ratio (NLR) and the systemic immune-inflammation index (SII, calculated from neutrophil, lymphocyte, and platelet counts), have been proposed as accessible markers of peripheral inflammation in both dementia and periodontal disease.

Our findings indicate that, overall, neither NLR nor SII shows significant differences between patients with dementia and healthy controls, nor between individuals with Alzheimer's disease and those with non-Alzheimer dementias. However, within the non-Alzheimer dementia subgroup, the presence of periodontitis is associated with a significant increase in both NLR and SII, attributable to elevated circulating neutrophil counts. In contrast, in Alzheimer's disease, periodontal status does not modify inflammatory indices, suggesting the presence of early immune dysregulation. The absence of a detectable peripheral inflammatory response in patients with Alzheimer's disease and concomitant periodontitis—despite the chronic inflammatory burden imposed by the latter—together with the early deficit in salivary lactoferrin, supports the hypothesis of an early systemic immune alteration. This pattern may reflect dysfunction in the activation or regulation of innate immunity, particularly within the neutrophil lineage, thereby limiting the peripheral expression of inflammation. In this context, reduced lactoferrin levels may function not only as an early biomarker of Alzheimer's disease but also as a potential indicator of immune dysregulation capable of modulating the biological interplay between periodontal inflammation and the neurodegenerative process.

Keywords: Alzheimer's disease, periodontitis, lactoferrin, innate immune dysregulation

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Biography

I am a researcher in special care dentistry, with a particular focus on patients with dementia and other cognitive impairments. My research focuses on identifying salivary biomarkers for the early diagnosis of Alzheimer's disease. My work also centers on improving oral healthcare strategies and clinical protocols for vulnerable populations.

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Angela Minnella, Kevin P. McCusker, Akiko Amagata, Marla Weetall, Joey C. Latham, Matthew B. Klein, Jeffrey K. Trimmer

PTC Therapeutics, Inc. Warren, NJ, USA

Targeting alpha-synuclein with the lipoxygenase inhibitor PTC-041 as a therapeutic approach for the treatment of Parkinson's disease

Parkinson's disease (PD) is a common neurodegenerative disorder in which α -synuclein aggregation and neuronal dysfunction are central pathological features. Ferroptosis, a regulated cell-death pathway driven by iron-dependent lipid peroxidation (including 15-lipoxygenase activity), has been implicated in PD and may contribute to synucleinopathy. We evaluated the therapeutic potential of PTC-041, an anti-ferroptotic lipoxygenase inhibitor, to mitigate lipid peroxidation, ferroptotic cell death, α -synuclein pathology, and functional deficits across PD-relevant models. PTC-041 was assessed in primary human PD patient-derived fibroblasts, primary rat neuronal co-cultures, and engineered immortalized rat dopaminergic neuronal cells for effects on viability, lipid peroxidation, and α -synuclein aggregation-related outcomes. In vivo efficacy was evaluated in Line 61 mice overexpressing human α -synuclein (aggregation/phosphorylation and proteomic markers of neuronal health) and in the 6-hydroxydopamine (6-OHDA) hemiparkinsonian rat model (motor deficits). PTC-041 potently protected PD patient fibroblasts from lipid peroxidation and subsequent ferroptotic death, prevented ferroptosis-related neuronal loss and astrogliosis in primary rat neuronal cultures, and inhibited ferroptosis-associated α -synuclein aggregation and nitrosylation in cell models. In Line 61 mice, PTC-041 reduced α -synuclein aggregation and phosphorylation and preserved neuronal and non-neuronal cell populations, supported by maintenance of neurofilament markers; proteomics indicated rescue of synaptic and cytoskeletal proteins required for NMDA-receptor function. Finally, PTC-041 protected against 6-OHDA-induced motor deficits in rats. These findings support continued development of PTC-041 and related lipoxygenase inhibitors as a disease-modifying strategy targeting ferroptosis-linked, pathogenic α -synuclein biology in PD.

Keywords: Parkinson's disease, ferroptosis, α -synuclein, 15-lipoxygenase, lipid peroxidation

Biography

Dr. Angela Minnella is the Director of Neuroscience at PTC Therapeutics, where she leads research efforts focused on developing innovative therapies for neurological and neurodegenerative disorders. With extensive experience in neuroscience and translational research, she oversees scientific programs that integrate molecular biology, pharmacology, and disease modeling to advance potential treatments from discovery through preclinical development.

Throughout her career, Dr. Minnella has contributed to research aimed at understanding the biological mechanisms underlying disorders of the central nervous system and identifying novel therapeutic strategies. Her work emphasizes the translation of fundamental scientific discoveries into targeted therapies, supporting the development of medicines designed to address unmet medical needs and improve outcomes for patients with rare and complex neurological diseases.

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Zhou Wu^{1,2}

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Microglia-Mediated Neuroinflammation Drives Neuropathology in Alzheimer's Disease during *P. gingivalis* Infection

Alzheimer's disease (AD) is the leading cause of dementia, yet its underlying drivers remain incompletely understood. Increasing evidence identifies microglia-mediated neuroinflammation as a central mechanism linking environmental risk factors to AD neuropathology. In this talk, I will present our findings demonstrating that *Porphyromonas gingivalis* (P.g) infection drives AD-related neuropathology through microglial activation. Using experimental models of systemic P.g infection and exposure to its lipopolysaccharide (P.gLPS), we show that infection-induced neuroinflammation promotes neuronal amyloid beta (A β) accumulation, tau hyperphosphorylation, and synaptic loss, key features associated with cognitive decline. In parallel, P.g infection enhances peripheral A β generation, suggesting a systemic contribution to brain pathology and highlighting a potential link between peripheral infection and central neurodegeneration. Mechanistically, we identify key enzymes, including cathepsin B and glycogen synthase kinase 3 β , as critical mediators of microglia-driven inflammatory pathways that exacerbate AD neuropathology. These findings provide insight into the molecular mechanisms by which infection-triggered immune responses influence disease progression. Together, our results support a model in which P.g infection amplifies microglia-mediated neuroinflammation, thereby accelerating the progression of AD neuropathology. Targeting infection-associated neuroinflammatory pathways may therefore represent a promising strategy for the prevention and early intervention of AD, particularly at stages when therapeutic efficacy is most achievable.

Keywords: microglia, neuroinflammation, *Porphyromonas gingivalis*, amyloid beta, Cathepsin B, glycogen synthase kinase 3 β

Biography

Dr. Wu holds an MD and PhD and studies how systemic inflammation drives neurodegeneration. Her research focuses on microglia-mediated neuroinflammation and the roles of enzymes such as cathepsins in immune signaling. Her work highlights how *Porphyromonas gingivalis* infection contributes to Alzheimer's disease and identifies potential targets for early intervention.

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Microglia promotes Amyloid beta accumulation and tau hyperphosphorylation in neurons after exposure to LPS from Porphyromonas gingivalis

A growing body of evidence highlights microglia as key regulators of neurodegenerative processes in Alzheimer's disease (AD), with bacterial components emerging as potential modulators of disease pathology. LPS from Porphyromonas gingivalis (P.gLPS), an oral pathogen-derived virulence factor, has been identified in AD brains, yet its functional role in disease progression is not fully defined. Here, we demonstrate that P.gLPS exposure provokes a robust microglial inflammatory response characterized by elevated secretion of pro-inflammatory mediators such as IL-1 β and TNF- α . These cytokines differentially influence neuronal pathology: IL-1 β enhances amyloidogenic processes via a cathepsin B-associated mechanism, whereas TNF- α facilitates tau pathology through activation of glycogen synthase kinase 3 β signaling. In vivo, systemic administration of P.gLPS leads to sustained cognitive impairment in middle-aged wild-type mice, accompanied by increased intracellular accumulation of amyloid β . In contrast, in transgenic AD models with established amyloid burden, P.gLPS primarily exacerbates disease severity by promoting tau-related neurodegenerative changes. Together, these results delineate a pathway by which infection-related factors modulate microglial activity to differentially impact amyloid and tau pathology across disease stages. Interventions targeting inflammation triggered by chronic oral infection may therefore offer therapeutic potential for delaying or mitigating AD progression.

Keywords: microglia, cytokines, LPS from Porphyromonas gingivalis LPS, amyloid beta, tau hyperphosphorylation.

Biography

Dr. Gui received her PhD in 2025 and studies microglia-mediated neuroinflammation in Alzheimer's disease. Her work reveals that Porphyromonas gingivalis-derived LPS activates microglia to drive amyloid- β accumulation and tau pathology, uncovering key mechanisms by which infection-triggered immune responses accelerate AD onset and progression, and identifying potential therapeutic targets.

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Victor Danelon¹, Nicholas C Morano^{2,3}, Sarah C Garrett-Thomson¹, Steven C Almo², Francis S Lee³, Barbara L Hempstead¹

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4 Department of Psychiatry, Weill Cornell Medicine, New York, New York, USA.

Human immunomodulatory ligand B7-1 mediates synaptic remodeling via the p75 neurotrophin receptor

Cell surface receptors, ligands, and adhesion molecules underlie development, circuit formation, and synaptic function of the central nervous system and represent important therapeutic targets for many neuropathologies. The functional contributions of interactions between cell surface proteins of neurons and nonneuronal cells have not been fully addressed. Using an unbiased protein-protein interaction screen, we showed that the human immunomodulatory ligand B7-1 (hB7-1) interacts with the p75 neurotrophin receptor (p75NTR) and that the B7-1:p75NTR interaction is a recent evolutionary adaptation present in humans and other primates, but absent in mice, rats, and other lower mammals. The surface of hB7-1 that engages p75NTR overlaps with the hB7-1 surface involved in CTLA-4/CD28 recognition, and these molecules directly compete for binding to p75NTR. Soluble or membrane-bound hB7-1 altered dendritic morphology of cultured hippocampal neurons, with loss of the postsynaptic protein PSD95 in a p75NTR-dependent manner. Abatacept, an FDA-approved therapeutic (CTLA-4-hFc fusion) inhibited these processes. In vivo injection of hB7-1 into the murine subiculum, a hippocampal region affected in Alzheimer's disease, resulted in p75NTR-dependent pruning of dendritic spines. Here, we report the biochemical interaction between B7-1 and p75NTR, describe biological effects on neuronal morphology, and identify a therapeutic opportunity for treatment of neuroinflammatory diseases.

Biography

Victor Danelon is a neuroscientist and biomedical researcher serving as a Research Associate in the Department of Medicine at Weill Cornell Medical College since 2022. His research focuses on the molecular and cellular mechanisms that regulate brain development, neuronal function, and neurodegenerative diseases. He earned his bachelor's degree in 2011 and a Ph.D. in Biological Sciences in 2016 from the National University of Córdoba, Argentina, where he developed expertise in molecular neuroscience, cell biology, advanced microscopy, and experimental models of neurological disorders.

Dr. Danelon has contributed to numerous peer-reviewed publications investigating topics such as brain-derived neurotrophic factor (BDNF) signaling, TrkB receptor biology, synaptic plasticity, dendritic morphogenesis, epilepsy-induced neuronal injury, and neuroprotection. By integrating molecular, cellular, and translational research approaches, his work advances the understanding of the biological mechanisms underlying neurological diseases and supports the development of potential therapeutic strategies for disorders affecting the nervous system.

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Volha Skrahina^{*1}, Ulrike Grittner^{2,3}, Christian Beetz¹⁶, Thomas Skripuletz⁴, Martin Juenemann⁵, Heidrun H Krämer⁵, Katrin Hahn⁶, Andreas Rieth⁷, Volker Schaechinger⁸, Monica Patten⁹, Christian Tanislav¹⁰, Stephan Achenbach¹¹, Birgit Assmus¹², Fabian Knebel^{3,13,14}, Stefan Gingele⁴, Aliaksandr Skrahin¹⁷, Sabine Roesner¹⁶, Arndt Rolfs¹⁷

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Hereditary transthyretin-related amyloidosis is frequent in polyneuropathy and cardiomyopathy of no obvious aetiology

Hereditary Transthyretin-Related Amyloidosis (hATTR), a clinically heterogeneous autosomal dominant disease caused by pathogenic variants in the TTR gene. The study objective was to define the prevalence of hATTR in patients with polyneuropathy and/or cardiomyopathy of no obvious aetiology.

A multicenter observational "Epidemiological analysis for the hereditary Transthyretin-Related AMyloidosis"-TRAM study was performed in Germany, Austria, and Switzerland.

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A total of 5141 participants were recruited by 50 neurologic and 27 cardiologic specialized centres. Genetic analysis demonstrated a 1.1% hATTR positivity rate. Twenty-one various TTR variants were identified. Body Mass Index was lower in the TTR-positive patients as an indicator for the involvement of the autonomic nervous system; the age of onset of clinical manifestations was higher in TTR-positive patients. There were no other genotype-phenotype correlations or the prevalence of specific clinical manifestations in TTR-positive patients.

Our data support the fact that Hereditary Transthyretin-Related Amyloidosis is clearly underdiagnosed in polyneuropathy and cardiomyopathy patients. Routine implementation of genetic testing is important and crucial in patients with unexplained polyneuropathy and/or cardiomyopathy to accelerate the earlier diagnosis and the time-sensitive treatment initiation.

Keywords: Hereditary transthyretin-related amyloidosis; cardiomyopathy; genetic testing; polyneuropathy.

Biography:

Volha investigated the translational regulation of the innate immune response in tuberculosis infection during her Master's thesis at the Max Planck Institute in Berlin, Germany. She has completed her PhD thesis on the host fungal micronutrient interaction at the Hans Knoell Institute in Jena, Germany in 2018. Volha has worked as a Director of Clinical Studies at CENTOGENE and as a Scientific Manager in Rhythm Pharmaceuticals. She executed multiple international clinical studies to analyse the prevalence of rare diseases allowing patients to be diagnosed and obtain treatment at the earliest possible moment.

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Eldem Sadikoglou¹,✉*, Daniel Domingo-Fernández², Natalia Savytska¹, Noemia Fernandes¹, Patrizia Rizzu¹, Anastasia Illarionova¹, Tabea Strauß^{3,4}, Sigrid C Schwarz^{3,5}, Alpha Kodamullil², Günter U Höglinger^{3,6}, Ashutosh Dhingra¹, Thomas Gasser^{1,7}, Peter Heutink¹

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Chr17q21.31 locus risk haplotype H1 susceptibility to Ferroptosis is mediated by the endolysosomal system

GWAS consistently identifies chr:17q21.31 locus H1 haplotype as a major risk factor for a wide spectrum of neurodegenerative diseases (NDs), including AD, PD, PSP & ALS. Despite the strong genetic association, functional experimental evidence linking the H1 haplotype with NDs related molecular signatures was missing. Chr:17q21.31 is a complex genomic locus harboring several genes, including MAPT, thousands of SNPs, an inversion polymorphism, and structural variants under strong linkage disequilibrium (LD), giving rise to two major haplotypes, the H1 and H2. In a Genome - Environment Interaction study with iPSC-derived neurons, we observed an increased susceptibility of the H1 haplotype to Mild Chronic Oxidative Stress. The sensitivity was manifested as extensive signs of axonal degeneration in the form of blebs/swellings due to microtubule (MT)-unbundling, followed by neuronal death that progressed and spread in a wave-like manner, mimicking the pathophysiology of NDs. Drug screening and Cell Painting assay showed that the neuronal death observed earlier in the H1 versus the H2 haplotype was via the iron-dependent lipid oxidation-mediated ferroptosis, while RNA-Seq and longitudinal assays revealed the differential reactive oxygen species and lysosomal biogenesis between the two haplotypes. We conclude that the increased susceptibility of the H1 haplotype to ferroptosis is attributable to SNPs and variants in LD on functionally related genes clustered within the locus. It is anticipated that an altered endolysosomal clearance pathway in the H1 genetic background, in conjunction with other risk variants causing protein misfolding and aggregates, will exaggerate the causality of risk genes, explaining the increased association of the locus in genomic studies.

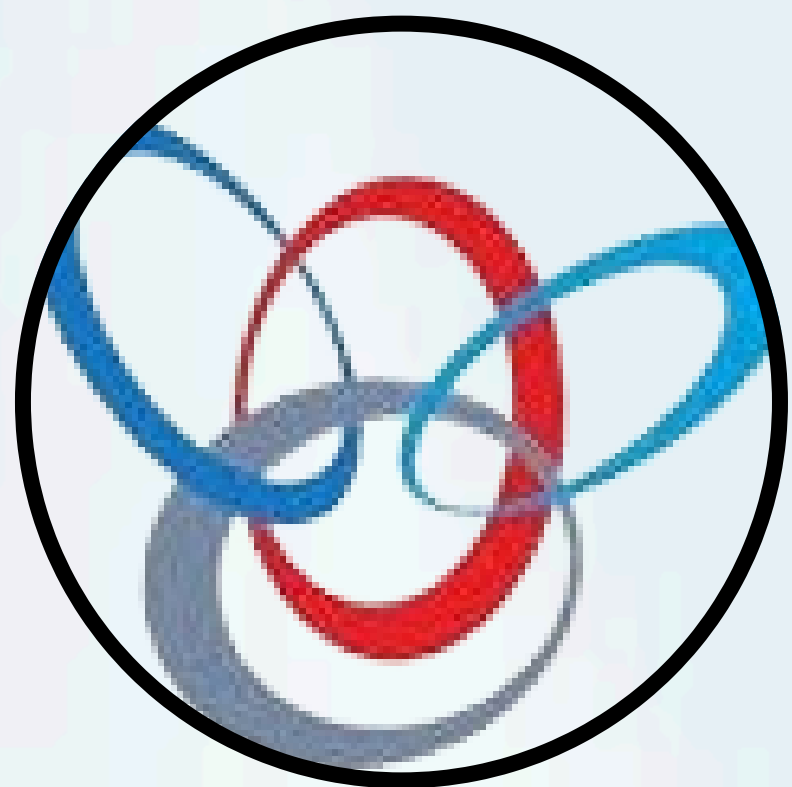
Keywords: Drugs-screening, chronic oxidative-stress, cell-painting, lysosomes, MT-unbundling

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Biography

Dr. Eldem Sadikoglou is a senior research technician in Genome Biology of Neurodegenerative Diseases (NDs) at DZNE-Tübingen. Her research focuses on the genome-environment intersection of NDs. Dr. Sadikoglou employs among others, high-throughput screening and longitudinal imaging assays to monitor neuronal resilience in real-time. Previously, she received her PhD from DUTH in Greece, where she investigated the DNA-binding properties of a neurodevelopmental transcription factor. Her current work is focused on identifying specific genetic alleles that confer resilience to NDs.

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Nyree Penn

Nysomnia Sleep, USA

Arousal Threshold and Sleep Stability Endotypes Under Controlled GABAergic Modulation: A Prospective Cohort Study

Background

Arousal threshold is a key non-anatomical determinant of sleep stability in both obstructive sleep apnea (OSA) and insomnia-spectrum disorders, yet remains incompletely characterized outside of respiratory event-based frameworks. Low arousal threshold contributes to ventilatory instability and sleep fragmentation, while chronic hyperarousal states—common in trauma-exposed and autonomically dysregulated populations—may impair sleep continuity independent of overt respiratory events.

Polysomnography (PSG) effectively identifies respiratory and movement-related abnormalities but does not directly quantify intrinsic arousal stability or continuity resilience. We hypothesized that controlled GABAergic modulation under monitored anesthesia care (MAC) could serve as a translational physiologic model to probe arousal threshold behavior and characterize sleep stability endotypes independent of respiratory event burden.

Methods

In this prospective observational cohort, 50 ASA Physical Status I–II adults with chronic insomnia or non-restorative sleep despite normal or mild PSG findings underwent propofol-based MAC under standard ASA monitoring (ECG, noninvasive blood pressure, pulse oximetry, and capnography).

Propofol was titrated using processed EEG guidance (Patient State Index) to achieve controlled cortical inhibition. Continuous neurophysiologic data included Patient State Index (PSI), Spectral Edge Frequency (SEF95), Suppression Ratio (SR), artifact burden, and hemodynamic parameters.

A standardized modulation window (mean 30 minutes; mean dose 4 mg/kg) was maintained, followed by discontinuation and structured observation of post-modulation recovery dynamics.

Strict signal integrity criteria were applied, including exclusion of epochs with SR >2% and artifact-controlled spectral analysis. No claims of physiologic REM or NREM replication were made.

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Primary endpoints included feasibility, safety, and reproducible signal acquisition. Secondary analyses focused on identification of arousal-stability phenotypes based on recovery trajectories.

This approach utilizes controlled propofol-mediated GABAergic modulation under MAC to transiently reduce cortical arousal constraints and enable physiologic characterization of intrinsic stability patterns during recovery.

Results

All 50 patients completed the protocol. Target EEG ranges were achieved in 100% of cases.

No airway compromise requiring advanced intervention occurred. Transient hypotension occurred in <10% of patients and was managed per protocol. No sustained hypoxemia (<90%) beyond 30 seconds was observed. No unplanned admissions occurred.

Signal integrity gating successfully excluded suppression-contaminated epochs. Post-modulation recovery analysis demonstrated reproducible heterogeneity in stability dynamics, including:

- Rapid arousal re-emergence profiles consistent with low arousal threshold physiology
- Sustained spectral continuity profiles suggestive of higher stability resilience
- Fragmented recovery trajectories consistent with instability-dominant phenotypes

These patterns were observed despite normal or near-normal PSG findings.

Conclusions

Controlled propofol-mediated GABAergic modulation under MAC is feasible, safe, and reproducible as a translational model for probing arousal threshold behavior and intrinsic sleep stability dynamics.

These findings support the concept that arousal instability represents a measurable physiologic dimension independent of respiratory events and may define clinically relevant endotypes across insomnia and OSA spectra.

Transient modulation of cortical arousal pathways provides a novel physiologic window into sleep stability dynamics not captured by conventional PSG.

This framework has potential implications for refining phenotypic classification in sleep-disordered breathing, understanding non-anatomical contributors to ventilatory instability, and advancing precision approaches to perioperative sleep risk stratification.

Further investigation is warranted to correlate these phenotypes with arousal threshold metrics, ventilatory control parameters, and clinical outcomes.

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Model of environment dependent amyloid transformation

Structural transformation of proteins producing the amyloid forms is presented. The fuzzy oil drop model allowing assessment of external force field participation in folding process (including structural transformations). This model applied for analysis of WT and amyloid forms identifies the necessary change of environmental conditions as the driving force for this transformation. The influence of environment (external force field) is quantitatively expressed in this model by K parameter. Three scenarios are recognised for fibril formation: 1 – the globular form of native protein representing low K gets changed to amyloid form described by high K values (transthyretin, VL domain of IgG); 2 – the biologically active form of protein characterised by high K value (effect of external force field specificity) changes its status to the form described by low K value (adaptation to water environment) (α -Synuclein) and 3 – the biologically active protein in form of fibril represents the status described by low K for both: individual chain as well as in 6-chains fibril treated as form of reservoir. It is interpreted as self-control for fibril propagation limited to 8 chain in maximum controlled by water environment. The fibrils of higher number of chains represent increasing K value what requires changes in external force field. As long as water is participating in folding process, the increase of fibril length (above 8 chains) is forbidden.

In conclusion: the amyloid transformation is driven by environment changes (external force field) expressed in fuzzy oil drop model by the value of K parameter.

Keywords: amyloid; environment; Alpha-synuclein; transthyretin; endorphin; hydrophobicity

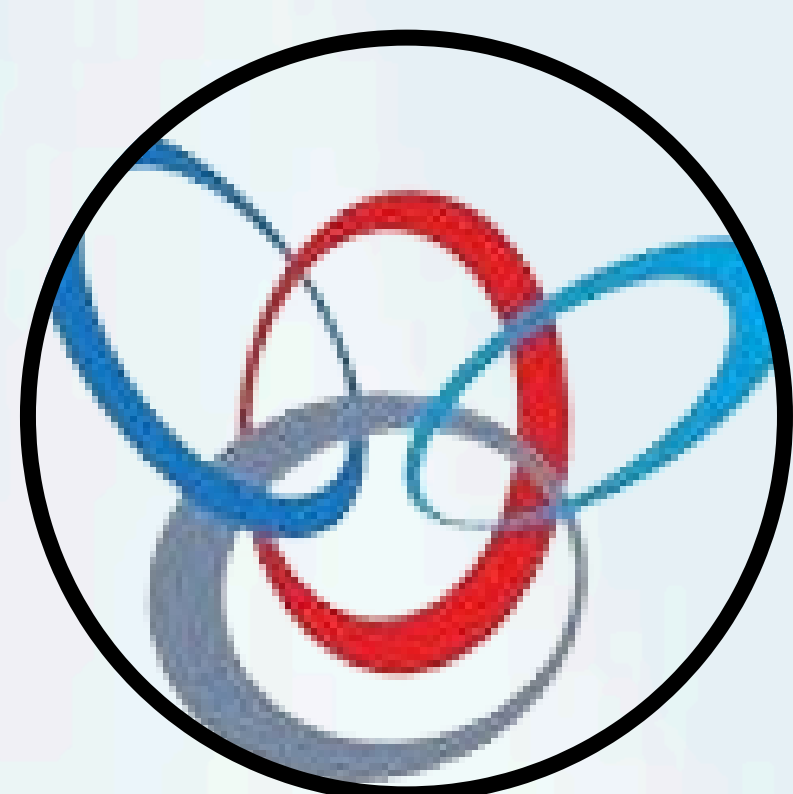
Biography

Irena Roterman-Konieczna - Professor Department of Bioinformatics and Telemedicine – Jagiellonian University – Medical College – Krakow, Poland

Educated in theoretical chemistry. Two years postdoc at Cornell University – H.A. Scheraga's group 1987-1989. In 2000-2020 chief of the Department of Bioinformatics and Telemedicine.

Author of fuzzy oil drop model treating the proteins as intelligent micelles with centric hydrophobic core and polar surface. Chief Editor of the journal Bio-Algorithms and Med-Systems (2000-2020). Author of about 200 publications and books: Systems Biology: 2023, Springer Simulations in Medicine: 2015, Walter de Gruyter (ISBN (978-3-11-04064403), Protein structure: 2019 Elsevier, From Globular Proteins to Amyloids - Elsevier 2026 - Simulating Protein Folding in Variable Environmental Conditions. Present on the 2% top scientists list (since 2020)

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**Pushpa Verma, Pruthvi Gowda, Nika N Danial,
David Van Vactor**

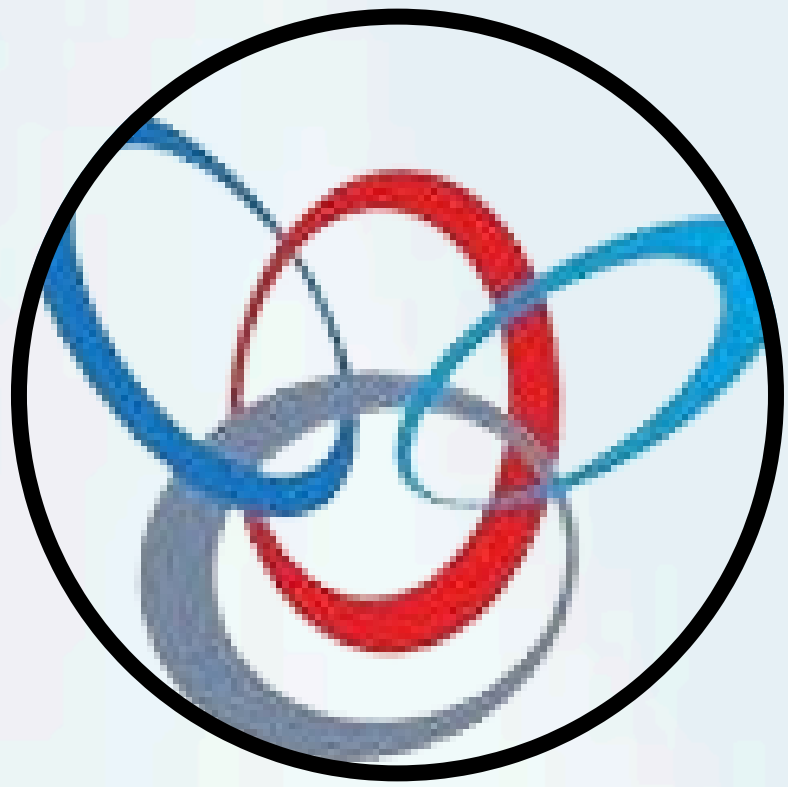
Harvard Medical School, 240 Longwood Avenue, Boston, Massachusetts, USA 02115

Brain-specific microRNA-mediated regulation of metabolic homeostasis in *Drosophila melanogaster*

To maintain optimal metabolic balance, an organism must coordinate its food intake, fat storage, and appropriate energy mobilization to meet physiological demands and support overall well-being. These metabolic processes require precise regulation to ensure timely responses to environmental and internal cues. Small non-coding microRNAs (miRNAs) are well suited for this role, as they can regulate physiological and adaptive processes that require rapid or localized control of gene expression.

We have identified a novel neural circuit in *Drosophila* that plays an important role in metabolic homeostasis, mediated by the conserved Central Nervous System (CNS)-specific miRNA miR-1000. We previously identified miR-1000 to be a neuroprotective miRNA by regulating Glutamatergic signaling in the brain (Verma et al., Nature Neuroscience, 2015). In addition to its role in neuroprotection, we recently discovered that miR-1000 also regulates key aspects of metabolism, including body weight, fat storage, survival during nutrient deprivation, and longevity (Verma et al, Genes and Development, 2026). miR-1000 achieves this by repressing the Neuropeptide Like Peptide 1 (Nplp1) gene in the CNS. Loss of miR-1000 and consequent increase in Nplp1 expression result in flies with higher fat storage and extended survival under nutrient-deprived conditions. While the biological function of Nplp1 neuropeptides has been largely unknown, this study highlights their critical role in maintaining metabolic homeostasis in *Drosophila*. Additionally, we observed that miR-1000 and Nplp1 levels change dynamically during refeeding after starvation, suggesting their involvement in adaptive metabolic responses. miR-1000 regulates triacylglycerol (TAG) synthesis and storage in lipid droplets, supports fat mobilization during starvation, and facilitates its storage upon refeeding. These findings underscore the importance of miR-1000 and Nplp1 in metabolic regulation and provide new insights into the mechanisms of energy balance.

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Agentic Multimodal Speech Analysis for Parkinson's Disease Detection: A Small LLM-Orchestrated Pipeline

Background

Speech is a promising non-invasive biomarker for Parkinson's Disease (PD). Yet most automated detectors are acoustic black boxes that ignore linguistic content and offer no clinician-interpretable rationale.

Objective

We introduce an agentic multimodal pipeline. A small open-source large language model (LLM) — Qwen2.5-1.5B-Instruct, 4-bit quantized — acts as an autonomous fusion controller rather than a fixed mathematical rule, orchestrating four specialized tools: it calls each, parses its output, weighs the evidence, and emits structured JSON with a PD/healthy decision, calibrated probability, per-modality importance weights, and a natural-language rationale.

Tools

(1) DistilHuBERT, a self-supervised acoustic transformer; (2) Whisper-tiny automatic speech recognition (ASR) plus the multilingual MiniLM sentence encoder; (3) prosodic features — jitter, shimmer, harmonics-to-noise ratio (HNR), fundamental-frequency (F0) variability — extracted via Praat and classified by a support vector machine (SVM); (4) per-modality calibrated logistic regressors (LR).

Methods

We use the openly licensed Italian Parkinson's Voice and Speech corpus (CC-BY-4.0), age-matched subset: 50 speakers (28 PD vs 22 controls; 786 utterances). Per-modality probabilities use leave-one-speaker-out to prevent leakage; a thin multi-layer perceptron (MLP) consumes the agent's 7-dimensional output. We report speaker-stratified 5-fold cross-validation with bootstrap 95% confidence intervals (CIs).

Results

Single-modality baselines (AUC, 95% CI): prosodic SVM 0.992 [0.956, 1.000]; DistilHuBERT+LR 0.967 [0.904, 1.000]; linguistic 0.404 [0.216, 0.549] (below chance — same-passage corpus). Agentic fusion: AUC 0.968 [0.878, 1.000], macro-F1 0.980, accuracy 0.980, sensitivity 0.964, specificity 1.000, with per-speaker JSON rationales naming the top modality. Label-permutation on the best baseline: $p < 0.001$.

Conclusion

A small-LLM-orchestrated pipeline matches state-of-the-art multimodal PD screening on a fully open corpus and produces interpretable rationales on a single CPU. Cross-corpus validation and a neurologist reader study follow.

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Table 1: Speaker-stratified performance on the Italian PVS corpus (n = 50).

Model	Modality	Dim	Accuracy	Macro-F1	AUC	AUC 95% CI
Prosodic SVM	Prosodic	12	0.920 ± 0.075	0.912 ± 0.088	0.992 ± 0.016	[0.956, 1.000]
DistilHuBERT + LR	Acoustic	1536	0.980 ± 0.040	0.980 ± 0.040	0.967 ± 0.067	[0.904, 1.000]
MiniLM + LR	Linguistic	395	0.480 ± 0.075	0.447 ± 0.104	0.404 ± 0.147	[0.216, 0.549]
Static late fusion	Aco+Ling+Pros	1943	0.980 ± 0.040	0.980 ± 0.040	0.967 ± 0.067	[0.890, 1.000]
Agentic-MLP (ours) †	LLM-orch.	7	0.980	0.980	0.968	[0.878, 1.000]

† Agentic-MLP uses leave-one-speaker-out (50 LLM calls, one per speaker); baselines use 5-fold speaker-stratified CV (mean ± SD across folds). AUC 95% CI from 1000-resample bootstrap. Permutation test on best baseline (prosodic SVM, 1000 permutations): p < 0.001. SVM = support vector machine; LR = logistic regression; MLP = multi-layer perceptron.

Keywords: Parkinson's disease; speech biomarker; multimodal; agentic AI; large language models; interpretability

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Impaired Dynamic Cerebral Autoregulation: A Potential Mechanism of Orthostatic Hypotension and Dementia in Parkinson's Disease

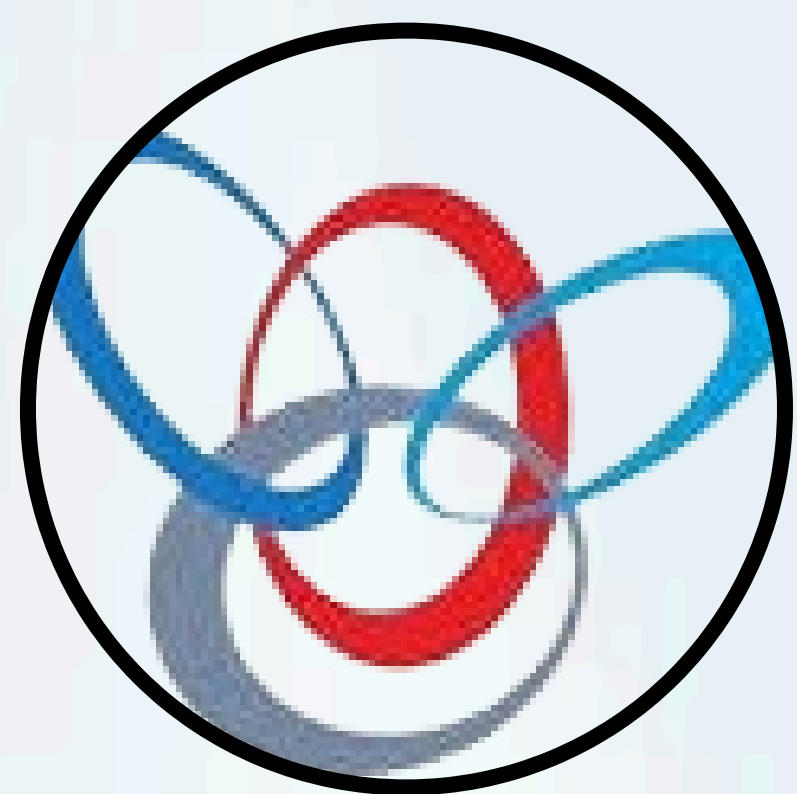
Orthostatic hypotension (OH) and cognitive impairment are common non-motor symptoms in Parkinson's disease (PD), yet the underlying mechanisms linking them remain unclear. This study investigated whether impaired dynamic cerebral autoregulation (dCA) mediates the relationship between OH and Parkinson's disease dementia (PDD). We enrolled 89 PD patients and 20 age- and sex-matched healthy controls. Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA). Cerebral blood flow velocity and beat-to-beat blood pressure were measured using transcranial Doppler and servo-controlled finger plethysmography in supine and standing positions. dCA was evaluated via transfer function analysis. Results showed that 23.6% of PD patients had OH, and they exhibited worse performance in language and orientation domains. PD patients with OH had significantly lower phase in the very low frequency range in both postures and higher normalized gain in the low frequency range when standing, indicating impaired dCA. Patients with PDD also showed higher low-frequency normalized gain in standing, which was significantly associated with dementia (OR: 3.756, 95% CI: 1.241–11.367). Lower education level was also a significant risk factor. These findings suggest that impaired dCA may serve as a potential mechanism linking OH and cognitive impairment in PD. Early evaluation of dCA could aid in identifying patients at risk for dementia.

Keywords: Parkinson's disease, orthostatic hypotension, cognitive impairment, cerebral autoregulation, dementia

Biography

Dr. Fubo Zhou is a vascular ultrasonography physician and researcher at Xuanwu Hospital, Capital Medical University, Beijing. Her research focuses on cerebrovascular hemodynamics, cerebral autoregulation, and neurovascular coupling in neurodegenerative diseases, particularly Parkinson's disease. She has co-authored 16 peer-reviewed articles in the field of aging neuroscience and vascular neurology.

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Analysis of Plant based Drugs and of Essential Oils from Indian and African sources: HPTLC, GC-MS, Anti-oxidant assay and In-silico approaches

It is well known that the use of traditional plant-based drugs has witnessed phenomenal growth in the last few decades. In our Institute, we are engaged in the analysis of plant-based drugs and essential oils with medicinal properties.

We have carried out extensive studies on a number of Indian Medicinal plants, which are well-reputed in Ayurveda. We briefly present here our investigations on *Hemidesmus indicus* R. Br. roots (Anantamul), *Ailanthus excelsa* Roxb. (Mahanimba) stem bark and four *Piper* species. In addition to a number of known triterpenoids and coumarino-lignoids a new triterpenoid designated hemidesterpene ($\Delta^{12,13}$ - dehydro-taraxesteryl acetate) has been isolated and characterised from *H. indicus*. A new compound Alru-1 was obtained from *Ailanthus excelsa* and was characterised as stigmasta-4,22-dien-3b-ol.

Several plants of both Indian and Africa are used in respective ethnomedical practices. We report here our comparative studies on two plant species used in both India and Africa. These are Shigru leaves (*Moringa oleifera* Lamb.) and *Cinnamomum zeylanicum* Blume from India and Tanzania.

Cinnamomum zeylanicum Blume (Lauraceae), is native to Sri Lanka and cultivated in India, Southeast Asia, and parts of Africa. Its stem bark is widely used in Ayurveda, Siddha in India and traditional medicine in Africa for treating digestive and respiratory ailments, rheumatism, and diabetes. Modern clinical studies have confirmed its antioxidant, anti-inflammatory, antimicrobial, anticancer, and antidiabetic effects. The phytochemical profile of *C. zeylanicum* varies markedly with genotype, soil, cultivation practices, and climate. We have carried out comparative studies comprising chromatographic profiling, volatile analysis, and antioxidant evaluation across regions in India and Africa. This study involves by comparing bark samples from India (Shillong and Kolkata) and Africa (Tanzania). Constituents identified include cinnamaldehyde, eugenol, coumarins, flavonoids, and other phenolics. HPTLC and GC-MS analyses revealed distinct chemical differences, including the exclusive presence of eugenol and higher cinnamaldehyde dimethyl acetal in the Tanzanian sample. Organoleptic and physicochemical properties also reflected regional influence. Antioxidant assays (DPPH and FRAP) showed superior activity in the Tanzanian variety, with the lowest IC₅₀ value (22.05 μ g/mL) and highest FRAP value (579 μ M). These results emphasize the impact of geographical origin on the quality and therapeutic potential of *C. zeylanicum*, supporting the need for standardisation. Molecular docking further demonstrated strong binding of eugenol to diabetes-related targets (PTP1B, PPAR γ , PPAR δ , PPAR α) and the Alzheimer's-linked enzyme BACE1, reinforcing its pharmacological relevance.

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Brain-oscillation-synchronized stimulation to enhance motor recovery in early subacute stroke: A randomized controlled double-blind three arm parallel-group exploratory trial comparing personalized, non-personalized and sham repetitive transcranial magnetic stimulation (BOSS-STROKE)

Globally, stroke is the third most frequent cause of death and the most frequent cause of permanent disability. Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive and relatively painless form of therapeutic brain stimulation. It showed moderate effect size in improving paretic arm-/hand function in stroke patients, indicating a strong medical need for better interventions. BOSS-STROKE is a multicenter randomized controlled double-blind three-arm parallel-group exploratory trial comparing novel personalized, conventional non-personalized and sham rTMS on arm-/hand function in subacute stroke patients. Personalized rTMS is a highly innovative form of stimulation that synchronizes rTMS to specific brain states identified by real-time analysis of electroencephalography (EEG). The TMS pulses are applied at the optimal instants in time to maximize the desired neuromodulatory effect: a long term increase in the activity of the motor networks affected by the stroke lesion. In preparatory studies, we have repeatedly demonstrated that personalized rTMS synchronized to the negative peak of the ongoing sensorimotor μ -oscillation induced more reliable and larger neuromodulatory effects than standard non personalized rTMS. The question we address for the first time here is to what extent this novel treatment translates into a meaningful clinical benefit, i.e., improved paretic hand-/arm function in stroke. We expect that this approach can relevantly improve the quality of life of many stroke patients in Germany.

Keywords: TMS-EEG, personalized treatment, brain stimulation, stroke

Biography

Dr. Anne Lieb is a neurologist and researcher specializing in neurorehabilitation and non-invasive brain stimulation. Her work focuses on the clinical translation of neuromodulation techniques, particularly repetitive transcranial magnetic stimulation, for motor recovery in stroke patients. She is involved in the development and clinical implementation of neurorehabilitative programs and contributes to translational research bridging neurophysiology and rehabilitation medicine.