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2026
Conference

The 5th International Electronic Conference on Brain Sciences & 1st International Electronic Conference on Neurosciences

9–11 March 2026 | Online

Program and Abstract Book

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MDPI

The 5th International Electronic Conference on Brain Sciences & 1st International Electronic Conference on Neurosciences

9–11 March 2026 | Online

MDPI

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Welcome from the Chair

Dear Colleagues,

It is our pleasure to invite you to attend the 5th International Electronic Conference on Brain Sciences & 1st International Electronic Conference on Neurosciences (IECBS-IECNS 2026) after the strong success of the previous annual online conference. IECBS-IECNS 2026 will be held online on 9–11 March 2026.

The field of neuroscience is one of the most important frontiers in biomedical studies, as many of the details that control brain function are still not well understood. This makes the study of the nervous system very exciting and fast-moving. This conference will address a variety of research topics that reflect some of the current areas of focus. The topics of the conference will be organized around the following sessions:

S1: Neurodegenerative Diseases;
S2: Cellular and Molecular Neuroscience;
S3: Behavioral Neuroscience;
S4: Cognitive Neuroscience;
S5: Neurorehabilitation;
S6: Neurotechnology and Neuroimaging;
S7: Systems Neuroscience.

The aim of this online conference is to bring together well-known worldwide experts who are currently working on brain sciences and to provide an online forum for presenting and discussing new results.

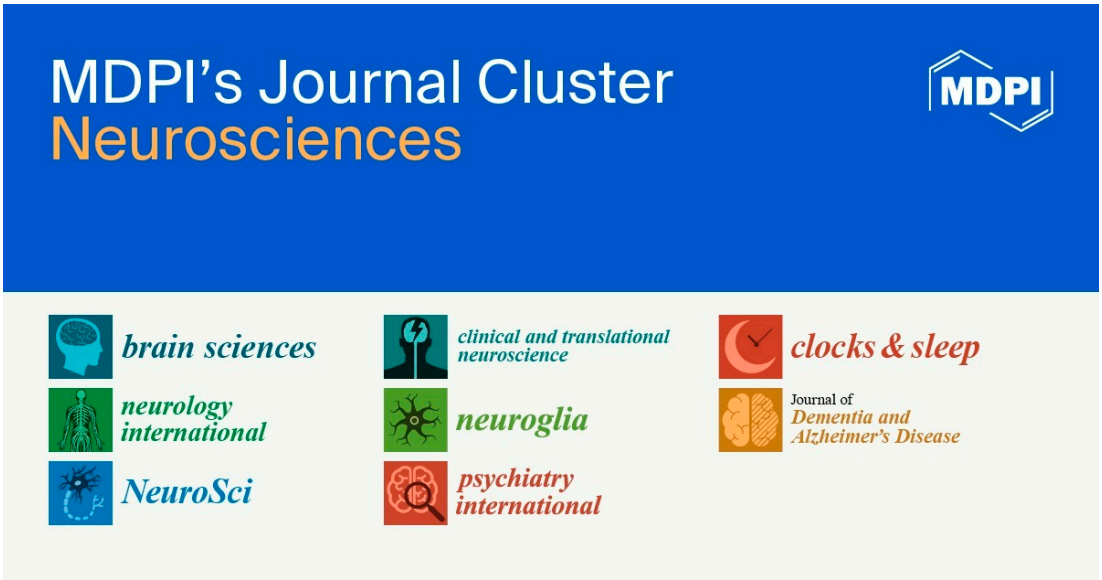
All accepted abstracts will be displayed on the conference website. Abstract submissions should be between 200 to 300 words in length. For detailed guidelines, please refer to the "Instructions for Authors".

We look forward to your contributions.

Prof. Dr. Stephen D. Meriney
Event Chair
Department of Neuroscience,
University of Pittsburgh, PA, USA

Brain Sciences (ISSN 2076-3425) is a peer-reviewed scientific journal that publishes original articles, critical reviews, research notes, and short communications on the topic of neuroscience. Our aim is to encourage scientists to publish their experimental and theoretical results in as much detail as is required to fully convey the information. There is no restriction on the maximum length of the papers. Electronic files reporting the extended details of computational, statistical, or experimental procedures, if unwieldy for presentation in the body of the manuscript, can be deposited as supplementary material.

Impact Factor: 2.8 (2024); 5-Year Impact Factor: 3.1 (2024)



MDPI's Journal Cluster of Neurosciences has a mission to publish recent advances in this field that have profound implications for understanding the nervous system, treating neurological disorders, and overall improving mental health. The eight participating journals include: Brain Sciences, Neurology International, NeuroSci, Clinical and Translational Neuroscience, Neuroglia, Psychiatry International, Clocks & Sleep, and Journal of Dementia and Alzheimer's Disease. These journals will collaborate to offer the scientific community the most up-to-date research and discoveries within the field of neuroscience.

Keynote Speakers

Prof. Dr. Matthew Huentelman

Early Detection and Prevention Division, Translational Genomics Research Institute (TGen), Phoenix, AZ, USA

Prof. Dr. Neera Ghaziuddin

Department of Psychiatry, University of Michigan, Ann Arbor, MI, USA

Invited Speakers

Dr. Irene Giovanna Aprile

IRCCS Fondazione Don Carlo Gnocchi, Firenze, Italy

Dr. James Robert Brasic

NYU Grossman School of Medicine, New York, USA

Dr. Nizhuan Wang

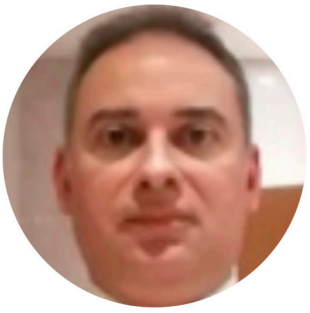
Department of Language Science and Technology, The Hong Kong Polytechnic University, Hong Kong SAR, China

Dr. Tommaso Ercoli

Department of Neurology, University of Sassari, Sassari, Italy

Assoc. Prof. Carla Masala

Department of Biomedical Sciences, Section of Physiology, University of Cagliari, Cagliari, Italy



Assoc. Prof. Paolo Solla

Clinical Neurological Unit, University of Sassari, Sassari, Italy

Professor Jarosław Sławek

Department of Neurology, Neurodegenerative Disorders and Neuroimmunology, Medical University of Gdansk, Gdańsk, Poland

Prof. Dr. Alon Kalron

Department of Physical Therapy, Sackler Faculty of Medicine, School of Health Professions, Tel-Aviv University, Tel-Aviv, Israel

Prof. Dr. Antoine Nonclercq

École Polytechnique de Bruxelles, Université Libre de Bruxelles, Brussels, Belgium

Program Overview

	Day 1	Day 2	Day 3
Morning	**Session 4.** Cognitive Neuroscience **Session 5.** Neurorehabilitation	**Session 3.** Behavioral Neuroscience	Flash Poster Sessions
Afternoon	**Session 1.** Neurodegenerative Diseases	**Session 6.** Neurotechnology and Neuroimaging	**Session 2.** Cellular and Molecular Neuroscience **Session 7.** Systems Neuroscience

IECBS-IECNS 2026 Program

9th March 2026 (Monday)

Session 4. Cognitive Neuroscience
Session 5. Neurorehabilitation

Time: 09:00 (CET, Basel) / 04:00 (EDT, New York) / 16:00 (CST Asia, Beijing)

Time in CET	Speaker	Title
9:00–9:10	*Welcome from the Session Chair* *Assoc. Prof. Carla Masala*	
9:10–9:30	**Assoc. Prof. Carla Masala** Invited Speaker	Correlations between olfactory, gustatory function, and cognitive abilities in different age ranges
9:30–9:50	**Assoc. Prof. Paolo Solla** Invited Speaker	Strategic management of infusional therapies in Parkinson's disease: impact on motor and non-motor symptoms
9:50–10:10	**Dr. Tommaso Ercoli** Invited Speaker	Neuropsychiatric Disorders in Patients Undergoing Advanced Therapies for Parkinson's Disease Management
10:10–10:25	**Nikol Petrović** Selected Speaker	EEG markers of cognitive load and mental fatigue in university students: a systematic review
10:25–10:40	**Kathrin Mertel** Selected Speaker	Can Music Training Enhance/Affect Working Memory and Speech-in-Noise Perception in Cochlear Implant Users? A Randomized Controlled Study of EEG Measures of Improvement
10:40–10:50	**Prof. Dr. Rocco Salvatore Calabrò** Session Chair	Welcome from the Session Chair
10:50–11:10	**Prof. Dr. Alon Kalron** Invited Speaker	Advanced technologies to improve neurorehabilitation outcomes
11:10–11:30	**Dr. Irene Giovanna Aprile** Invited Speaker	Upper limb rehabilitation: motor and cognitive outcomes
11:30–11:45	**Paula da Cruz Peniche** Selected Speaker	Telehealth intervention involving the HEARTS Technical Package and the additional use of an activity monitor to increase physical activity level post-stroke: a feasibility randomized controlled trial

Session 1. Neurodegenerative Diseases

Time: 14:00 (CET, Basel) / 09:00 (EDT, New York) / 21:00 (CST Asia, Beijing)

Time in CET	Speaker	Title
14:00–14:10	*Welcome from the Session Chair* *Dr. Grażyna Lietzau*	
14:10–14:25	**Alan Michael Palmer** Selected Speaker	The Cerebrocortical Disconnection Hypothesis: Reframing Alzheimer's Treatment Strategy
14:25–14:40	**Mayuri Nitin Bhosale** Selected Speaker	Bleomycin hydrolase deficiency and Homocysteine metabolism promote ER Stress, UPR dysregulation and apoptosis in the mouse neuroblastoma N2a-APPSwe cells
14:40–14:55	**Łukasz Mencel** Selected Speaker	Dysregulation of amyloid-β precursor protein processing by treatment with Hcy, Hcy-thiolactone, N-Hcy-proteins and deficiency of bleomycin hydrolase in mouse neuroblastoma N2a-APPswe cells
14:55–15:15	**Professor Jarosław Sławek** Invited Speaker	Many faces of Parkinson's Disease with special focus on interplay between dementia and dysautonomia

15:15–15:30	**Sonia Di Tella** Selected Speaker	Social Cognition impairments in Huntington's Disease progression
15:30–15:45	**Carlos Domínguez-Vargas** Selected Speaker	Impact of Genetic Modifiers on Clinical Severity and Neurodegeneration in Huntington Disease
15:45–16:00	**Chiara Scopa** Selected Speaker	Neurodegeneration and Transposable Element–Driven Immune Responses in C9orf72-ALS/FTD

10th March 2026 (Tuesday)

Session 3. Behavioral Neuroscience

Time: 08:00 (CET, Basel) / 03:00 (EDT, New York) / 15:00 (CST Asia, Beijing)

Time in CET	Speaker	Title
8:00–8:10	*Welcome from the Session Chair* *Assoc. Prof. Woon-Man Kung*	
8:10–8:30	**Dr. Nizhuan Wang** Invited Speaker	Single-Channel EEG-Based Brain-Computer Interfaces: Current Landscape and Future Directions
8:30–8:45	**Paula Denisa Saragea** Selected Speaker	The Importance of Behavioral Phenotyping in Evaluating Multitarget Phytochemicals in the 5xFAD Model of Alzheimer's Disease
8:45–9:00	**Eleftheria Taousani** Selected Speaker	Neurobiology of early mother–infant bonding, the role of oxytocin and favorable midwifery practices
9:00–9:15	**Abdelwahab Elshourbagy** Selected Speaker	Remote evaluation of stereotypy in hypokinetic catatonia with the Timed Stereotypies Rating Scale
9:15–9:30	**Maryam Behjat** Selected Speaker	The role of Prostaglandin E2 in alcohol-related inflammation in Drosophila melanogaster
9:30–9:45	**Amanda Gollo Bertollo** Selected Speaker	Intergenerational effects of childhood stress on depressive-like behaviors and function of glucocorticoids in the Nucleus Accumbens: therapeutic potential of Centella asiatica

Session 6. Neurotechnology and Neuroimaging

Time: 14:00 (CET, Basel) / 09:00 (EDT, New York) / 21:00 (CST Asia, Beijing)

Time in CET	Speaker	Title
14:00–14:10	*Welcome from the Session Chair* *Dr. James Robert Brasic*	
14:10–14:30	**Dr. James Robert Brasic** Invited Speaker	In vivo measurement of metabotropic glutamate receptor subtype 5 in humans with fragile X syndrome and idiopathic autism spectrum disorder
14:30–14:45	**Xuanling Chen** Selected Speaker	Pulsed arterial spin labeling MRI insights: Sevoflurane's effects on moyamoya disease and cerebral ischemia
14:45–15:00	**Daiki Matsuda** Selected Speaker	Learning-Dependent Modulation of Corticospinal Excitability During TMS-Based Neurofeedback
15:00–15:30	**Prof. Dr. Neera Ghaziuddin** Keynote Speaker	Electroconvulsive Therapy (ECT): a life-saving treatment
15:30–15:45	**Ismat Almadani** Selected Speaker	Protocol for Thalamocortical Functional Connectivity and Loss of Consciousness Investigation in Children with Epilepsy Using Stereo-EEG Data

11th March 2026 (Wednesday)

Flash Poster Session

Time: 09:00 (CET, Basel) | 04:00 (EDT, New York) | 16:00 (CST Asia, Beijing)

Time in CET	Poster Presenter	Title
Session 1		
9:00–9:05	**Maja Wojasiewicz**	Homocysteine and homocysteine thiolactone contribute to Alzheimer's disease via TAU modifications in N2A-APPswe cells
9:05–9:10	**Javiera Vicuña**	Plants with antioxidant potential in the ancestral knowledge of the Mapuche people in Chile: natural alternatives for neurodegenerative diseases
9:10–9:15	**Jose Joaquin Merino**	Is there any relationship between mercury from dental amalgams and Alzheimer's disease?: Role of mercury and peripheral CCL2 chemokines as biomarker of disease progression
9:15–9:20	**Loredana Maria Agavriloaei**	Toward Reproducibility in Preclinical Alzheimer's Research: The Case for Standardizing Aβ1-42-Induced Rodent Models
9:20–9:25	**Ephraim Chidi Ezeigbo**	Proteomic Profiling Reveals Systemic Effects of Peripheral mHtt Expression in Huntington’s Disease
9:25–9:30	**Paula Barciela Álvarez**	Targeting Neurotrophin Regulation by Polyphenols: Mechanistic Basis for Cognitive Resilience
9:30–9:35	**Ansab Akhtar**	Improvement of cognitive functions by indirubin-3′-oxime through GSK-3β- and CDK-mediated tau hyperphosphorylation inhibition in a rat model of Alzheimer's disease
Session 2		
9:35–9:40	**Lucas Brandalise**	Metal-dependent neural protection by HP-derived coordination compounds against mitochondrial dysfunction in glial cells
9:40–9:45	**Carlos Domínguez-Vargas**	Genome-Wide Variant Associations and Biological Pathways in Postherpetic Neuralgia
Session 3		
9:45–9:50	**Abdelwahab Elshourbagy**	Feasibility of identifying catatonia using the Bush-Francis Catatonia Rating Scale via remote motor assessments
Session 4		
9:50–9:55	**Ilenia Pinna**	Long COVID-19 effects on the olfactory, gustatory, and cognitive functions
9:55–10:00	**Grigorii Vyacheslavovich Iskarevskii**	The impact of age and sports experience on balance control: physiological and cognitive insights
10:00–10:05	**Anna Mikhailovna Beknazarova**	Acute physical exercise enhances semantic memory processing speed: evidence from a reaction-time lexical decision task
10:05–10:10	**Elizaveta Kozhevnikova**	Psychophysiological Correlates of Geometric Visual Illusions: A Comparative Study of the Ponzo and Müller-Lyer Effects.
10:10–10:15	**Yingying Wang**	Cognitive Neurological Mechanisms and Causal Analysis of Brain-to-Brain Synchronization Between Teachers and Students
10:15–10:20	**Xinbi Zhang**	Effects of Different Stress States on Athletes’ Cognitive Function and the Underlying Neural Mechanisms
10:20–10:25	**Olena Gennadiivna Aliyeva**	Concomitance of endothelium disorders in the brain and heart vessels in ischemic heart disease
10:25–10:30	**Nadejda Bocheva**	Neuronal similarity in perceiving illusory and real rotation directional flips
Session 5		
10:30–10:35	**Victoria Kuvaeva**	Assessment of knee muscle performance in para-athletes with unilateral transtibial amputation
Session 6		

10:35–10:40	**Moses O. Sokunbi**	Normative Convolutional Neural Network Modelling of Structural MRI for Personalised Neuroimaging
Session 7		
10:40–10:45	**Margarita Nikulina**	Sensorimotor adaptation in virtual environments: the critical role of individual cognitive style

Session 2. Cellular and Molecular Neuroscience
Session 7. Systems Neuroscience

Time: 14:00 (CET, Basel) | 09:00 (EDT, New York) | 21:00 (CST Asia, Beijing)

Time in CET	Speaker	Title
14:00–14:10		*Welcome from the Session Chair* *Dr. Keehoon Lee*
14:10–14:40	**Prof. Dr. Matthew Huentelman** Keynote Speaker	MindCrowd and the Mobile Laboratory: scalable and inclusive approaches for the study of the aging brain
14:40–14:55	**Ávila Guerrero Antonio** Selected Speaker	Quercetin suppresses mRNA Expression of Fto and the TNF-α/NF-κB/NLRP3 Inflammasome Pathway in Hypothalamus of Diet-Induced Obese Rats
14:55–15:10	**Sheila Sousa Gomes Fortes** Selected Speaker	Modeling the blood–brain barrier under neuroinflammation using a BBB-on-a-Chip
15:10–15:25	**Bikri Samir** Selected Speaker	A 100-Day Study of Biochemical, Behavioral, and Cognitive Changes Associated with Hippocampal and Prefrontal Cortex Alterations in Experimental Type 1 and Type 2 Diabetes
15:25–15:40	**Mariia Zhukova** Selected Speaker	Response of microglia and neural cells in the ICR mouse brain to experimental reserpine therapy due to traumatic brain injury
15:40–15:55	**Alexandra-Mara Cimpanu** Selected Speaker	Targeting Neuroinflammation in Alzheimer's Disease with a Nicotinic Metabolite: Insights from the 5xFAD Mouse Model
15:55–16:05		*Welcome from the Session Chair* *Dr. Vasileios Papaliagkas*
16:05–16:25	**Prof. Dr. Antoine Nonclercq** Invited Speaker	Vagus Nerve Activity as a Biomarker for Seizures: Insights from 3D Modelling
16:25–16:40	**Mousa Javidialsaadi** Selected Speaker	Task-dependent modulation of aftereffects during visuomotor adaptation
16:40–16:55	**Vsevolod Lyakhovetskii** Selected Speaker	WM/GM ratio of mammalian spinal cord: insights from African hedgehog (Atelerix albiventris)

Session 1. Neurodegenerative Diseases

sciforum-163610: *Persea americana* Seed Extract Mitigates 3-NPA-Induced Neurotoxicity in a Rat Model of Huntington's Disease via Antioxidant, Anti-Inflammatory, and Anti-Apoptotic Mechanisms

Yusuf Salami [1,2,*], Osaretin Albert Taiwo Ebuehi [1] and Holiness Stephen Adedeji Olasore [1]

[1] Department of Biochemistry, Faculty of Basic Medical Sciences, University of Lagos, Lagos, Nigeria
[2] Department of Chemical Sciences, College of Natural and Applied Sciences, Crescent University Abeokuta, Abeokuta, Ogun State, Nigeria

Abstract

Introduction: The global rise in age-related neurodegenerative disorders, including Alzheimer's, Huntington's, and Parkinson's diseases, represents a major health challenge, as existing therapies are largely symptomatic and fail to prevent progressive neuronal loss. Consequently, increasing attention has focused on natural compounds with neuroprotective and antioxidant properties. This study investigated the neuroprotective potential of *Persea americana* (avocado) seed extract (ASD) against 3-nitropropionic acid (3-NPA)-induced neurotoxicity, with emphasis on its antioxidant, anti-inflammatory, and anti-apoptotic mechanisms.

Methods: Methanol-extracted *P. americana* seed powder was orally administered to thirty-six male Wistar rats (150–200 g), randomly divided into six groups (n = 6): control, 3-NPA only, 3-NPA + 300 mg/kg ASD, 3-NPA + 600 mg/kg ASD, 300 mg/kg ASD only, and 600 mg/kg ASD only. The extract was administered for one week prior to 3-NPA exposure (10 mg/kg, intraperitoneally) and continued for two additional weeks. Behavioural, biochemical, and histopathological evaluations were performed. Oxidative stress indices, antioxidant enzyme activities, and succinate dehydrogenase (SDH) activity were measured, alongside gene expression analyses of Keap1, NF-κB, TNF-α, COX-2, Bad, and Bcl2.

Results: *Persea americana* seed extract significantly attenuated 3-NPA-induced behavioural deficits and restored SDH and antioxidant enzyme activities. The extract reduced lipid peroxidation and markedly downregulated the expression of NF-κB, TNF-α, COX-2, Bad, and Keap1, while upregulating the anti-apoptotic gene Bcl2. Histopathological examination further demonstrated reduced neuronal degeneration in extract-treated groups.

Conclusion: These findings indicate that *Persea americana* seed extract confers substantial neuroprotection against 3-NPA-induced neurotoxicity through antioxidant, anti-inflammatory, and anti-apoptotic mechanisms. The results suggest that the extract holds promise as a natural therapeutic candidate for oxidative stress-associated neurodegenerative disorders.

sciforum-154999: Blood-Based Biomarkers for Early Detection of Alzheimer's Disease: A Systematic Review and Meta-Analysis

Marina Corrêa Freitas

ICBS – International Center for Biomedical & Space Sciences, LIASTRA International Astronomy League, Rio de Janeiro, Brazil

Alzheimer's disease (AD) is the most common cause of dementia, characterized by amyloid-β plaques, tau neurofibrillary tangles, and progressive neuronal loss. Current diagnostic tools, including cerebrospinal fluid (CSF) analyses and positron emission tomography (PET) imaging, are accurate but invasive, costly, and not widely available. The identification of blood-based biomarkers reflecting AD neuropathology represents a major breakthrough toward accessible and scalable diagnostics.

This systematic review and meta-analysis aimed to evaluate the diagnostic performance of four plasma biomarkers—phosphorylated tau at threonine 181 (p-tau181), phosphorylated tau at threonine 217 (p-tau217), amyloid-beta 42/40 ratio (Aβ42/Aβ40), and neurofilament light chain (NfL)—for early and prodromal stages of AD. Following PRISMA 2020 guidelines, PubMed, Scopus, and Web of Science databases were searched for studies published between 2015 and 2025. Sixty-four studies encompassing 23,456 participants were included and analyzed using random-effects models (DerSimonian–Laird method).

The pooled area under the curve (AUC) was highest for plasma p-tau217 (AUC = 0.91, 95% CI: 0.88–0.94), followed by NfL (AUC = 0.86), p-tau181 (AUC = 0.84), and Aβ42/Aβ40 (AUC = 0.82). Combining p-tau217, NfL, and Aβ42/Aβ40 improved overall diagnostic accuracy (AUC = 0.94) with a sensitivity of 90%. Heterogeneity was moderate (I^2 = 47%) and mainly related to assay type and cohort variability.

These results demonstrate that plasma p-tau217 and NfL can achieve near-CSF accuracy for detecting AD pathology, providing robust, non-invasive, and cost-effective biomarkers suitable for large-scale screening and early intervention. Integrating plasma biomarkers with machine learning models may further enhance diagnostic precision, supporting a paradigm shift toward accessible and personalized approaches in Alzheimer's disease detection and monitoring.

sciforum-158548: Strategic management of infusional therapies in Parkinson's disease: impact on motor and non-motor symptoms

Paolo Solla

University of Sassari

Background: The management of advanced Parkinson's disease (PD) is often complicated by the failure of conventional oral therapies to provide sustained symptom control. PD patients progressively develop debilitating motor fluctuations and "OFF" periods, alongside long-term treatment complications which severely impact functional autonomy. Advanced PD is also characterized by a heavy burden of non-motor symptoms (NMS), including neuropsychiatric features, sleep disturbances, and autonomic dysfunction. These NMS are often underestimated but profoundly impact overall well-being. Strategic management of these patients can be improved by the administration of infusion therapies which represent a critical advancement in addressing these challenges. In this scenario, continuous delivery of levodopa-carbidopa intestinal gel (enteral), subcutaneous foslevodopa/foscarbidopa and apomorphine (subcutaneous) may be useful to achieve a more stable and continuous pharmacokinetic profile compared to pulsatile oral dosing. Strategic administration of infusion therapies represents a major advancement in addressing these challenges.

Objective: To describe the primary impact of this strategy in the significant reduction not only on motor fluctuations but also on non-motor fluctuations.

Discussion: PD infusion therapies are related to the concept of continuous dopaminergic stimulation and dramatically are able not only to reduce motor "OFF" time and duration of dyskinesias but also to significantly improve non-motor fluctuations, restoring functional independence and enhancing patients' quality of life. In fact, the efficacy of infusion therapy extends beyond motor control. Its strategic management is fundamentally reliant on a comprehensive, multidisciplinary care model. This team-based approach allows for intensive monitoring during dose titration and long-term follow-up, enabling the active identification and treatment of NMS.

Conclusions: Tailored infusion PD treatments represent a cornerstone of advanced PD care. Its success is optimized when embedded within a strategic, multidisciplinary framework that utilizes advanced monitoring to concurrently manage both motor and non-motor symptoms, leading to superior clinical outcomes.

sciforum-164325: ANTIOXIDANT EFFECT OF *HIBISCUS SABDARIFFA* AQUEOUS EXTRACT IN MICE BRAINS EXPOSED TO MONOSODIUM GLUTAMATE- INDUCED ALTERATIONS IN MICE

Adaeze Ngozi Adebesin [1,*], Esther Ayomide Ambali [1] and Abayomi Mayowa Ajayi [2]

[1] Department of Pharmacology and Therapeutics, Olabisi Onabanjo University, Ogun State, Nigeria
[2] Department of Pharmacology and Therapeutics, University of Ibadan, Ibadan, Nigeria

Introduction: Monosodium glutamate (MSG), a widely used flavor enhancer, is associated with neurotoxicity and oxidative stress in the brain. *Hibiscus sabdariffa* aqueous extract (HSAE), rich in polyphenols and anthocyanins, is known for its antioxidant properties and may mitigate MSG-induced brain damage. Nothing has been done previouisly on effect of HSAE on MSG-induced toxicity hence, the reason for this study which is also a part of a depression study.

Objectives: This study aimed to evaluate the antioxidant and neuroprotective effects of HSAE on MSG-induced alterations in mice brains, focusing on oxidative stress markers, antioxidant enzyme levels, acetylcholinesterase activity, and histological changes in the hippocampal dentate gyrus.

Methodology: Male Swiss mice of ages between 8 to 10 weeks were used and divided into six groups (n=6): control, MSG (2.5 g/kg), fluoxetine (Flx as standard from previous literature reviews, 20 mg/kg), and HSAE (50, 100, and 200 mg/kg) co-treated with MSG.

The extract, standard and MSG were prepared freshly and administered daily for 14 days. MSG was administered via subcutaneous route while the extract and standard was administered via intraperitoneal route. 30 minutes after administration, behavioural tests were carried out then sacrificed to extract brain samples used for the antioxidant assays. Samples were extracted, homogenised and supernatants were decanted and kept for further use. The supernatant was assayed for malondialdehyde (MDA), nitrite, reduced glutathione (GSH), catalase (CAT), superoxide dismutase (SOD) and acetylcholinesterase activities (AchE) activities using spectrophotometric methods. Histological analysis of the dentate gyrus was performed to assess neuronal damage.

Results: MSG significantly increased MDA, nitrites, and acetylcholinesterase levels while reducing GSH, CAT, and SOD levels, indicating oxidative stress and cholinergic disruption. HSAE (100 and 200 mg/kg) and Fluoxetine significantly reversed MDA, nitrites, and acetylcholinesterase elevations. HSAE (200 mg/kg) and Fluoxetine significantly restored GSH. CAT and SOD levels were also improved, with HSAE (200 mg/kg) showing CAT levels comparable to Fluoxetine. Histological analysis revealed MSG-induced degeneration in the dentate gyrus, with HSAE (200 mg/kg) and Fluoxetine showing significant restoration of neuronal architecture.

Conclusion: HSAE, particularly at 200 mg/kg, exhibits potent antioxidant and neuroprotective effects against MSG-induced neurotoxicity by reducing oxidative stress, enhancing antioxidant defenses, and preserving neuronal structure, comparable to fluoxetine. These findings suggest HSAE's potential as a therapeutic agent for oxidative stress-related neurodegenerative conditions.

This study is part of a depression study that is ongoing as well.

sciforum-164053: Association Between *Helicobacter pylori* Infection and Motor Severity and Levodopa Response in Parkinson's Disease: A Systematic Review

Carlos Domínguez-Vargas [1,*], Paloma Marylí Prado-López [2], Jesús Eduardo García-Hernández [1], Miranda Citlali Pérez-Castellón [1], Samantha Jonnue Ramírez-Flores [1], Emiliano Peña-Durán [1], Ramsés Emiliano Martínez-Hernández [3], Ulises Moisés González-Reyes [4] and Gerardo Amaya-Tapia [5]

[1] Medicine and Surgery, University Center of Health Sciences (CUCS), University of Guadalajara, 44430, Guadalajara, Mexico
[2] Neuropsychology, University Center of Health Sciences (CUCS), University of Guadalajara, 44340, Guadalajara, Mexico
[3] Medicine and Surgery, Faculty of Medical and Biological Sciences "Dr. Ignacio Chávez", Universidad Michoacana de San Nicolás de Hidalgo (UMSNH), 58010, Morelia, México
[4] Nursery, University Center of Health Sciences (CUCS), University of Guadalajara, 44340, Guadalajara, Mexico
[5] Infectious Diseases, Hospital General de Occidente, 45170, Zapopan, Mexico

Introduction: Parkinson's disease (PD) is a progressive neurodegenerative disorder frequently accompanied by gastrointestinal dysfunction. Chronic **Helicobacter pylori** infection has been proposed as a modifiable factor influencing motor severity and levodopa pharmacokinetics. This systematic review aimed to evaluate whether *H. pylori* infection is associated with worse motor outcomes or impaired levodopa response in patients with Parkinson's disease.

Methods: A systematic review was conducted in accordance with PRISMA guidelines. PubMed/MEDLINE, Scopus, Web of Science, EMBASE, and regional databases were searched for studies published between 2010 and 2025 in English or Spanish. Eligible publications included human observational studies, interventional studies assessing *H. pylori* eradication, and systematic reviews or meta-analyses reporting motor severity (UPDRS-III), motor fluctuations, or levodopa response. A qualitative narrative synthesis was performed due to clinical and methodological heterogeneity across studies.

Results: Eighteen studies met our inclusion criteria, including 11 observational studies, 4 interventional eradication studies, and 3 systematic reviews/meta-analyses. Observational studies consistently reported that *H. pylori*-positive PD patients exhibited higher motor severity scores, increased motor fluctuations, delayed levodopa onset, and reduced daily "on" time compared with non-infected patients. Interventional studies demonstrated that successful *H. pylori* eradication was associated with improvements in motor fluctuations and, in some cases, reductions in UPDRS-III scores. Systematic reviews and meta-analyses supported a detrimental association between *H. pylori* infection and motor outcomes, although heterogeneity and moderate risk of bias were noted across included studies.

Conclusions: Available evidence suggests that *Helicobacter pylori* infection is associated with worse motor severity and impaired levodopa response in Parkinson's disease. While eradication therapy appears to improve motor fluctuations, variability in study design and risk of bias limit definitive conclusions. Future well-designed randomized controlled trials and quantitative syntheses are warranted to clarify causality and inform clinical recommendations.

sciforum-161108: Association Between Salivary Cortisol, Xerostomia, and Nitric Oxide Levels in Patients With Multiple Sclerosis: A Cross-Sectional Study

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Introduction: Multiple sclerosis (MS) is characterized by neuroinflammation, autonomic dysfunction, and alterations in the hypothalamic–pituitary–adrenal (HPA) axis. Salivary cortisol is a useful biomarker of physiological stress, while xerostomia and nitric oxide (NO) dysregulation may reflect autonomic or inflammatory changes frequently observed in MS. However, the relationship between cortisol, NO, xerostomia, and anxiety symptoms remains unclear. This study aimed to evaluate the associations among these biomarkers and clinical features in patients with MS.

Methods: A cross-sectional study was conducted in 39 patients with MS. Salivary cortisol and NO were measured through validated assays. Xerostomia was assessed using the Abslang Test (0 = no xerostomia, 1 = xerostomia). Anxiety levels were measured using the Beck Anxiety Inventory (BAI). MS duration was converted to years for analysis. Due to non-normal distributions, Spearman correlations and Mann–Whitney U tests were applied. Logistic regression was performed to examine the predictive value of cortisol for xerostomia, expressed as odds ratios (OR).

Results: Xerostomia was present in 41% of participants. Cortisol levels were higher in patients with xerostomia compared with those without (median 6.02 vs. 4.56; $p = 0.050$). Logistic regression showed that cortisol increased the odds of xerostomia by 33% per unit increase (OR = 1.33; 95% CI: 0.96–1.85; $p = 0.085$). Cortisol showed a weak-to-moderate positive correlation with xerostomia ($\rho = 0.32$; $p = 0.047$) and a moderate negative correlation with NO ($\rho = -0.46$; $p = 0.0035$). No associations were found between cortisol and age, MS duration, or anxiety scores.

Conclusions: Salivary cortisol is associated with xerostomia and lower NO levels in MS, suggesting a possible neuroendocrine–salivary pathway. Although the predictive effect of cortisol on xerostomia did not reach statistical significance, the consistent trends across analyses support a potential physiological link. Larger studies are warranted to further explore these interactions.

sciforum-164311: Bleomycin hydrolase deficiency and Homocysteine metabolism promote ER Stress, UPR dysregulation and apoptosis in the mouse neuroblastoma N2a-APPSwe cells

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Background: Alzheimer's disease (AD) is a neurodegenerative disorder characterised by endoplasmic reticulum (ER) stress, unfolded protein response (UPR) and apoptosis. Elevated plasma total homocysteine (tHcy), known as Hyperhomocysteinemia (HHcy) have been linked to an increased risk of AD. HHcy and related metabolites such as Hcy thiolactone (HTL), *N*-homocysteinylated (*N*-Hcy)-protein and bleomycin hydrolase (BLMH), an enzyme that detoxifies HTL, are also associated with AD pathology. However, it is not fully understood how *Blmh* gene deficiency, Hcy and its metabolites promote ER stress, UPR dysregulation and apoptosis in the development of AD.

Aim: To study the effects of Hcy, HTL, and *N*-Hcy-protein treatments and *Blmh* gene silencing on the expression of genes involved in ER stress, UPR and apoptosis pathway in mouse N2a-APPswe cells.

Method: N2a-APPswe mouse neuroblastoma cells, which contain a human APP transgene with Swedish mutations, were cultured in complete DMEM/F12 medium. Cells were treated with different concentrations of Hcy, HTL, and *N*-Hcy-protein in methionine-free medium for 24 h. *Blmh* gene expression was silenced using *Blmh*-specific siRNA with Lipofectamine in Opti-MEM medium for 48 h. Proteins involved in ER stress, UPR, and apoptosis were quantified by Western blotting, and the corresponding mRNAs were analysed by RT-qPCR.

Results: *Blmh* gene silencing, Hcy, HTL, and *N*-Hcy-protein increased levels of ER chaperone GRP78, indicating the induction of ER stress, which further activated the UPR pathway by increased levels of ATF3 and CHOP. The proapoptotic proteins BAX and CASPASE 3 were increased, whereas the anti-apoptotic protein BCL-2 were reduced, suggesting a shift towards cell death. RT-qPCR analysis confirmed that mRNA expression changes were similar to the protein level changes.

Conclusion: The *Blmh* gene silencing, Hcy, HTL and *N*-Hcy-protein may contribute to the development and progression of AD by inducing ER stress, dysregulation of the UPR pathway and apoptosis.

sciforum-161110: Clinical and Paraclinical Predictors of Conversion from Clinically Isolated Syndrome to Multiple Sclerosis: A Retrospective Cohort Study

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Introduction: Clinically isolated syndrome (CIS) represents the first demyelinating event suggestive of multiple sclerosis (MS). However, the risk of conversion to MS varies widely among individuals. Identifying clinical and paraclinical predictors of conversion is essential for early risk stratification and timely therapeutic decision-making. This study aimed to evaluate the clinical, neuroimaging, laboratory, and neurophysiological factors associated with MS conversion in a real-world CIS cohort.

Methods: A retrospective analysis was conducted with 273 patients diagnosed with CIS. The primary outcome was conversion to MS, defined by the treating neurologist using standard diagnostic criteria. Bivariate analyses (t-tests, χ^2, and Fisher's exact test) were used to compare clinical and paraclinical variables between converters and non-converters. Predictors with p 0.20 and clinically relevant variables were included in a multivariate logistic regression model to identify independent predictors of conversion.

Results: Of the 273 patients included, 148 (54.2%) converted to MS. Age did not differ significantly between converters and non-converters (33.4 vs. 34.8 years, p=0.29). Neuroimaging markers were strongly associated with conversion: periventricular lesions (74.3% vs. 20.0%; OR_adj ≈ 8.1, 95%CI 4.0–16.1), infratentorial lesions (88.5% vs. 49.6%; OR_adj ≈ 4.8, 95%CI 2.2–10.3), and spinal cord lesions (73.6% vs. 62.4%; OR_adj ≈ 2.16, 95%CI 1.02–4.59). Oligoclonal bands in CSF were significantly different across groups (χ^2=48.3; p≈3.3×10^{-11}) and remained an independent predictor (p≈0.014). Gender exhibited a trend toward significance after adjustment (p≈0.051). Initial EDSS could not be reliably compared due to missing data.

Conclusions: In this CIS cohort, the strongest independent predictors of conversion to MS were periventricular, infratentorial, and spinal cord lesions, along with the presence of oligoclonal bands. These findings support the central prognostic role of MRI and CSF biomarkers in early MS risk stratification and highlight the need for prompt monitoring and therapeutic considerations in high-risk CIS patients.

sciforum-148573: Comprehensive investigation of dysregulated gene expression in MS CSF lymphocytes reveals novel insights on disease pathology, population risk, and treatment

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Multiple sclerosis (MS) is an autoimmune disease that produces a complex range of symptoms including movement trouble, cognitive impairment, and bowel dysfunction. The disease progresses through the central nervous system (CNS), particularly the cerebrospinal fluid (CSF). Previous research suggests that CSF lymphocytes contribute significantly to disease progression, resulting in the misregulation of many genes. This thorough study examined the bulk tissue expression and single-nucleotide polymorphisms (SNPs) of 16 misregulated CSF genes. The study noted ethnic populations that are more susceptible to genetic variation along with drugs that can be used as MS treatments. The bulk tissue plots revealed a correlation between gene expression changes in the CSF and damage in the gut-associated lymphoid tissue (GALT). The SNP data further affirmed this finding with the tibial nerve—often associated with bowel damage in MS—having the most variants. Analyzing the population genetics of the collected SNPs showed that ethnic groups in Latin America and Africa were most likely to have the highest frequency in the least common alleles. Lastly, the study identified 12 drugs that regulate the dysregulated CSF genes with the vast majority being anti-cancer agents or histone deacetylase (HDAC) inhibitors. In short, discovering the effects and treatments of the misregulated CSF genes produced findings that deviate from prior MS studies. The results unveiled a much greater significance of the peripheral nervous system (PNS) and enteric nervous system (ENS), non-European susceptibility to symptoms, and drugs similar to cancer treatment.

sciforum-164277: Dysregulation of amyloid-β precursor protein processing by treatment with Hcy, Hcy-thiolactone, *N*-Hcy-proteins and deficiency of bleomycin hydrolase in mouse neuroblastoma N2a-APPswe cells

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Introduction: Homocysteine (Hcy), homocysteine thiolactone (HTL), *N*-Hcy-proteins and dysfunction of bleomycin hydrolase (BLMH) are associated with Alzheimer's disease (AD). AD is characterized by the accumulation of amyloid-β (Aβ) plaques. Aβ is formed from amyloid-β precursor protein (APP) via the amyloidogenic pathway, involving β-secretase BACE1 and γ-secretase complex (Nicastrin, PSEN1, PEN2, APH-1). The impact of Hcy, its metabolites and BLMH dysfunction on the development and progression of AD is not fully understood.

Objective: We tested the hypothesis that Hcy, its metabolites and *Blmh* silencing affect APP processing pathways in mouse neuroblastoma N2a-APPswe cells.

Methods: Neuroblastoma N2a-APPswe cells harboring a human transgene with a mutation in the *APP* gene were treated with different concentrations of Hcy, HTL or *N*-Hcy-proteins. The expression of the *Blmh* gene was silenced using a *Blmh*-specific siRNA. Scrambled siRNA was used as a negative control. Proteins involved in APP metabolism pathway, i.e., APP, phospho-APP, BACE1, PSEN1, PEN2 and Nicastrin, were quantified by Western blotting. The expression of *APP*, *Bace1 Psen1*, *Psenen* and *Ncstn* mRNAs was analyzed by RT-qPCR.

Results: Hcy increased APP, phospho-APP, BACE1 and PSEN1. At mRNA level, Hcy downregulated the expression of the *Psen1* gene. HTL increased protein levels of APP, BACE1, PSEN1 and Nicastrin. This treatment also decreased the expression of the *Psenen* gene (encoding PEN2). Treatments with *N*-Hcy-proteins increased level of PSEN1 protein and downregulated the expression of the *Psenen* gene. There was no significant impact of treatments on PEN2 protein level. Silencing of the *Blmh* gene resulted in upregulation of APP, Nicastrin, PEN2 and downregulation of BACE1 protein. Silencing significantly upregulated the expression of *APP* and *Psenen* mRNAs.

Conclusions: Hcy, HTL, *N*-Hcy-protein and *Blmh* gene silencing resulted in dysregulated APP processing in N2a-APPswe cells, suggesting their association with AD progression.

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sciforum-164034: Histomorphology and histomorphometry changes in the hippocampal regions of hydrocephalic adult rats induced using intracisternal kaolin suspension.

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Abstract

Background: Kaolin, an aluminum silicate usually injected intracisternally, induces hydrocephalus by causing an inflammatory reaction and fibrous scarring in the meninges; this leads to an obstruction of the cerebrospinal fluid (CSF) pathways, and ventricular enlargement ensues. Its effect on the histomorphometry of the hippocampus is yet to be fully explored; therefore this study aimed at assessing the histomorphology and histomorphometry changes in the hippocampus of hydrocephalic adult rats induced with sterile kaolin. Methods: Comminicating Hydrocephalus was induced intracisternally in adult rats, both sexes, using 0.04ml of 250mg/ml of kaolin (n=6), while the sham procedure was performed on controls (n=6), where a needle was introduced into their cisternal magna to mimic kaolin-induced hydrocephalus but no injection was performed. Rats were weighed weekly and then sacrificed on the fourth week via intracardiac perfusion to harvest the brain tissues. Harvested brains were processed for Cresyl violet-staining to assess neuronal damage. Data were analysed using GraphPad Prism 8 and ImageJ software. Results: In the quantitative analysis, the body weights of the rats induced with kaolin were significantly lower than those of the control rats (p0.0001), while in the qualitative analysis, photomicrographs of Cresyl violet-stained hippocampal regions, CA1, CA3 and the Dentate Gyrus, showed a severely disorganized laminal cytoarchitecture of hydrocephalic rats compared to the control, and the pyknotic indices (PIs) of CA1, CA3 and the Dentate Gyrus of the 250mg/ml group were significantly higher than those of the control. Conclusion: Findings from this study showed that hydrocephalus altered the cytoarchitecture of the pyramidal neurons of the hippocampus of adult rats following intracisternal kaolin injection.

sciforum-164146: Homocysteine and homocysteine thiolactone contribute to Alzheimer's disease via TAU modifications in N2A-APPswe cells

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Background: Elevated levels of homocysteine (Hcy) and/or homocysteine thiolactone (HTL) are linked to neurodegenerative diseases, including Alzheimer's disease (AD). Hyperphosphorylation or elevated acetylation of TAU protein are hallmarks of AD. However, the mechanistic roles of Hcy and HTL in the development and progression of AD are not fully understood.

Aim: We tested a hypothesis that Hcy and HTL promote TAU accumulation via hyperphosphorylation and acetylation of TAU in mouse neuroblastoma N2A-APPswe cells.

Methods: Neuroblastoma N2A-APPswe cells (N2A-APPswe) harboring a human transgene with mutation in the amyloid precursor protein (APP) gene were grown on the complete DMEM/F12 medium. Cells were treated with 20-200 μM Hcy or HTL. Phosphorylation and acetylation of TAU, as well as selected enzymes involved in TAU modification, were quantified by Immunofluorescence and Western blotting.

Results: Immunofluorescent analysis showed that Hcy and HTL upregulate phosphorylated TAU at threonine 205 and serine 396 and acetylated TAU at lysine 174, relative to untreated cells. However levels of total TAU were not affected in cells after Hcy and HTL treatment compared to control cells. At the same time, Western blots showed upregulation of CDK5 and GSK3α after Hcy and HTL treatments compared to untreated cells, while GSK3β level was not affected.

Conclusion: Treatment with Hcy or HTL affects TAU modifications which, in turn, promote TAU aggregation in mouse neuroblastoma N2A-APPswe cells. The hyperphosphorylated TAU at Ser396 and at Thr205 results from elevated levels of TAU kinases, CDK5 and GSK3α, which are upregulated by Hcy and HTL treatments.

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* First seven authors contributed equally to this work

sciforum-161113: Impact of Genetic Modifiers on Clinical Severity and Neurodegeneration in Huntington Disease

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Introduction: Huntington disease (HD) is an autosomal dominant neurodegenerative disorder caused by an expanded CAG repeat in the HTT gene. Although disease onset and progression are known to vary between individuals, recent research has highlighted the role of genetic modifiers—both cis- and trans-acting—in influencing somatic instability, neuronal vulnerability, and age of onset. However, the extent to which these modifiers affect cross-sectional clinical severity and neuroimaging markers remains unclear. This study evaluates the phenotypic impact of genetic modifier categories in a large real-world HD dataset.

Methods: We analyzed a cohort of 48,536 genetically confirmed HD cases containing demographic variables, HTT CAG repeat length, motor severity (Chorea Score), functional impairment (Functional Capacity), and a quantitative neurodegeneration index (Brain Volume Loss). Genetic information included HTT (primary cause), trans-acting modifiers (e.g., MSH3, MLH1), and cis-acting somatic expansion events, classified into three categories. We compared clinical and MRI variables across categories using ANOVA and assessed correlations between CAG length, age, and severity measures using Pearson coefficients.

Results: The distribution of modifier categories was balanced (primary: 25.2%; trans-acting: 50.0%; cis-acting: 24.8%). Mean values of Chorea Score, Brain Volume Loss, and Functional Capacity were nearly identical across groups, with no statistically significant differences ($p = 0.27$, $p = 0.48$, $p = 0.10$, respectively). CAG repeat length was not correlated with motor severity, neurodegeneration, or functional status (all $r \approx 0$; $p > 0.05$). Similarly, age showed no significant associations with any clinical variable. Disease stage (pre-symptomatic, early, middle, late) did not yield significant group differences.

Conclusions: Across this large real-world cohort, genetic modifier categories did not produce meaningful differences in cross-sectional clinical severity or MRI-based neurodegeneration. These findings suggest that modifier effects are subtle and likely act on longitudinal trajectories, age of onset, or progression rates rather than on isolated clinical snapshots. Longitudinal modeling is warranted to capture their true phenotypic impact.

sciforum-155319: Improvement of cognitive functions by indirubin-3′-oxime through GSK-3β- and CDK-mediated tau hyperphosphorylation inhibition in a rat model of Alzheimer's disease

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder affecting nearly 45 million people worldwide. Current therapeutic options, including cholinesterase inhibitors (rivastigmine, donepezil, and galantamine) and the NMDA receptor antagonist memantine, offer only symptomatic relief without halting disease progression. Thus, there is an urgent need to identify novel therapeutics capable of targeting the molecular drivers of AD pathology.

The hallmark features of AD—amyloid plaques and neurofibrillary tangles (NFTs)—are closely linked to tau hyperphosphorylation mediated by glycogen synthase kinase-3β (GSK-3β) and cyclin-dependent kinases (CDKs). Hyperactive GSK-3β promotes NFT formation, oxidative stress, and neuronal death.

In this study, we evaluated Indirubin-3′-oxime, a potent GSK-3β inhibitor with antioxidant properties, in a streptozotocin-induced rat model of AD. Indirubin-3′-oxime treatment (10 mg/kg, i.p.) administered for 21 days significantly improved cognitive performance in the Morris water maze and novel object recognition tests compared to untreated AD rats (p 0.01). Biochemical assays revealed a 40–50% reduction in phosphorylated tau levels and marked inhibition of GSK-3β and CDK5 activities. Oxidative stress markers such as MDA and nitrite were significantly decreased, while antioxidant enzymes (SOD, catalase) were restored toward normal values.

Histopathological analysis showed reduced neuronal degeneration in the hippocampal CA1 region, confirming neuroprotection. These findings demonstrate that Indirubin-3′-oxime mitigates tau hyperphosphorylation and oxidative stress, thereby improving cognitive function. The results support its therapeutic potential as a disease-modifying agent in Alzheimer's disease.

sciforum-164383: Investigating Subtype-Specific Neuroplastic Changes through White Matter tractography in Parkinson's Disease

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Introduction

Parkinson's disease (PD) presents heterogeneous motor deficits associated with disruptions in distinct neural circuits. White-matter plasticity can be examined using diffusion tensor imaging (DTI) and fractional anisotropy (FA). Rhythmic Auditory Stimulation (RAS) induces compensatory plasticity within the basal ganglia-thalamo-cortical (BGTC) and cerebello-thalamo-cortical (CTC) networks, which are differentially preserved in rigidity-dominant (RD) and tremor-dominant (TD) PD, respectively. However, tract-specific plasticity underlying these effects remains poorly understood. This study examined FA differences in the internal capsule (IC), cerebellar projections, and corpus callosum (CC) to characterize subtype-specific neuroplasticity relevant to Neurologic Music Therapy (NMT).

Methods

Seventeen PD patients were classified as TD (n=11) or RD (n=6) using UPDRS scores. DTI data were processed in FSL and analyzed using tract-based spatial statistics. FA differences were assessed in predefined white-matter tracts. Principal component analysis (PCA) examined cerebellar peduncle (CP) organization.

Results

RD patients showed significantly higher FA in the left IC and across all CC subregions compared to TD patients. No group differences were observed in cerebellar projections. PCA revealed a unilateral left superior CP contribution in RD, whereas TD patients showed distributed bilateral contributions across all CPs.

Discussion

Findings suggest localized motor tract alterations and stronger interhemispheric connectivity in RD, contrasted with preserved, distributed cerebellar organization in TD. These subtype-specific patterns have implications for tailoring NMT interventions, with RD potentially benefiting from bilateral and cognitive-motor strategies, and TD from cerebellar-based motor sequencing approaches.

sciforum-161097: Is there any relationship between mercury from dental amalgams and Alzheimer's disease?: Role of mercury and peripheral CCL2 chemokines as biomarker of disease progression

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Studies comparing mercury (Hg) levels with those of age-matched controls often focus on identifying exposure risks in patients with Alzheimer's disease (AD), including the release of mercury from long-term dental amalgams. Mercury exposure can lead to oxidative stress and inflammation, along with Abeta and hyperphosphorylated tau accumulation in the brain. Mercury accumulation has been demonstrated in AD transgenic mice, and certain chemokines have been shown to contribute to cognitive impairment. However, the link between mercury, cognitive dysfunction and chemokines remains to be elucidated in AD patients with long-term dental amalgams.

Our study aims to determine whether peripheral CCL2 chemokine and mercury levels differ between AD patients with long-term dental amalgams compared to patients without AD.

Mercury levels were quantified by ICP-MS (in hair: µg/g), peripheral CCL2 chemokine ligand levels were measured by ELISA, and cognitive impairment was evaluated by a Mini Mental Test. We compared the CCL2 biomarker and mercury levels in patients with long-term dental amalgams without AD (n = 42), AD patients without dental amalgams (n = 55), AD patients with long-term dental amalgams (n = 13 AD + dental amalgams), and age-matched controls without dental amalgams (n = 42 without AD).

Our results indicate a 17% rise in mercury levels in patients with AD and dental amalgams compared to AD patients without dental amalgams, while a rise in MCP-1 levels was observed using the Mann–Whitney test (p = 0.048). Within the AD+dental amalgam group, 16% showed cognitive impairment according to their Mini Mental scores. These peripheral MCP-1 increases correlated with mercury levels in AD patients with long-term dental amalgams. MCP-1 levels correlated with Mini Mental scores using the Spearman correlation test (r = 0.45, p 0.05, AD + dental amalgam group). Additionally, mercury levels correlated with MMSE scores. However, these correlations were absent in the other study groups.

Conclusion: AD patients with long-term dental amalgams have higher peripheral MCP-1 levels and lower Mini Mental scores.

sciforum-154784: Multi-Omic Integration and AI-Powered Biomarker Discovery in Neurodegenerative Diseases: Towards Precision Neurodiagnostics

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Abstract:

Neurodegenerative diseases (NDDs) such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis are among the most debilitating disorders worldwide, characterized by progressive neuronal loss, molecular heterogeneity, and limited therapeutic options. Despite extensive research, the early detection and mechanistic understanding of these diseases remain major clinical challenges.

This study employs a comprehensive **multi-omics framework** integrating transcriptomic, proteomic, and metabolomic datasets from human post-mortem brain tissues and cerebrospinal fluid. By leveraging **artificial intelligence (AI)** and machine learning algorithms, including random forest classifiers and deep autoencoders, data from over 2,000 patient samples (ADNI, GEO, and AMP-PD) were analyzed to identify robust biomarkers and molecular subtypes.

Results demonstrate 31 shared molecular networks dysregulated across NDDs, prominently involving mitochondrial impairment, autophagy dysfunction, and neuroinflammatory pathways. The AI-based diagnostic model achieved **92% classification accuracy** for distinguishing early Alzheimer's disease from age-matched controls. Key hub genes—*LRRK2*, *TREM2*, and *SYNGR3*—were identified as central regulatory nodes. **In-silico drug repurposing** further suggested metformin and rapamycin analogs as potential modulators of these targets.

In conclusion, this research underscores the potential of AI-driven multi-omics integration in unveiling cross-disease biomarkers and accelerating precision diagnostics in neurodegenerative disorders. Future work aims to validate these findings through clinical cohorts and digital neurophenotyping.

In conclusion, this research underscores the potential of AI-driven multi-omics integration in unveiling cross-disease biomarkers and accelerating precision diagnostics in neurodegenerative disorders. Future work aims to validate these findings through clinical cohorts and digital neurophenotyping.

sciforum-158498: Multi-Omics and Structural Modeling for Identifying Phytochemical Modulators of Pathogenic Tau States: Integrative Discovery Pipeline of Tau-Targeting Phytochemicals Using SNP Biomarker Screening, Structural Modeling, and Network Pharmacology

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Tau being a difficult target: intrinsically disordered, conformationally plastic, heavily post-translationally modified, and capable of adopting multiple fibrillar states in disease. Genetic variation within MAPT (e.g., coding SNPs at regulatory phosphorylation sites such as S262, S356) and splicing-related haplotypes modulates tau's biochemistry and disease risk. Tau hyperphosphorylation, misfolding and aggregation drive axonal transport failure, synaptic loss, and neuronal death across tauopathies. Traditional ligand-screens using a single tau construct miss the broader landscape of proteoforms and patient-specific variants. Phytochemicals or Polyphenols (e.g., curcumin, EGCG, resveratrol), alkaloids (berberine, huperzine A), and terpenoids (ginkgolides) emerged as dominant scaffolds. Quantitative mapping revealed these compounds consistently attenuate tau phosphorylation at key epitopes (S262, S356, S396/S404), inhibit fibrillization, and restore microtubule stability through multitarget actions on GSK3β, CDK5, and PP2A pathways. In this systematic review and scientometric analysis, we identified and profiled 186 studies investigating phytochemicals with tau-modulating activity. Mining of MAPT variant data highlighted regulatory SNPs near phosphorylation sites S262 and S356 that modulate tau's phosphorylation kinetics and aggregation propensity. Network pharmacology linked tau and its regulatory enzymes to central hubs including VEGFA, IL1B, ESR1, and APP, suggesting that efficacious phytochemicals confer combined tau-directed and anti-inflammatory or neurotrophic effects. AlphaFold-based structural modeling of wild-type and variant tau isoforms enabled molecular docking of 42 prioritized compounds. Top-ranked hits curcumin analogs, EGCG, and asiaticoside exhibited strong binding near the microtubule-binding domain with favorable CNS permeability and low predicted toxicity based on ADMET profiling. Together, these analyses delineate phytochemical scaffolds that combine direct biochemical modulation of pathogenic tau states with network-level neuroprotective activity. The integrated genetic–structural–systems framework presented here provides a rational basis for advancing multitarget phytochemicals as stratified therapeutics in tauopathies.

sciforum-157914: Neurodegeneration and Transposable Element–Driven Immune Responses in C9orf72-ALS/FTD

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Neurodegenerative diseases with a dementia component, such as Alzheimer's disease (AD) and C9orf72-associated ALS/FTD (C9-ALS/FTD), exhibit genomic instability and aberrant activation of transposable elements. Our previous work in AD uncovered a pathogenic mechanism whereby retrotransposon (RTE) mobilization generates RNA::DNA hybrids that trigger innate immunity through the cGAS–STING pathway. Here, we aim to determine whether this RTE-RNA::DNA hybrid-STING axis is also engaged in C9-ALS/FTD, and to determine its clinical relevance while assessing its potential as a therapeutic target using patient-derived cerebral organoids.

We employed a translational approach combining C9-ALS/FTD patients postmortem tissue with patient-derived cerebral organoids. In human brain tissue, activation of the RTE-RNA::DNA hybrid-STING axis was assessed using single-cell transcriptomic profiling, biochemical assays and immunofluorescence microscopy. Six-month-old cerebral organoids were validated as an in vitro model recapitulating the molecular signatures observed in patient tissue and subsequently used for pharmacological testing. Organoids were treated with the FDA-approved reverse transcriptase inhibitor lamivudine, and effects on RNA::DNA hybrid accumulation and downstream signaling were quantified.

In the frontal cortex of C9-ALS/FTD patients, we observed aberrant RTE activation, accompanied by cytoplasmic RNA::DNA hybrid accumulation and STING pathway activation. Organoids faithfully recapitulated these phenotypes. Treatment with lamivudine significantly reduced RNA::DNA hybrid accumulation, demonstrating pharmacological modulation of this pathway and supporting its therapeutic potential in C9-ALS/FTD.

This study provides the first demonstration that the RTE–RNA::DNA hybrid–STING axis is aberrantly activated in C9-ALS/FTD, linking retrotransposon dysregulation to disease-relevant innate immune signaling. By showing that RNA::DNA hybrid accumulation can be reduced with lamivudine, we highlight a translatable therapeutic entry point targeting this pathway in C9-ALS/FTD and related dementias.

sciforum-160301: Neuroprotective Effects of CS-PLGA@Au Nanocomposites Against Amyloid-β aggregation and Oxidative Stress in *In Vitro* and *In Vivo* Models of Alzheimer's Disease

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Alzheimer's disease (AD) features a range of pathological hallmarks that are multifactorial, including the buildup of amyloid-β (Aβ) plaques, neuronal cell death, oxidative stress, and dysfunctions in cholinergic and mitochondrial systems. In this study, the curcumin-functionalized chitosan–PLGA nanocomposite embedded with gold nanoparticles (CS-PLGA@AuNCs) was synthesized via co-precipitation to enhance targeted neuroprotection. Detailed physicochemical characterization using FT-IR, XRD, and HRTEM-EDX confirmed the successful creation, structural stability, and consistent morphology of CS-PLGA@AuNCs. In vitro studies using neuronal cell models showed excellent compatibility, effective cellular uptake, and a notable reduction in Aβ protein expression. CS-PLGA@AuNCs significantly reduced neuronal apoptosis and oxidative damage. Confocal microscopy further confirmed the downregulation of both Aβ and tau proteins, suggesting strong potential to alleviate AD-related pathological signaling. To assess in vivo effectiveness, we treated ICV-STZ-induced AD rats with CS-PLGA@AuNCs. Behavioral evaluations indicated enhanced learning and memory, supporting the compound's neuroprotective function in reducing cognitive and synaptic deficits. Biochemical assessments revealed significant decreases in acetylcholinesterase (AChE) and malondialdehyde (MDA) levels, reflecting restored cholinergic function and reduced lipid peroxidation. Additionally, the activities of antioxidant enzymes, including SOD, CAT, and GPx, were substantially increased in the cortex and hippocampus, indicating enhanced internal defense mechanisms. The activities of mitochondrial complexes were also positively adjusted, suggesting improved cellular respiration and diminished mitochondrial dysfunction. Overall, these results underscore CS-PLGA@AuNCs as promising in multifunctional nanotherapy, capable of targeting critical features of AD. The findings support their potential for further preclinical development and eventual clinical application for the management of AD.

sciforum-164342: Omega-3 Supplementation Improves Cortico-Limbic Neuroplasticity and Reduces Amyloid-Related Pathology

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Omega-3 polyunsaturated fatty acids (n-3 PUFA) are crucial for brain health and neuroplasticity. n-3 PUFA deficiency is associated with deficits in mood regulation and memory. This study evaluated the effects of chronic n-3 PUFA supplementation on the prefrontal cortex (PFC) and hippocampus (HIPP) in female rats exposed to a lifelong n-3 PUFA–deficient diet.

Female Wistar rats were exposed to either an n-3 PUFA–poor diet (rich in omega-6) or an n-3 PUFA–enriched diet (rich in α-linolenic acid) from conception until 8 weeks. After 9 weeks, n-3 PUFA supplementation was introduced until 16 weeks. At 16 weeks, neurochemical analyses were conducted in the PFC and HIPP, measuring neurotransmitters (5-HT, DA, NA), neurotrophic factors (BDNF, NGF), synaptic markers (SYN, CAMKII), and amyloid-related markers (amyloid oligomers, APP) using HPLC and Western blotting.

Our results showed that n-3 PUFA supplementation reversed most neurochemical alterations induced by the n-3 PUFA–poor diet in both the PFC and HIPP. Reduced levels of 5-HT and DA in both brain regions under the n-3 PUFA–poor diet were restored to control values following supplementation, while increased NA levels were normalized. SYN expression was reduced in both regions under the n-3 PUFA–poor diet and was restored after supplementation. CAMKII expression was also restored in the PFC, whereas no significant changes were observed in the HIPP. BDNF levels were reduced in both the PFC and HIPP under the n-3 PUFA–poor diet and were fully restored following supplementation. NGF levels were similarly restored in the HIPP, and no significant changes were detected in PFC. Amyloid-related markers showed limited recovery and remained elevated compared to control levels.

We concluded that n-3 PUFA supplementation restores neurotransmitter balance, synaptic function, and neurotrophic support, suggesting its therapeutic potential in neurodegenerative disorders. However, the partial reversal of amyloid-related markers indicates that early-life nutritional deficiency may lead to persistent neurodegenerative vulnerability.

sciforum-161733: Plants with antioxidant potential in the ancestral knowledge of the Mapuche people in Chile: natural alternatives for neurodegenerative diseases

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The ancestral Mapuche people of southern Chile have a rich ethnobotanical tradition based on knowledge of the use of medicinal plants for health care and spiritual balance. Currently, oxidative stress is recognized as one of the mechanisms involved in the pathophysiology of neurodegenerative diseases such as Alzheimer's and Parkinson's, which is why research in the field of medicinal chemistry has focused on finding natural compounds from species with high neuroprotective potential. This paper presents a systematic review with an ethnopharmacological focus on native Chilean plants used by the Mapuche community to treat symptoms compatible with neurodegenerative processes. Based on semi-structured interviews and a review of scientific databases, the species *Aristotelia chilensis, Buddleja globosa, Fuchsia magellanica, Drymis winteri, Peumus boldus*, and *Ugni molinae* were highlighted. These species have been evaluated through the metabolomic characterization of polar and aqueous extracts with high concentrations of polyphenolic compounds and have demonstrated strong antioxidant effects and inhibition of the intracellular production of reactive oxygen species (ROS). Likewise, *Adiantum capillus-veneris, Baccharis tola, Berberis darwinii, Crytocarpa alba, Gevuina avellana, Greigia sphacelata, Lapageria rosea, Laurelia sempervirens, Lophosoria quadripinnata, Otholobium glandulosum*, and *Weinmannia trichosperma* are also reported to have neuroprotective potential. Infusions and decoctions are the main forms of preparation in communities, which is directly related to the study methodologies for their chemical validation. This study contributes to the assessment and relevance of the *Chilean Pharmacopoeia* and the importance of preserving traditional knowledge. Ethnobotany is a source of inspiration and biomedical research in the search for therapeutic alternatives for diseases related to neuronal damage.

sciforum-157250: Proteomic Profiling Reveals Systemic Effects of Peripheral mHtt Expression in Huntington's Disease

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Huntington's Disease (HD) is a neurodegenerative condition triggered by the elongation of a polyglutamine (polyQ) segment located at the N-terminus of the huntingtin protein (HTT). This elongation leads to the aggregation of HTT, which is ubiquitously expressed, suggesting that its aggregation may have effects beyond neurodegeneration to include peripheral tissues. However, the consequences of HTT aggregation in peripheral regions are not as thoroughly understood as those in the central nervous system. In this study, we utilized a Caenorhabditis elegans (*C. elegans*) model of HD, which expresses either a non-pathogenic (15Q) or a pathogenic (128Q) N-terminal fragment of HTT in body-wall muscle cells, to investigate changes in the proteome. We examined four conditions—15Q and 128Q at two different time points (days 2 and 7 of adulthood, labeled as 15D2, 15D7, 128D2, and 128D7). Compared to the 15D2 worms, those with the 128Q expansion at day 2 exhibited reduced levels of ribosomal proteins and cytoskeletal elements such as actin, profilin, calponin, and myosin, alongside an increased expression of galectin, a protein linked to stress and inflammation. By day 7, the 15D7 worms displayed developmental markers indicative of ribosome biogenesis, signal transduction, and vesicle trafficking. Meanwhile, increased levels of proteins related to stress response pathways—including proteostasis, protein folding, and cytoskeletal remodeling—were observed in the 128D7 worms. These findings elucidate the complex, stage-specific disruptions within the HD proteome tied to the expression of the disease in peripheral tissues.

sciforum-152821: Rivastigmine tartrate-loaded functionalized MWNT for the management of alzheimer's disease

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Introduction

Alzheimer's disease (AD) is a chronic neurodegenerative disorder characterized by progressive memory loss, cognitive decline, and behavioral disturbances. Pathological hallmarks include amyloid-β deposition, tau hyperphosphorylation, and oxidative stress. Rivastigmine tartrate, a cholinesterase inhibitor approved for AD management, is typically administered orally but suffers from poor bioavailability due to extensive first-pass metabolism and is further associated with adverse cardiovascular and gastrointestinal effects. To address these limitations, the present study explored the development of an intranasal nanocarrier-based formulation of rivastigmine tartrate using multi-walled carbon nanotubes (MWCNTs) functionalized with carboxyl and polyethylene glycol (MWCNT-COOH-PEG).

Methods

Intranasal administration was chosen to bypass the blood–brain barrier, enable direct nose-to-brain transport, and reduce systemic side effects. Functionalized MWCNTs were selected as drug carriers for their high surface area, biocompatibility, and reported neuroprotective potential. The formulation was optimized using a 3^2 factorial design. Preformulation assessments, including Fourier-transform infrared spectroscopy and differential scanning calorimetry, confirmed drug–excipient compatibility. Critical formulation parameters such as particle size, zeta potential, and entrapment efficiency were studied. In vitro release testing and ex vivo permeation studies using goat nasal mucosa were conducted. Stability studies were performed to evaluate formulation robustness, while in vivo pharmacokinetic and biodistribution assessments were carried out using male Wistar rats.

Results

The optimized intranasal formulation exhibited favorable particle size distribution, surface charge stability, and high drug entrapment efficiency. In vitro release studies revealed a sustained drug release pattern. Ex vivo permeation results demonstrated 56% cumulative drug release at 12 hours and 65.55% at 24 hours across goat nasal mucosa, confirming controlled and prolonged diffusion. Stability studies validated the physical and chemical stability of the formulation. Pharmacokinetic and biodistribution studies indicated improved brain uptake of rivastigmine tartrate compared to conventional administration routes.

Conclusion

The study demonstrated that intranasal delivery of rivastigmine tartrate-loaded MWCNT-COOH-PEG nanocarriers is a promising alternative to conventional oral therapy for Alzheimer's disease.

sciforum-158471: Social Cognition impairments in Huntington's Disease progression

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Beyond its hallmark motor symptoms, Huntington's disease (HD) involves early and progressive disruption of cognitive and psychiatric functions critical for social interaction. Theory of Mind (ToM), the ability to infer one's own and others' mental states, is particularly vulnerable in HD. The present study investigated cognitive and affective ToM across disease stages and their association with neuropsychiatric symptoms.

A total of 24 HD patients (12 stage I, 12 stage II) and 24 healthy controls completed the Yoni Task, assessing cognitive and affective ToM. Neuropsychiatric symptoms were evaluated using the Problem Behaviors Assessment—short version, and quality of life with the 12-item Short Form Health Survey.

Cognitive ToM was significantly impaired already in stage I HD compared to controls (mean accuracy: 68.5% vs. 87.6%, p = 0.004), while affective ToM was relatively preserved (76.4% vs. 86.2%, p = 0.095). Stage II patients showed marked deficits in both domains (cognitive ToM: 46.3%; affective ToM: 52.3%), performing significantly worse than controls (p 0.001). Total ToM performance declined progressively across stages (p 0.001). Lower affective ToM scores were associated with greater irritability/aggression (ρ = –0.40, p = 0.049), obsessive–compulsive symptoms (ρ = –0.51, p = 0.015), and poorer self-reported physical functioning (ρ = 0.42, p = 0.049).

ToM impairments in HD are stage-dependent, with early cognitive deficits and later involvement of affective processes. Decline in ToM performance is clinically meaningful and associated with neuropsychiatric symptoms, particularly irritability. ToM assessment may therefore provide a sensitive marker of socio-cognitive dysfunction and disease progression in HD.

sciforum-156148: Targeting Neurotrophin Regulation by Polyphenols: Mechanistic Basis for Cognitive Resilience

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Background:

Synaptic plasticity in neurodegenerative disorders (NDs), cognitive impairment, and mental health conditions is regulated by brain-derived neurotrophic factor (BDNF). Even healthy individuals have different levels, which are affected by complex epigenetic, inflammatory, and metabolic regulation. BDNF expression changes are associated with both typical and abnormal aging, as well as mental health conditions. These changes affect brain areas that are crucial for memory, such as the hippocampus and the parahippocampal cortex. Neurotrophins (NTs), including nerve growth factor (NGF) and BDNF, are essential for neuronal differentiation via tropomyosin receptor kinase (Trk) and the p75 neurotrophin receptor (p75NTR). Dysregulated NT signaling contributes to synaptic dysfunction and neuroinflammation.

Objective:

This systematic review synthesizes preclinical evidence of the potential of naturally derived compounds to modulate NTs for neuroprotection and their incorporation into novel foods.

Methodology:

A review of major databases found studies that examined the impact of dietary polyphenols and other bioactive substances on NT signaling, oxidative stress, inflammation, and neuronal plasticity.

Results:

Compounds such as epigallocatechin gallate, resveratrol, curcumin, quercetin, and flavanols can positively impact NTs, reducing reactive oxygen species (ROS)/reactive nitrogen species (RNS), enhancing cell survival, and increasing the expression of trophic factors such as Nrf2, NGF, and vascular endothelial growth factor (VEGF) in neural stem cells. However, their bioavailability, optimal dosage, and dietary interactions require further research.

Conclusions:

The consumption of BDNF-promoting foods can potentially stimulate BDNF synthesis, support optimal neurotransmission, and fortify neural plasticity. Evidence supports a polyphenol-rich diet for preventing NDs and promoting brain health. Observational studies consistently support the protective effects of polyphenols on brain health through their impact on the gut–brain axis.

sciforum-154890: The Cerebrocortical Disconnection Hypothesis: Reframing Alzheimer's Treatment Strategy

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Alzheimer's disease (AD), the most common cause of dementia, is defined by amyloid plaques, neurofibrillary tangles (NFTs) of hyperphosphorylated tau, and neuronal loss. The traditional **amyloid cascade hypothesis** long held that amyloid-β (Aβ) accumulation is the primary instigating event, supported by early genetic evidence linking AD to mutations in Aβ-processing genes (APP, PSEN1/2).

However, this paradigm is challenged by clinical trial results. Approved anti-Aβ immunotherapies, despite significantly reducing plaque levels, confer only **modest clinical benefits**, slowing cognitive decline slightly. Tau-based therapies have also failed, though they have not yet successfully cleared NFTs from the perikarya of cortical pyramidal neurons.

This evidence supports a proposed shift to the **cerebrocortical disconnection hypothesis**. This theory posits that dementia results from the breakdown of neural networks, initiated when **tau aggregates into NFTs**. This disruption damages axonal transport, creating an **energetic crisis** at nerve terminals, which promotes the production of Aβ42 and subsequent amyloid plaque formation.

The neural breakdown is further worsened by the dysfunction and loss of myelin-maintaining oligodendrocytes, leading to **demyelination**. This forces the damaged axons to expend even more energy for signalling, severely exacerbating their bioenergetic deficit. This combination of faulty transport, amyloid pathology, and myelin loss ultimately destroys critical long corticocortical and corticofugal neurons, along with (cholinergic, noradrenergic and serotonergic) corticopetal neurons. We conclude that therapeutic focus must now shift toward protecting the **integrity of neural circuitry** directly to prevent disconnection and meaningfully slow cognitive decline.

sciforum-158137: Toward Reproducibility in Preclinical Alzheimer's Research: The Case for Standardizing $A\beta_{1-42}$-Induced Rodent Models

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Introduction: Animal-induced models are necessary to bridge the translational gap between transgenic models and clinical trials. Although several models are standard, experimental designs differ, and protocols are not standardized.

Methods: A PRISMA-guided search was performed in PubMed, Embase, and Cochrane, and 60 studies with Aβ1-42 injected into rats were included. Primary outcomes were rat strain, sex, injection site, and dose. Secondary outcomes followed the time between induction and cognitive testing. Data was analyzed using JASP statistics program and applying descriptive statistics, t-tests, chi-square, and correlations.

Results: Bilateral injections showed up in 65% of the studies, most often in Wistar rats (64%), and hit either the intracerebroventricular (i.c.v., 48.7%) or intrahippocampal (i.h.c., 51.3%) regions. Most unilateral models—85.7%—used i.c.v. injections. On average, researchers injected 3.8 ± 2.1 µl in bilateral setups and 4.9 ± 2.6 µl in unilateral ones. They ran behavior tests about 18.4 ± 10.6 days after bilateral and 20.7 ± 12.9 days after unilateral injections. Statistically, those differences were not significant ($p > 0.05$).

Conclusions: The field still lacks a standard way to run $A\beta_{1-42}$-based Alzheimer's models. Even when timing and doses line up, choices around animal strain, injection site, and laterality vary widely. Establishing standardized reference parameters—dose, site, and timing—appears critical to enhance reproducibility and strengthen the translational validity of preclinical Alzheimer's research.

Session 2. Cellular and Molecular Neuroscience

sciforum-157101: *Salvia palaestina* Essential Oil as a Molecular Modulator of AMPA Receptors

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Introduction Phytochemicals, particularly those from Salvia species, exhibit notable neuroprotective effects by modulating neurotransmission and limiting excitotoxic damage. Since excessive glutamate release and overactivation of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors contribute to neuronal injury, targeting these receptors presents a promising molecular approach. This study investigates the modulatory effects of *Salvia palaestina* essential oil on AMPA receptor subunits to elucidate its potential role in regulating excitatory synaptic signaling.

Methods Human embryonic kidney 293T cells were transiently transfected with the DNA of four AMPA receptor subunits (GluA1, GluA2, GluA1/2, and GluA2/3) using jetPRIME. Enhanced green fluorescent protein aided expression tracking. After 36 hours, fluorescence-sorted cells were plated for whole-cell patch-clamp recordings. Currents were evoked by 10 mM glutamate with or without *Salvia palaestina* essential oil, and whole-cell current responses along with deactivation and desensitization kinetics were analyzed using SutterPatch and Igor Pro7 software.

Results Whole-cell recordings revealed that *Salvia palaestina* essential oil markedly inhibited AMPA receptor activity across all tested subunits. Current amplitudes were reduced approximately 5-fold in GluA1, GluA1/2, and GluA2/3 and 6-fold in GluA2. The oil also altered receptor kinetics, increasing deactivation rates nearly 3-fold in all subunits while slowing desensitization 4-fold in GluA2, 3-fold in GluA2/3, 2.2-fold in GluA1, and 1.4-fold in GluA1/2, indicating strong modulatory effects on receptor function.

Conclusions *Salvia palaestina* essential oil strongly modulates AMPA receptor function by reducing whole-cell currents and altering deactivation and desensitization kinetics. These effects suggest anti-excitotoxic potential, likely mediated by major constituents such as carvacrol, eucalyptol, and thujones, supporting its role in regulating glutamatergic transmission and synaptic activity at the molecular level.

sciforum-158005: A 100-Day Study of Biochemical, Behavioral, and Cognitive Changes Associated with Hippocampal and Prefrontal Cortex Alterations in Experimental Type 1 and Type 2 Diabetes

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Despite growing evidence linking diabetes to brain dysfunction, the specific long-term effects of type 1 (T1D) and type 2 diabetes (T2D) on hippocampal and prefrontal cortex (PFC) function remain poorly characterized. This study aimed to provide a comprehensive comparison of the chronic neurobiological, cognitive, and behavioral consequences of sustained hyperglycemia in experimental models of T1D and T2D. By integrating behavioral assessments with biochemical and neurochemical analyses, we sought to delineate diabetes type-specific patterns of dysfunction within the hippocampus and PFC. Adult rats were randomly divided into three groups: Sham, T1D, and T2D. T1D was induced by a single intraperitoneal injection of streptozotocin (STZ), while T2D was established via nicotinamide (NA) administration 15 minutes prior to STZ injection. Behavioral and cognitive assessments were conducted during the final phase of the 100-day experimental period. Following testing, blood samples were collected for biochemical analyses, and the hippocampus and PFC were dissected to assess oxidative stress markers, inflammatory mediators, acetylcholinesterase (AChE) activity, BDNF levels, and Na^+/K^+-ATPase activity. Histological examinations using Nissl staining were performed to evaluate neuronal integrity. After 100 days of hyperglycemia, both diabetic models exhibited significant functional and structural alterations in the hippocampus and PFC. T2D was associated with pronounced oxidative stress and inflammation, correlating with anxiety- and depression-like behaviors (P 0.05). Conversely, T1D rats displayed more extensive cognitive impairment, along with severe neurochemical and structural disruptions, including marked BDNF depletion, significant Na^+/K^+-ATPase reduction, and elevated AChE activity (P 0.05), indicative of greater neuronal stress and degeneration. These findings underscore diabetic encephalopathy as a multifactorial disorder involving interrelated deficits in neurotrophic support, metabolic regulation, and neurotransmitter balance. While T2D is characterized predominantly by oxidative and inflammatory stress, T1D exerts more profound neurochemical and structural damage within the hippocampus and prefrontal cortex.

sciforum-154966: Analyzing the Spatiotemporal Regulatory Mechanism of Ferroptosis-Related GPX4 in TBI Based on Single-Cell and Spatial Transcriptome Sequencing

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Introduction: TBI is a major global public health issue, and its secondary injury involves multiple pathological processes. In recent years, ferroptosis has attracted increasing attention for its role in TBI. This study aims to systematically analyze the spatiotemporal regulatory mechanism of GPX4 in TBI using single-cell sequencing and spatial transcriptome sequencing technologies.

Methods: Sequencing was performed using the 10×Genomics and Visium platforms. The data were integrated, and the expression of GPX4 was analyzed in combination with ferroptosis-related databases. At the cellular level: Primary neurons were cultured; GPX4 expression was regulated using siRNA and lentivirus; ferroptosis was induced by Erastin or RSL3; and cell viability and ferroptosis markers were detected. At the animal level: AAV was injected via stereotaxis to regulate GPX4 in the injured area, followed by behavioral evaluations and histological analyses. Co-IP was used to explore the molecular interaction mechanism of GPX4.

Results: Single-cell sequencing identified dynamic changes in cell subsets after TBI, and differential expression of ferroptosis-related genes was observed in all subsets. Spatial transcriptome sequencing revealed the specific distribution of these genes in the injured area. Integrated analysis showed that GPX4 expression changed significantly in the injured area, with obvious spatiotemporal specificity in neurons. Cellular experiments confirmed that GPX4 silencing exacerbated damage caused by ferroptosis inducers and increased lipid peroxidation levels, while GPX4 overexpression alleviated ferroptosis. Animal experiments indicated that regulating GPX4 significantly affected the recovery of neurological function and cognitive ability in TBI mice.

Conclusion: This study is the first to integrate the two sequencing technologies, revealing the spatiotemporal regulatory pattern of GPX4 in TBI and its cell-specific role. It confirms that GPX4 plays an important role in TBI-induced secondary injury, affects ferroptosis by regulating iron metabolism, and provides a new approach for TBI treatment—with significant theoretical and clinical value.

sciforum-164203: Computational Modeling and Molecular Dynamics of Nonsynonymous *CSDE1* Variants Associated with Autism Spectrum Disorder

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Background: Autism Spectrum Disorder is a complex neurodevelopmental condition with a strong genetic basis, yet the molecular mechanisms by which many risk-associated variants disrupt neuronal function remain poorly understood. *CSDE1*, a post-transcriptional regulator implicated in neurodevelopment, has recently emerged as a candidate susceptibility gene in Autism Spectrum Disorder, although the structural and functional consequences of its coding variants remain largely unexplored.

Objective: This study aimed to systematically characterize the structural and dynamic effects of nonsynonymous single-nucleotide polymorphisms in *CSDE1* associated with Autism Spectrum Disorder using an integrative computational framework.

Methods: A comprehensive in silico pipeline was employed to prioritize potentially deleterious *CSDE1* variants using sequence- and structure-based prediction tools, including SIFT, PolyPhen-2, MutPred2, I-Mutant 2.0, MUpro, DynaMut, and ConSurf. The *CSDE1* protein structure was obtained from the AlphaFold Protein Structure Database, refined using GalaxyRefine, and validated with MolProbity. Functionally relevant domains harboring high-confidence variants were subjected to all-atom molecular dynamics simulations. Structural dynamics were evaluated over 100 ns simulation trajectories using root mean square deviation, root mean square fluctuation, radius of gyration, hydrogen bond occupancy, and solvent-accessible surface area.

Results: Comparative analyses between wild-type and mutant *CSDE1* proteins revealed pronounced conformational instability, altered residue flexibility, disrupted compactness, and changes in intramolecular interaction networks in selected variants. These perturbations suggest compromised structural integrity and functional dynamics of *CSDE1*, supporting their potential pathogenic relevance in Autism Spectrum Disorder.

Conclusion: This study provides the first detailed molecular-level characterization of Autism Spectrum Disorder-associated *CSDE1* nonsynonymous variants using computational modeling and molecular dynamics simulations. The findings highlight candidate variants with functional relevance and establish a framework for future experimental validation, offering insights into the mechanistic role of *CSDE1* in neurodevelopmental dysfunction and its potential utility as a molecular biomarker.

sciforum-164210: Concomitance of endothelium disorders in the brain and heart vessels in ischemic heart disease

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Introduction. Endothelial dysfunction (ED) is a key pathogenetic mechanism underlying cardiovascular and cerebrovascular diseases and is increasingly recognized as a common link between ischemic heart pathology and cerebral vascular impairment. In addition to postnatal risk factors, adverse intrauterine conditions, particularly prenatal hypoxia (PH), may program long-term endothelial alterations. The aim of this study was to investigate the structural and molecular features of ED in the myocardial and cerebral vessels in experimental chronic heart failure (CHF) and PH, as well as to evaluate the endothelioprotective potential of pharmacological agents targeting the nitric oxide system.

Methods. This study was conducted on Wistar rats using experimental models of CHF (doxorubicin administration, cumulative dose 15 mg/kg) and PH (sodium nitrite 50 mg/kg administered to pregnant females on gestational days 16–21). The endothelial status of cerebral and myocardial vessels was assessed using immunohistochemistry, ELISA, morphometric analysis, and real-time PCR. Key markers of endothelial function, inflammation, nitric oxide metabolism, oxidative stress, and angiogenesis were evaluated. The endothelioprotective potential of nitric oxide-modulating pharmacological agents was also evaluated.

Results. CHF and PH induced pronounced structural and functional endothelial alterations in the microcirculatory and muscular-type vessels of the heart and brain. These changes were characterized by reduced endothelial cell density, suppressed eNOS expression, increased iNOS expression, nitric oxide deficiency, elevated nitrotyrosine levels, and activation of proinflammatory cytokines. VEGF levels were significantly decreased, while apoptotic features of endothelial cells were intensified. Angiolin and Hypertril demonstrated the most pronounced endothelioprotective effects among the tested agents.

Conclusions. CHF and PH induce persistent ED in cerebral and myocardial vessels through disruption of the nitric oxide system, oxidative stress, and inflammatory activation. PH may act as an early trigger increasing susceptibility to cardiovascular and cerebrovascular diseases later in life. These findings support the rationale for targeted endothelioprotective therapy in ischemic cardiovascular pathology.

sciforum-161399: Cytoprotective Epigenetic and DNA-Damage Repair Molecular Mechanisms of α-Lipoic Acid (ALA) Against Carbon Tetrachloride Induced Hepatotoxicity and Neurotoxicity in Rats

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Introduction: Carbon tetrachloride (CCl_4), is industrial solvent, hose living close to textile and chemical factories are exposed to CCl_4 and are at great risk. The toxic effect of CCl_4 is believed to be due to trichloromethyl radicals ($CCl_3^{\bullet}$) which causes extensive membrane molecular and mitochondrial damages, and ultimately cell death by necrosis and ammonia toxicity. Many common medications cause hepatotoxicity and neurotoxic chemicals including ethanol also cause OS in the brain.

Aim: The aim of this present study was to determine whether administration of α-lipoic acid, a potent iron-chelating antioxidant ergogenic aid could decrease CCl_4-induced hepatotoxicity and neurotoxicity.

Materials & Methods: Male albino rats (4-months old, 250 gm body wt) were pretreated with α-lipoic acid (Started 3-days prior to CCl_4 administration) (ALA:100mg/Kg b.wt, orally via an oral cannula for 11 days), followed by oral ingestion with the CCl_4 (CCl_4 in Olive oil, 1.6 mL/kg b.wt). At the end of the 11-days, the animals were sacrificed and biological samples were collected for the estimation of biomarkers of liver dysfunction, biomarkers of OS and inflammation in the brain.

Results: Application of α-lipoic acid mitigated the toxic onslaught of CCl_4-induced-toxicity and exerted hepatoprotective effects by significantly (p0.001) reducing SGOT, SGPT, ɣ-GT, bilirubin, MDA, 8- OHdG, IL-6, TNF-α, XO, NOS, NADPH-oxidase and augmentation of serum total protein, A/G ratio. Further, ALA protects the liver and brain by increasing antioxidants such as GSH, SOD, Catalase, GST, GPx, GR, G6PD, spares ATP, DNA and modulation of PARP-1.

Conclusion: Knowledge of the epigenetic and molecular mechanisms involved in the CCl_4-induced hepatotoxicity in this model is the key to identifying potential therapeutic targets for liver dysfunction associated with a variety of hepatic disorders including alcoholic cirrhosis and brain dysfunction due to ammonia toxicity.

sciforum-161119: Genome-Wide Variant Associations and Biological Pathways in Postherpetic Neuralgia

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Introduction: Postherpetic neuralgia (PHN) is the most frequent chronic complication of herpes zoster and is characterized by persistent neuropathic pain caused by sensory nerve injury and neuroinflammatory responses. Although clinical risk factors are well documented, the genetic architecture underlying PHN susceptibility remains poorly defined. Genome-wide association studies (GWASs) represent a powerful tool to identify common variants that may contribute to neuronal vulnerability, persistent inflammation, or dysregulated pain signaling.

Methods: We performed a secondary analysis of publicly available summary statistics from the GWAS Catalog (GCST012124), derived from a genome-wide association study conducted in individuals with PHN. The dataset included all reported SNP-level associations for the trait MONDO_0041052. Variants were evaluated based on reported effect allele, beta coefficients, p-values, genomic context, and nearest-gene annotation. Biological interpretation was performed through gene-function review and known pathways implicated in neuropathic pain.

Results: Five genome-wide significant or near-significant loci were identified. The strongest associations were rs4773840 mapped to KIF1B (β = 0.18, p = 3.8×10^{-8}) and rs10982356 near PTPRZ1 (β = 0.17, p = 7.6×10^{-8}), both genes involved in axonal transport, neuronal signaling, and glial modulation. Additional variants included rs9655009 within PRKCE (p = 4×10^{-7}), a key kinase in nociceptive sensitization, rs10887816 near CXCR4, implicated in neuroinflammation, and rs17057519 in a regulatory non-coding region (WDR11-AS1). These loci converge on pathways relevant to neuronal injury, synaptic modulation, and chronic pain maintenance.

Conclusions: This GWAS highlights candidate genetic factors that may increase susceptibility to PHN by altering axonal integrity, inflammatory responses, and pain-signaling pathways. The findings support a multifactorial neurobiological basis for PHN and provide mechanistic targets for future translational and therapeutic research.

sciforum-164359: Metal-dependent neural protection by HP-derived coordination compounds against mitochondrial dysfunction in glial cells

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Coordination compounds are promising redox-active systems for applications across multiple fields, including neuroscience; however, their effects on mitochondrial function in neural cells remain poorly investigated. Here, we evaluated the neuroprotective potential of Cu^{II}, Fe^{III}, and Mn^{II} coordination compounds derived from the ligand bis(pyridin-2-ylmethylamine) -3-chloropropan-2-ol (HP), targeting mitochondrial dysfunction and oxidative stress in C6 glial cells. A system with impaired mitochondrial activity was elicited by exposing C6 glial cells to rotenone (50 µM; 1 h). The treatment severely impaired mitochondrial respiration by reducing basal oxygen consumption from 55 ± 13.2 to 14 ± 7.2 pmol O_2 s^{-1} $1x10^4$ cells and maximal respiration from 123 ± 8.2 to 35 ± 3.5 pmol O_2 s^{-1} $1x10^4$ cells. CuHP, FeHP, and MnHP treatment (after rotenone) restored basal respiration (56 ± 8.9, 50 ± 7.5, and 57 ± 3.8 pmol O_2 s^{-1} $1x10^4$ cells, respectively) and maximal respiration (134 ± 6.8, 118 ± 5.2, and 152 ± 3.2 pmol O_2 s^{-1} $1x10^4$ cells), comparable to the SOD mimetic EUK-8 (148 ± 8.2 pmol O_2 s^{-1} $1x10^4$ cells). Exposure to the HP series (after rotenone; 1 h) inhibited ROS generation at 3–30 µM in rotenone-induced cytotoxicity, with FeHP showing consistent suppression across all concentrations and superior performance to EUK-8. At 100 µM, CuHP and MnHP reduced ROS generation by approximately 80%, whereas the reference compound EUK-8 achieved only 60% inhibition. HP coordination compounds showed significantly greater efficacy than EUK-8 in suppressing rotenone-induced ROS production. The HP series effectively counteracted rotenone-induced mitochondrial dysfunction and oxidative stress in C6 glial cells. The recovery of mitochondrial respiration and ROS inhibition demonstrated that the neuroprotective effects were dependent on the nature of the central metal ion (MnHP > FeHP > CuHP). Thus, coordination compounds can modulate mitochondrial bioenergetics and redox balance, supporting their potential as mitochondria-targeted neuroprotective agents.

sciforum-158194: Modeling the blood–brain barrier under neuroinflammation using a BBB-on-a-Chip

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Neuroinflammation plays a crucial role in the development and progression of various neurodegenerative diseases. Therapeutic strategies aimed at protecting physiological functions of brain endothelial cells are being explored as potential treatments for these conditions. It is necessary to have experimental models that accurately represent the effects of neuroinflammation, as existing models are often not sufficiently representative of human physiology and the specific challenges of central nervous system diseases with a vascular pathology. Considering the need for a more accurate simulation strategy of the selective properties and limitations of the blood-brain barrier (BBB), we established a BBB-on-a-chip model, which consists of a co-culture of human stem cell-based brain endothelial cells and brain pericytes, to study how BBB permeability is altered under pathological conditions and to discover protective compounds to counteract BBB injury. Our preliminary results showed that the thrombin-fibrinogen interaction-based hydrogel self-assembled model holds great potential for use in preclinical studies, as it was possible to mimic BBB properties in vitro. Brain endothelial cells formed an established vascular network visualized by PECAM staining along with a positive signal for α-smooth muscle actin for pericytes. Modeling BBB injury with pro-inflammatory cytokines to promote neuroinflammation was successful with a lower calcein AM signal and higher positivity for propidium iodide. This setup will be used to test novel ways to improve BBB functions under neuroinflammation. In the future we aim to identify and optimize new therapeutic compounds before testing them on animal or human models.

sciforum-154785: Neuroinflammatory Modulation of Synaptic Plasticity: Unraveling Glia–Neuron Crosstalk through Multi-Scale Molecular Profiling

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Synaptic plasticity forms the cellular foundation of learning and memory, yet its regulation under neuroinflammatory conditions remains incompletely understood. Growing evidence suggests that immune-derived cytokines dynamically influence neuronal structure and function through bidirectional communication between glial and neuronal networks.

In this study, we sought to elucidate the molecular architecture of **glia–neuron crosstalk** that shapes synaptic remodeling during inflammation. Using **human cortical organoids** and **murine hippocampal slice cultures**, we induced controlled inflammatory states via interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α) exposure. We employed **single-cell RNA sequencing**, **quantitative proteomics**, and **super-resolution imaging** to map cell-type-specific transcriptional and structural responses. Gene-regulatory network reconstruction identified a cascade centered on **NF-κB, complement C3, and PGC-1α**, linking immune activation to metabolic reprogramming and synaptic pruning.

Functional validation using **CRISPR interference** and pharmacological inhibition confirmed that NF-κB blockade restored dendritic spine density, enhanced long-term potentiation (LTP), and rescued mitochondrial integrity. Conversely, astrocytic overexpression of C3 exacerbated synaptic loss, demonstrating a direct glial contribution to excitatory circuit instability. Mitochondrial respiration assays further revealed that inflammatory signaling suppresses oxidative phosphorylation while increasing glycolytic flux, coupling **energy dysregulation with synaptic impairment**.

Collectively, these data delineate a multi-layered signaling network whereby neuroinflammation modulates synaptic plasticity through transcriptional, metabolic, and structural mechanisms. Targeting the NF-κB–PGC-1α axis and glial complement pathways may therefore offer new strategies to restore circuit homeostasis in neurodegenerative and psychiatric disorders.

sciforum-158443: Neuroprotective Effect of *Centella Asiatica* Extract on Experimentally Induced Brain Ischemia in Aged Rats

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Centella asiatica (CA), a well known Ayurvedic medicine, enriched with bioflavonoids is believed to have beneficial effects in improving learning and memory in a variety of neurodegenerative diseases including Alzheimer's disease, Parkinsonism, multiple sclerosis and senile dementia. It also exerts a potent antioxidant-iron chelating and neuroprotective effects and enhances cognition in a variety of clinical condditions. Acute ischemia (AI) followed by reperfusion is known to bring about a major biochemical and behavioral alterations. In the present study the effect of *Centella asiatica* extract (CAE) on acute cerebral reperfusion (ACR) and long-term cerebral hypoperfusion (LTCH) in aged rats (Body wt 300g, 28-month old). was investigated. Transient brain ischemia (TBI) was induced under anaesthesia by clamping bilateral common carotid arteries (BCCAO) for 30 min and then reperfusion was allowed for 60 mins. The biomarkers of oxidative stress like malondialdehyde (MDA), superoxide dismutase (SOD), proinflammatory cytokines (PICKs), DNA content, 8-OHdG, nitric oxide synthase (NOS), poly(ADP-ribose polymerase)-1 and relevant biomarkers and behavioral and biochemical studies were estimated. In the present investigation, acute ischemia-reperfusion (AI-R) induced increases all the biomarkers of oxidative stress, PICKs, oxidative DNA-damage, DNA- repair enzymes such as NOS, PARP-1. CAE pre-administration (300 mg/kg body wt, p.o. for 7 days, CAE administration started 3-days prior to occlusion) ameliorated the reperfusion induced biochemical alterations in aged rats. Long-term cerebral hypoperfusion in rats caused a propensity towards anxiety and listlessness accompanied by deficits in learning and memory confirmed by related tests). Further, CAE treatment (200 mg/kg body wt, p.o. for 1 month) alleviated these behavioral, cognitive and biochemical alterations significantly. The results suggest that CAE enriched with bioflavonoids and tritripenoids may be useful in cerebrovascular insufficiency conditions associated with normal aging, hypertension and brain stroke.

sciforum-155378: Neuroprotective Pathways and Synaptic Restoration: Molecular Insights into Neuron–Glia Crosstalk and Oxidative Stress Modulation

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The dynamic communication between neurons and glial cells forms the molecular foundation of brain homeostasis, synaptic maintenance, and resilience to injury. Disturbances in this neuron–glia dialogue underlie many neurodegenerative disorders, where oxidative stress, mitochondrial dysfunction, and neuroinflammation converge to promote neuronal loss. The present study investigates the cellular and molecular mechanisms governing neuron–glia crosstalk under oxidative challenge, focusing on Nrf2-mediated antioxidant signaling, mitochondrial dynamics, and synaptic restoration.

Primary cortical neuron–astrocyte co-cultures were subjected to controlled oxidative stress using hydrogen peroxide (H_2O_2, 100 µM) for 24 hours, followed by treatment with Nrf2 activators (sulforaphane and curcumin). Cellular responses were assessed through RT-qPCR, Western blotting, confocal immunofluorescence, and targeted proteomic analysis. Oxidative insult significantly reduced neuronal viability (by 43%), disrupted mitochondrial membrane potential, and decreased expression of synaptic proteins (PSD-95, synaptophysin). Post-treatment, a robust activation of the Nrf2–ARE pathway was observed, with a 3–5-fold increase in antioxidant gene expression (HO-1, NQO1, and SOD2) and partial recovery of synaptic protein levels. Astrocyte-conditioned media from treated cultures enhanced neuronal survival by upregulating glutamate transporters (EAAT1/2) and reducing lipid peroxidation markers (MDA, 42% reduction). Furthermore, transmission electron microscopy revealed restoration of mitochondrial integrity and reduction in reactive astrocytosis.

These findings demonstrate that activation of Nrf2 signaling and glial antioxidant responses confer significant neuroprotection against oxidative damage. The study emphasizes the crucial role of astrocytes as metabolic and redox regulators, capable of restoring neuronal function and synaptic plasticity. Targeting the neuron–glia metabolic axis thus represents a promising therapeutic approach to mitigate oxidative neurotoxicity and promote neuroregeneration in disorders such as Alzheimer's disease, Parkinson's disease, and cerebral ischemia.

sciforum-163640: Quercetin suppresses mRNA Expression of Fto and the TNF-α/NF-κB/NLRP3 Inflammasome Pathway in Hypothalamus of Diet-Induced Obese Rats

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Introduction

The NLRP3 inflammasome plays a significant role in the pathogenesis of obesity by contributing to the development of chronic low-grade inflammation, systemic inflammation, and, consequently, neuroinflammation.

Quercetin is a flavonoid which possesses anti-inflammatory properties, therefore representing a potential modulator of neuroinflammatory process, metabolic regulators such as the fat mass obesity-associated gene (Fto) and TNF-α/NF-κB/NLRP3 Inflammasome pathway genes.

Studies in animal models have shown that quercetin exerts a wide range of effects over inflammatory processes, suggesting potential therapeutical applications. Fto is a well-known gene associated with obesity risk, body mass index, energy homeostasis and hypothalamic control of appetite and satiety. Altered Fto expression influences metabolic and inflammatory responses and plays a relevant role in inflammatory-related diseases.

We hypothesized that quercetin inhibits overexpression of Fto and the activation of the TNF-α/NF-κB/NLRP3 Inflammasome pathway in the hypothalamus of high-fat-diet Wistar rats.

Objectives

To evaluate mRNA expression of Fto and components of the TNF-α/NF-κB/NLRP3 inflammasome pathway in the hypothalamus of high-fat diet-fed Wistar rats by quantitative PCR (qPCR).

Methodology

After 2 weeks of acclimation, three groups of Wistar rats (200 ± 20 g) were randomly assigned to three groups (n = 6 each group): (a) standard diet (SD), (b) high-fat diet (HD), and (c) high-fat diet with quercetin (HD+Q). Quercetin was administered intragastrically at a dose of 50 mg/kg/day for 12 weeks.

At the end of the intervention period, rats were euthanized and the hypothalamus was collected. Total RNA was isolated using the TriZol method, purified with isopropanol and 75% ethanol, quantified, and its purity verified by nanodrop 2000 spectrophotometer.

cDNA was synthetized from 100 ng of total RNA using random primers. The mRNA expressions of Fto, Tnf, Tnfrs1a, Tradd, Ripk1, Map3k7, Ikbkb, Ikbkg, Nek7, Nfkbia, Nlrp3, Pycard, Casp1, Il1b and Il18 through qPCR. B-Actin were used as housekeeping genes.

Results

The HD+Q group showed a significant decrease in the mRNA expression of Fto and pro-inflammatory genes involved in the TNF-α/NF-κB/NLRP3 inflammasome pathway compared with the HD group. In contrast, the HD group showed a significant increase in the expression of these genes relative to the SD group.

Conclusions

Quercetin downregulates mRNA expression of Fto and key components of the TNF-α/NF-κB/NLRP3 inflammasome pathway in the hypothalamus of high-fat-diet-fed Wistar rats. These findings suggest that quercetin may represent a promising therapeutic candidate for targeting obesity-associated neuroinflammation.

sciforum-160851: Response of microglia and neural cells in the ICR mouse brain to experimental reserpine therapy due to traumatic brain injury

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Introduction: Neuroinflammation is the main factor of secondary brain injury following traumatic brain injury (TBI). At the same time, neural cells are most sensitive to damaging factors. The effectiveness of therapeutic approaches to treat neuroinflammation is limited. This study aims to identify new strategies for regulating neuroinflammation in TBI.

Methods: The effect of experimental therapy with sympatholytic reserpine on the expression of M1 and M2 microglial markers (Iba1, iNOS, CD206), the mature neuron marker (NeuN), and the expression of the apoptotic marker Caspase-3 by neurons and microglia in the motor cortex and subventricular zone (SVZ) of ICR mice with TBI was studied using the immunohistochemical method.

Results: Reserpine reduced the number of $Iba1^+$ microglia, including polarized M1 microglia, and $Caspase\text{-}3^+$ microglia in the motor cortex and SVZ of mice with TBI. Reserpine also reduced the number of polarized M2 microglia in the SVZ. However, experimental therapy did not affect the content of $NeuN^+$ cells, including $NeuN^+Caspase\text{-}3^+$ cells, in the motor cortex and SVZ of mice with TBI.

Conclusion: Sympatholytic reserpine may be a promising compound for developing a new approach to combating neuroinflammation in TBI. We believe that additional administration of drugs that stimulate the differentiation of neural progenitor cells may promote nerve cell regeneration.

sciforum-164180: Targeting Neuroinflammation in Alzheimer's Disease with a Nicotinic Metabolite: Insights from the 5xFAD Mouse Model

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Introduction: Alzheimer's disease (AD) is characterized by progressive cognitive decline and is largely driven by amyloid-β aggregation, tau hyperphosphorylation and widespread synaptic dysfunction. Chronic neuroinflammation, associated with altered cytokine signalling, plays a significant role in the progression of AD. Although nicotine has been shown to influence inflammatory pathways, its therapeutic use is constrained by adverse effects. 6-hydroxy-L-nicotine (6HLN), a nicotine metabolite, may exhibit anti-inflammatory activity while offering a more favorable safety profile. In this context, the present study examines the effects of 6HLN on specific molecular markers of inflammatory cytokine signalling to assess its potential biological relevance in AD-associated pathology.

Methods: The effects of chronic 6HLN treatment were evaluated in a 5xFAD transgenic mouse model of AD. 5xFAD mice underwent chronic intraperitoneal administration of 6HLN at two doses (0.3 and 0.6mg/kg for 30 days). Key inflammatory mediators (NF-κB, IL-6 and TNF-α) were quantified from brain homogenates using ELISA.

Results: 6HLN induced a dose-dependent reduction in NF-κB, IL-6 and TNF-α levels, indicating a significant attenuation of pro-inflammatory cytokine response in 5xFAD mice.

Conclusion: These findings suggest that 6HLN may serve as a promising anti-inflammatory candidate for mitigating inflammation-related pathological processes in AD. This work was supported by CNCSIS-UEFISCSU, project number PN-III-P4-PCE-2021-1692.

Session 3. Behavioral Neuroscience

sciforum-154721: *Chrysophyllum albidum* G.don Pectin attenuated colitis-associated neurobehavioral deficits and inflammation in dextran sodium sulphate-induced colitis in high-fat diet fed Swiss mice

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Inflammatory bowel disease (IBD) is a chronic inflammatory condition linked to dysregulated immune responses, gut barrier dysfunction, and oxidative stress. The African star apple, *Chrysophyllum albidum* peel is rich in pectin, a bioactive polysaccharide with potential anti-inflammatory and antioxidant properties, but its therapeutic role in IBD remains unexplored. This study investigated the effects of *C. albidum* pectin (CAP) on colitis and associated neurobehavioral and biochemical alterations in a mouse model of dextran sodium sulphate (DSS)-induced colitis in HFD-fed mice. Mice were fed either a normal diet (ND) or HFD with colitis induced by DSS administration. Treatment groups received CAP at doses of 250 and 500 mg/kg. Disease progression was assessed by the Disease Activity Index (DAI), behavioral tests for anxiety and pain sensitivity, and histological examination of colon and brain tissues. Pro-inflammatory cytokines (TNF-α, IL-6), oxidative stress markers (MDA, nitrites), antioxidant enzymes (GSH, CAT, SOD, GST), apoptotic markers (MMP-9, caspase-9), and neurotransmitter-related enzymes were analysed in the colon and brain tissues. The HFD and DSS co-treatment aggravated colitis severity, increasing DAI, pro-inflammatory cytokines, oxidative stress, apoptosis, and neurobehavioral deficits such as anxiety and hyperalgesia. The CAP treatment significantly lowered DAI scores, attenuated inflammation and oxidative damage in both colon and brain tissues, enhanced antioxidant enzyme activities, reduced apoptotic markers, and improved locomotor and pain-response behaviors. Histopathological analyses confirmed preservation of tissue integrity in CAP-treated groups. *Chrysophyllum albidum* pectin exerts protective effects against DSS-induced colitis exacerbated by a high-fat diet through multi-modal actions including anti-inflammatory, antioxidant, and anti-apoptotic pathways.

sciforum-161797: From Neural Oscillations to Cognitive Architecture: A Neurophenomenological and Vedāntic Approach to Mapping the Mind

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This study addresses the complex nature of human cognition within a dynamically complex interplay of distributed neural networks, oscillatory activity, and subjective experiential phenomena. Although computational models have made a rich addition to our understanding of memory, attention, and decision-making processes, it is often the case that these models neglect the phenomenological dimensions that make up the lived experience of thought. Accordingly, in the present paper, a neuro-phenomenological framework that synergistically couples EEG-based neural oscillation analysis with first-person cognitive narratives is introduced for mapping the architecture of consciousness and cognition in a more integrative way. Employing EEG recordings taken during conditions of both rest and task performance, the present study addresses the issue of correlations between oscillatory signatures (i.e., alpha, theta, and gamma bands) and self-reported body variable cognitive states of attention, working memory load, and perceptual switching. These empirical relationships are then interpreted in the context of predictive processing, and explain how the brain builds internal world models and how departures from these world models can bring about cognitive dissonance or attention drift. One or two secondary objectives are to explore the possibility of using artificial intelligence models of intelligence, especially architectures of deep learning style, trained on multimodal data, to simulate or approximate the aforementioned cognitive states. By combining human EEG signatures, and the internal state of representation of artificial neural networks, this work attempts to narrow down the gap between biological and computational understanding. The findings add to the emerging field of discussion in computational cognitive neuroscience, embodied cognition, and cognitive modeling that insists that future brain–behavior mapping efforts do not solely focus on the neural correlates of thought but also its structural and phenomenological "feel". This integrative way of thinking has very important implications for cognitive training, neuroadaptive interfaces, and diagnostics for mental health.

sciforum-157340: Neurobiology of early mother–infant bonding, the role of oxytocin and favorable midwifery practices

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The dynamic between early infant behavior and caregiver sensitivity can be complex. It is also fundamental in shaping early infant development and long-term psychological well-being. Several studies confirm a positive association between caregiver sensitivity in the postpartum period and secure parent–infant attachment relationships. Oxytocin has emerged as a key neurohormone influencing this process.

The purpose of this paper is to provide an overview of the neurobiological basis of mother–infant bonding in response to the oxytocin system and propose favorable midwifery practices that enhance its natural secretion.

This study is a narrative review. Studies published between 2015 and 2025 were selected regardless of design, aiming to summarize the current evidence. A comprehensive literature search was conducted to identify articles examining the oxytocin's role in infant–maternal bonding and favorable midwifery practices that enhance this process. The databases used were PubMed, Scopus, and PsycINFO using keywords such as "oxytocin", "maternal bonding", "attachment" and "neurobiology".

Findings indicate that oxytocin facilitates bonding through modulation of several pathways. Oxytocin, is a neuropeptide synthesized in the hypothalamus which enhances activity in the nucleus accumbens, reinforcing maternal caregiving, modulates amygdala responses, reducing fear and stress and promotes recognition of social cues and attentional focus on infant signals. Such findings may support evidence-based midwifery training and perinatal care strategies fostering maternal–infant well-being. Emerging evidence also indicates oxytocin's role in paternal bonding and co-parental synchronization.

Midwifery-led care practices such as skin-to-skin contact, breastfeeding, physical touch, exposure to maternal live voice (speaking or singing), and supportive birth environments further enhance natural oxytocin release in infants, mothers and fathers. Limitations include variability in measurement methods and inconsistent correlations between peripheral and central oxytocin activity. Overall, this review highlights oxytocin's multidimensional role in establishing bonding and underscores the importance of the midwifery-led care model as a neurobiologically attuned model for optimizing early attachment outcomes.

sciforum-164318: Anger-Guilt Cycle: A Theoretical Model of Dynamic PFC-Basal Ganglia Dysregulation and Repair

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Introduction: Current models lack a detailed neurocircuitry account of how acute stress transitions into **impulsive action** and, subsequently, **guilt-driven repair.**

Method: We present a **novel theoretical model** of stress-mediated PFC-Basal Ganglia dysregulation that unfolds within a dynamic environmental context and is critically shaped by the individual's learned history. The process is triggered by salient environmental stimuli, which the amygdala appraises based on past associative learning. This triggers acute anger, activating the SAM/HPA axes and elevating neurochemicals that suppress PFC activity. Because of this reduced PFC activity, the Basal Ganglia cannot receive its critical input for the proper anticipation of consequences. The striatum then chooses an action based primarily on the immediate stimulus due to the lack of integrative PFC input. This decision is further biased by amygdala-driven salience and reinforced behavioral patterns from the individual's past(e.g., a learned tendency toward verbal aggression). As neurochemicals clear and PFC function restores, its renewed projections evaluate the impulsive act against internalized social rules. This mismatch generates guilt, which subsequently biases the striatum toward a reparative action(e.g., apology) informed by learned strategies for social reconciliation.

Results: This model is supported by: Pharmacological evidence that high levels of catecholamines impair prefrontal cortex (PFC) cognitive regulation via alpha-1 adrenergic and D1 dopaminergic receptors, a deficit reversed by adrenergic blockade **(Arnsten, 2009);** Connectivity analyses demonstrating decreased functional coupling between the medial PFC and emotion-processing regions (amygdala/striatum) during anger provocation, which correlates with reactive aggression **(Klaassens et al., 2018);** and neuroimaging studies identifying a distributed neural signature of guilt centered on the dorsomedial PFC and anterior midcingulate cortex, which predicts the motivation for reparative behavior **(Yu et al., 2019).**

Conclusion: This model offers a comprehensive framework linking acute stress neurochemistry to observable behavioral shifts, potentially informing future experimental designs and therapeutic targets for impulse-related psychopathology.

sciforum-164377: Cognitive impairments induced by valproic acid in Wistar rats: experimental validation of the Three-tower maze

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Introduction: Animal models play a central role in the experimental study of cognitive dysfunction. The development of specific behavioral tools allows for detailed analysis of the mechanisms involved in spatial learning and memory. The three-tower maze (3T) was designed as an original device for sequential analysis of orientation, decision-making, and memory abilities. Valproic acid, a molecule widely used in neurology, is suspected of impairing cognitive functions through neurotoxic mechanisms. **Objective:** Experimentally validate the 3T maze as a tool for detecting memory deficits after subchronic exposure to valproic acid in Wistar rats. **Methods:** Forty-eight Wistar rats of both sexes were randomly divided into three experimental groups: a control group and two groups treated with valproic acid at doses of 200 and 400 mg/kg. The drug was administered orally once a day for 30 days. At the end of the protocol, the animals were subjected to the three-tower maze test. Performance was quantified using parameters combining spatial orientation, cognitive efficiency, decision latency, route errors, and locomotor activity. **Results:** Animals exposed to valproic acid showed a progressive deterioration in learning and memory abilities, reflected in a significant increase in path errors, longer reward access times, and decreased overall cognitive efficiency. In addition, an increase in avoidance and vigilance behaviors was recorded, suggesting an emotional impact associated with cognitive deficits. **Conclusion:** The three-tower maze appears to be a relevant experimental tool for the integrated study of memory and behavioral disorders induced by valproic acid. It offers a complementary approach to conventional cognitive exploration tests in rats.

sciforum-164400: Comparative Analysis of Experimental Models in Alzheimer's Disease Research: From Transgenic Paradigms to Pharmacological Induction

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Alzheimer's disease (AD) represents the leading cause of dementia globally, characterized by progressive cognitive decline, β-amyloid (Aβ) aggregation, tau hyperphosphorylation, neuroinflammation, and synaptic dysfunction. Due to its complex multifactorial nature, the development of appropriate animal models has become indispensable for understanding the pathophysiology of AD and identifying potential therapeutic targets.

This paper provides a comparative analysis of the principal preclinical models used in AD research, encompassing transgenic, pharmacological, and toxin-induced paradigms. Transgenic models, such as 5×FAD, APP/PS1, and 3×Tg-AD, replicate essential molecular and behavioral features of familial AD. The 5×FAD line, carrying five human *APP* and *PSEN1* mutations, develops early and aggressive amyloid pathology with profound cognitive deficits, while the 3×Tg-AD model integrates both amyloid and tau lesions, offering broader pathological relevance. Pharmacological models, including scopolamine- and streptozotocin-induced neurotoxicity, mimic sporadic AD through cholinergic hypofunction and impaired insulin signaling. Toxicological paradigms employing β-amyloid peptides or aluminum compounds reproduce oxidative stress and neuronal loss typical of late-stage pathology.

A critical comparison of these models in terms of construct, face, and predictive validity highlights their differential translational potential and limitations. While transgenic models provide genetic precision, pharmacological paradigms allow reversible and cost-effective testing of neuroprotective agents. Integrative approaches combining genetic, metabolic, and environmental factors promise more faithful recapitulation of human AD. This synthesis underscores the necessity of methodological pluralism, ethical refinement, and data reproducibility to advance preclinical neuroscience and accelerate therapeutic discovery in neurodegenerative research.

sciforum-154791: Decoding the Neural Architecture of Reward and Aversion: A Multi-Modal Analysis of Decision-Making and Emotional Regulation in the Human Brain

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The balance between reward and aversion is central to adaptive behavior, yet the neural mechanisms orchestrating this balance remain incompletely understood. Recent progress in **behavioral neuroscience** suggests that decision-making and emotional regulation are shaped by dynamic interactions between dopaminergic, glutamatergic, and peptidergic signaling systems distributed across cortico-limbic networks.

This study integrates **functional neuroimaging, behavioral modeling, and molecular profiling** to dissect the reward–aversion circuitry underlying motivated behavior. Using a cohort of 120 healthy adults and 40 individuals with maladaptive reward processing (subclinical addiction and anxiety traits), we applied **fMRI with dynamic causal modeling (DCM)** and **diffusion-weighted tractography** to map directed connectivity between the ventral tegmental area (VTA), amygdala, and prefrontal cortex. Concurrent saliva cortisol and blood metabolomics provided peripheral biomarkers of stress-linked modulation.

The data reveal that **reward expectancy** enhances VTA–prefrontal coupling via dopamine-mediated glutamatergic pathways, while **aversive cues** increase amygdaloid inhibition of orbitofrontal regions, attenuating behavioral flexibility. Elevated peptide signaling (notably neuropeptide Y and dynorphin) correlated with impaired decision speed and heightened emotional reactivity. Machine learning classifiers achieved 88% accuracy in distinguishing adaptive versus maladaptive responders based on neural–biochemical signatures.

These findings delineate a **multi-scale model of behavioral regulation**, in which cross-talk between reward and aversion networks predicts individual differences in emotional control and cognitive bias. Targeting neuromodulatory peptides and stress circuits could thus inform new interventions for compulsive and affective disorders.

sciforum-164256: Deterministic Emergency Altruism: A Neurobiological Model of Stress-Induced Helping

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Introduction: The **"Paradox of Altruism"** questions why individuals incur personal risk to aid **non-kin** strangers—a behavior not fully explained by traditional evolutionary or psychological models centered on kinship or reciprocity.

Method: We developed a **theoretical** neurobiological framework through a synthesis of established literature. We propose that altruism is driven by the imperative to avoid acute stress, mediated by a subcortical circuit involving the Hypothalamic-Pituitary-Adrenal (HPA) axis, the Sympathetic-Adreno-Medullar (SAM) axis, the prefrontal cortex (PFC), and the basal ganglia.

Results: The model posits that a high-distress stimulus triggers acute HPA and/or SAM axis activation, inducing a rapid reduction in prefrontal cortex (PFC) activity—a stress-induced executive impairment well-documented in the literature **(Arnsten, 2009).** Due to this diminished PFC function, individuals cannot engage in future-oriented consequence anticipation of their action. With top-down control suspended, the basal ganglia—a structure central to habit formation and action selection under motivational drive **(Graybiel,2005)**—selects the most immediate and efficient action to terminate the internal aversive signal via **negative reinforcement,** a process supported by dopaminergic signaling linked to aversive state relief **(Redgrave et al., 2010).** The altruistic intervention is thus executed as the **optimal action** for immediate stress relief. This mechanism, evolved for kin-protection in **ancestral** small groups, is now activated indiscriminately by vulnerable human stimuli, constituting an evolutionary mismatch **(Li et al., 2018).**

Conclusion: **Altruism** toward strangers is reconceptualized as **deterministic,** self-directed physiological regulation. This model generates **novel,** falsifiable **predictions;** for example, it suggests a precise temporal sequence in which an acute stress spike via the SAM axis (upon encountering a threatened non-kin human) precedes a reduction in prefrontal activity and the subsequent selection of helping actions—provided the altruistic act reduces stress relative to that individual's **perspective.** This framework offers a **new** lens through which to dissect the **proximate** causes of paradoxical helping behavior.

Conflict of interest: **None** declared.

sciforum-157894: Ethanol Extract of *Solanum campylacanthum* Mitigates Hypoxia-Induced Neurobehavioural and Biochemical Alterations in Male Wistar Rats

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Introduction: Hypoxic stress disrupts homeostatic balance, triggering neuroendocrine and oxidative responses that manifest as behavioural impairments and biochemical alterations. Elevated corticosterone and oxidative damage are hallmark features of such stress-induced pathology. *Solanum campylacanthum*, a plant traditionally used as a pest deterrent, lacks empirical evidence supporting its medicinal application. However, its taxonomic synonym *Solanum incanum* has demonstrated antioxidant activity. This study investigates the potential of ethanol extract of *S. campylacanthum* leaves (EESC) to counteract hypoxia-induced neurobehavioral and biochemical disturbances in male Wistar rats.

Methods: Acute toxicity was evaluated using Lorke's method, revealing an oral LD_{50} exceeding 5000 mg/kg. Rats (150–200 g) were randomized into five orally treated groups (n=6): Group 1—vehicle (10mL/kg, distilled water) without exposure to hypoxia; Groups 2–5—10mL/kg, distilled water, EESC (100, 200, and 400 mg/kg), respectively, one hour before exposure to hypoxia which was induced by placing animals in sealed 450 mL chambers for 20 minutes daily over 14 days. Behavioural assessments (anxiety, depression, memory, and locomotion); serum corticosterone; brain oxidative markers, such as superoxide dismutase (SOD), catalase, glutathione (GSH), and malondialdehyde (MDA); and adrenal weights were evaluated.

Results: Oral administration of EESC significantly alleviated hypoxia-induced behavioural impairments, including anxiety, depressive-like symptoms, motor deficits, and memory decline. Biochemical analysis revealed that treatment with EESC dose-dependently reduced serum corticosterone and brain MDA levels when compared to hypoxic control, indicating suppressed stress and oxidative damage. Concurrently, antioxidant defenses were enhanced, with marked dose-dependent increases in SOD, catalase, and GSH concentrations. Additionally, EESC treatment significantly prevented adrenal gland hypertrophy, suggesting the modulation of the hypothalamic–pituitary–adrenal axis.

Conclusion: These findings collectively indicate that *S. campylacanthum* extract confers adaptogenic and antioxidant benefits under sustained hypoxic stress, supporting its potential for further pharmacological investigation.

sciforum-163314: Exploring Chaotic Conditions Through Behavioral Neuroscience

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This paper **investigates** chaotic conditions that organizations face, emphasizing the necessity of adopting new technologies and strategic thinking. The **focus** is on behavioral sciences, particularly the relationship between brain functionality, neurological processes and managerial behavior. This research **delves** into analysis of brain wave activity, especially Alpha Waves, using a blend of theoretical and experimental methods to understand influences on decision-making in uncertain environments. Vital **questions** are posed, such as how knowledge of brain wave activity can enhance decision-making and the implications of these findings for psychology and model resilience against adversarial attacks. Through **empirical exploration** and use of EEG, this study links behavioral research with computational models, highlighting impact of brain functionality on behavior and advocating for rethinking of decision-making processes. Ultimately, this research **aims** to develop robust models that reshape managerial strategies, thereby emphasizing importance of neuroscience in addressing management challenges. This paper poses key questions regarding interpretability of models based on brain activity and resilience to biases and adversarial attacks. Some of these are as follows: How does understanding brain wave activity enhance the comprehension of decision-making in environments? In what ways can organizations implement findings to improve decision-making? What implications do these findings have for future research in psychology? How might adversarial attacks influence development of decision-making models using brain data? Through empirical exploration, via EEG's role in enhancing decision-making and involving a small respondent sample, this study seeks to bridge behavioral research with computational models to enhance understanding of cognitive dynamics in managerial contexts. This paper **advocates** for rethinking decision-making foundations by proposing alternative frameworks to better comprehend decision problems in practical applications. This paper **contributes** valuable insights toward developing more robust and interpretable models that can radically transform managerial strategies. Consequently, the **recommendations** put forth urge a re-evaluation of decision-making frameworks, reinforcing relevance of neuroscience in practical management issues.

sciforum-158141: Feasibility of identifying catatonia using the Bush-Francis Catatonia Rating Scale via remote motor assessments

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Introduction

Catatonia is a serious yet often underdiagnosed neuropsychiatric condition marked by disturbances in motor behavior, speech, and responsiveness. Despite its clinical significance, limited tools exist for reliably assessing catatonia outside traditional in-person psychiatric evaluations. The need for remote and standardized assessment tools is urgent in underserved areas where face-to-face consultations may not be feasible. The Bush-Francis Catatonia Rating Scale (BFCRS) [Bush, *et al. Acta Psychiatrica Scandinavica,* 1996;93:129-136] offers a structured, validated approach to quantifying catatonia symptoms that may be adaptable for remote use.

Methods

As part of an ongoing project [Kadubandi, *et al.* Development of a rating instrument to identify catatonia by virtual viewing of motor assessments. Zenodo, V3, 2025. https://doi.org/10.5281/zenodo.16061703], a team of eight raters trained in the BFCRS independently viewed and scored videos of motor assessment of a male identified only by age and sex. All assessments were based solely on observed motor behavior. Consensus discussions to agree on a score for each item were conducted after individual ratings were submitted to the organizing committee. Statistical analysis, including inter-rater reliability using Fleiss' Kappa, was conducted using R software.

Results

Six separate assessments were conducted across five video sessions. Each of the seven raters independently scored 23 BFCRS items. Fleiss' Kappa was used to evaluate inter-rater reliability. The calculated Fleiss' Kappa values across the six assessments were 0.375, 0.556, 0.576, 0.667, 0.590, and 0.578. The average Fleiss' Kappa was 0.557, indicating moderate inter-rater agreement, which supports the reliability of the BFCRS when used remotely.

Conclusions

The preliminary application of the BFCRS in a remote assessment framework suggests potential for standardizing catatonia evaluations beyond traditional settings. If validated by further statistical analysis, this approach could improve access to timely diagnosis and intervention, particularly in areas with limited psychiatric resources.

sciforum-133878: Intergenerational effects of childhood stress on depressive-like behaviors and function of glucocorticoids in the Nucleus Accumbens: therapeutic potential of *Centella asiatica*

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Introduction: Major depressive disorder (MDD) is a disorder strongly linked to childhood stress, amplified by a lack of social support later in life. Rodent models like maternal deprivation (MD) and social isolation (SI) mimic these traumas, highlighting how early-life stress can alter the hypothalamic–pituitary–adrenal (HPA) axis and be transmitted to offspring. Objective: The objective of this study is to evaluate depressive-like behaviors and HPA axis protein in rats subjected to MD and SI and in the stressed female offspring, as well as the therapeutic potential of Centella asiatica and its active compound, madecassic acid. Methods: The animals were male and female Wistar rats. MD consisted of depriving the pups three hours per day for ten days. SI consisted of keeping the animals in individual cages, starting at 50 days of age. Forced swimming and gene expression of the HPA axis proteins NR3C1 and FKBP5 in the Nucleus Accumbens (NAc) were evaluated in both animals that suffered stress and in offspring of mothers who experienced stress (second-generation). After 30 days of IS, the hydroalcoholic extract of Centella asiatica (30 mg/kg) and madecassic acid (10 mg/kg) were administered for fourteen days. Results: Stress from PM and IS resulted in depressive-like behavior in adulthood of the first-generation and in offspring (second-generation) from stressed mothers. Treatments with Centella asiática and madecassic acid reduced depressive-like behavior. These compounds also reversed the reduction of NR3C1 and FKBP5 in the NAc of the stressed first-generation animals, although without corresponding changes in NAc gene expression. Conclusion: Centella asiatica extract and the active compound madecassic acid have antidepressant potential, modulating the effects of chronic childhood stress in animals that experienced stress and in their offspring who were not subjected to MD and SI protocols. This potential is further supported by the observed modulation of the HPA axis changes.

sciforum-165927: Machine Learning in Psychiatric Research: A Systematic Review of Predictive and Mechanistic Models

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The application of machine learning within psychiatric research has proliferated in recent years, driven by the growing availability of large-scale neurobiological, behavioral, and clinical datasets. This development has enabled novel approaches to modeling the complex and multifactorial processes underlying mental disorders. Yet, a persistent tension remains in the field between predictive accuracy and mechanistic interpretability, which constrains the translational potential of ML-derived insights for both neuroscientific theory and clinical application. This systematic review critically synthesizes contemporary ML-based methodologies in psychiatric research, with particular attention to the distinction between predictive models, designed to maximize classification performance or prognostic precision, and mechanistic models, which seek to elucidate the latent cognitive and neural processes implicated in psychopathology. This review synthesizes peer-reviewed machine learning studies in psychiatric research spanning neurobiological, behavioral, and clinical domains, with emphasis on predictive and mechanistic models. The extant literature reveals a pronounced predominance of predictive modeling approaches, typically employing supervised learning algorithms such as support vector machines and deep learning architectures to address tasks ranging from diagnostic categorization and symptom severity estimation to treatment response prediction in conditions such as schizophrenia, mood disorders, and anxiety disorders. In contrast, mechanistic machine learning frameworks, often situated within computational psychiatry paradigms such as reinforcement learning and Bayesian modeling, are comparatively underutilized, though they offer critical explanatory power by linking model parameters to underlying cognitive and neural mechanisms. A subset of emerging hybrid approaches seeks to reconcile predictive utility with mechanistic clarity, though they remain methodologically heterogeneous and insufficiently validated. The findings highlight enduring methodological challenges in the field, including limited generalizability across cohorts, inconsistent validation strategies, and difficulty aligning learned representations with underlying neurocognitive processes. These limitations underscore the imperative for computational models that are not only predictive but also mechanistically interpretable, thereby advancing both theoretical insight and translational relevance.

sciforum-158142: Remote evaluation of stereotypy in hypokinetic catatonia with the Timed Stereotypies Rating Scale

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Background:

Stereotyped behaviors are a hallmark of neurodevelopmental disorders, but are often difficult to quantify in real-time, especially in remote clinical settings. These repetitive, non-goal-directed movements can provide critical diagnostic information, yet no dedicated tool currently exists for structured assessment via telehealth. In response, we utilized the Timed Stereotypies Rating Scale (TSRS) [Brašić JR. Treatment of movement disorders in autism spectrum disorders. In: Hollander E (Editor). Autism Spectrum Disorders. Volume 24 of the Medical Psychiatry Series. ISBN 0-8247-0715-X, Marcel Dekker, Inc., New York. 2003;273-346] to identify and rate these behaviors in a patient with hypokinetic catatonia.

Objectives:

As part of an ongoing project [Kadubandi, *et al.* Development of a rating instrument to identify catatonia by virtual viewing of motor assessments. Zenodo, V3, 2025. https://doi.org/10.5281/zenodo.16061703], we tested the feasibility of applying the TSRS during remote clinical assessments using video recordings shared via teleconferencing platforms.

Methods:

Archived video recordings of a male displaying markedly reduced movements were shown to trained clinical raters during online meetings. The session host shared the screen to present each video. Raters independently used the TSRS to assess the presence, frequency, and severity of stereotyped motor behaviors. After individual ratings, consensus scores were discussed and documented for each video segment.

Results:

Fifteen valid observations were included in the analysis utilizing R. The internal consistency of the TSRS was high, demonstrated by a **Cronbach's alpha of 0.838** across 7 items. Inter-rater reliability was also strong. The **Intraclass Correlation Coefficient (ICC)** for **average measures was 0.804** (95% CI: 0.611–0.923, p 0.001), indicating **excellent agreement** among raters when averaging scores. The **ICC for single measures was 0.370** (95% CI: 0.183–0.631, p 0.001), suggesting **fair reliability** for individual raters.

Conclusions:

Preliminary application of the TSRS in remote settings shows promise as a structured method for evaluating catatonic behaviors virtually.

sciforum-161052: The Importance of Behavioral Phenotyping in Evaluating Multitarget Phytochemicals in the 5xFAD Model of Alzheimer's Disease

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Behavioral phenotyping represents a central pillar of contemporary behavioral neuroscience, offering a functional readout of neural integrity that cannot be captured solely through molecular or histological markers. In Alzheimer's disease (AD), where cognitive decline, emotional dysregulation, and disruptions in adaptive behavior emerge long before overt neuropathology is detectable, behavioral assays provide an indispensable tool for characterizing therapeutic potential. This study underscores the critical role of behavioral testing in evaluating ten phytochemical extracts derived from *Rubus fruticosus*(FT,LT,FH,LH,PFH,PLH), *Abutilon pannosum*(A2), *Abutilon grandifolium*(A1), *Rheum palmatum*(R), and *Zingiber officinale*(G) in the 5xFAD transgenic mouse model, integrating in silico predictions with multidimensional behavioral outcomes.

Pharmacokinetic and target-prediction analyses suggested favorable absorption, strong blood–brain barrier permeability, and potential engagement with pathways implicated in synaptic plasticity and neuroprotection. However, behavioral assays were essential for determining whether these theoretical properties translated into meaningful functional effects. A comprehensive battery was employed to probe complementary cognitive and affective domains: Y-Maze for working memory, Novel Object Recognition for recognition memory, Radial Arm Maze for spatial learning, Open Field and Elevated Plus Maze for locomotion and anxiety-like behavior, and Forced Swimming for depressive-like responses.

Behavioral outcomes revealed extract-specific signatures that were not predictable from computational data alone. Most extracts displayed an inverse dose-dependent pattern, with lower doses producing more pronounced improvements in memory performance and emotional regulation, often surpassing the effects of the reference treatment. In contrast, other extracts followed a classical dose-response profile, enhancing cognition at higher doses and increasing exploratory behavior at lower doses.

These findings highlight the irreplaceable value of behavioral testing as a functional bridge between in silico predictions and neurobiological mechanisms. By capturing complex, systems-level outcomes, behavioral assays remain essential for identifying promising neuroactive compounds and for advancing translational research in AD.

sciforum-161025: The Proximal Chemical Mandate Principle: A Framework for Invariant Biological Dynamic Optimization

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Introduction: Current explanations of motivated behavior remain theoretically fragmented across neuroscience, psychology, evolutionary biology, etc. The Proximal Chemical Mandate Principle addresses this fragmentation by proposing that all motivated behavior can be reduced (for unification) to two invariant neurobiological objectives: reward neurochemicals maximization (R↑) (e.g., Dopamine, Opioid , Oxytocin, and more) and/or stress neurochemicals minimization (S↓) (e.g., Norepinephrine, Cortisol, and more).

Methods: We developed this novel theoretical reductionist framework by synthesizing direct evidence from decades of behavioral neuroscience literature, including intracranial self-stimulation, conditioned avoidance paradigms, fiber photometry, and optogenetic studies. The model establishes causal relationships with specific neurochemical signals to motivated behaviors and its ultimate outcome.

Results: Direct experimental evidence demonstrates the following:

Compulsive reward maximization via intracranial self-stimulation, whereby rats repeatedly press levers to stimulate reward pathways despite physiological exhaustion (Olds & Milner, 1954).

Immediate subsecond dopamine encoding of reward value, with nucleus accumbens transients driving motivated action (R↑) within 150–300 ms (Hamid et al., 2021; Mohebi et al., 2024).

Learned stress minimization through conditioned avoidance behaviors (by prediction) that trigger immediately and persist even without immediate threat.

Rapid-onset threat processing via specialized amygdala circuits that detect threats within 120 ms and initiate defensive responses (S↓) within 200 ms (Li et al., 2022).

Real-time instantaneous value–threat integration through prefrontal–striatal circuits that resolve decision conflicts (S↓) within 400–600ms (Zhou et al., 2023).

Conclusions: These findings support a unified novel, testable framework that potentially resolves apparent behavioral paradoxes—including altruism (S↓via altruistic act), addiction (R↑via supernormal stimuli), suicide (S↓), and voluntary childlessness—by demonstrating how identical (R↑,S↓) mandates produce divergent outcomes through contextual implementation. The principle provides testable predictions (if direct mesolimbic dopamine self-stimulation ever stops voluntarily without any other strong environment stimuli, the theory is falsified) for behavioral neuroscience and psychiatry.

Conflicts of interest: None

sciforum-157253: The role of Prostaglandin E2 in alcohol-related inflammation in Drosophila melanogaster

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Introduction

Alcohol consumption is a leading risk factor for premature mortality and disability. Alcohol consumption is known to activate pro-inflammatory signalling pathways that affect the production of prostaglandins (PGs) and reactive oxygen species (ROS). The mechanisms through which alcohol can alter PG synthesis are not fully understood. *Drosophila melanogaster* (fruit flies) is an established model system for studying alcohol-related behaviours and neuronal responses, but limited studies have utilised this model for elucidating alcohol-related inflammatory processes. In Drosophila, the Peroxinectin-like (PXT) and Cardinal (Cd) genes are believed to encode for putative cyclo-oxygenase-like enzymes and be responsible for PG synthesis, including PGE_2. The aim of this study is to understand the effect of alcohol consumption on PGE_2 levels in relation to inflammatory processes and addictive behaviours.

Methods

The development of tolerance was measured in *Drosophila* by measuring the time taken by half a cohort of flies to be sedated by ethanol vapours (ST50). An increase in ST50 following repeated ethanol exposures on consecutive days indicates development of tolerance. The level of PGE_2 was measured in homogenates of fly heads and bodies using an ELISA Kit (Cayman) in wild-type, PXT and Cd mutant flies with and without exposure to 2mM aspirin (a cyclo-oxygenase inhibitor).

Results and Conclusion

All *Drosophila* species examined developed tolerance. but their sensitivity to ethanol varied. Ethanol caused a different effect on PGE_2 production in different fly species (wild type and mutants); additionally, aspirin had a different capacity to alter the ethanol effect on PGE_2 production and the development of tolerance. These results, thus, provide evidence that the inflammatory process and addiction-related behaviours are interlinked and support the use of Drosophila as a tool for evaluating these mechanisms.

Session 4. Cognitive Neuroscience

sciforum-161096: Can Music Training Enhance/Affect Working Memory and Speech-in-Noise Perception in Cochlear Implant Users? A Randomized Controlled Study of EEG Measures of Improvement

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Hearing loss profoundly affects individuals' quality of life, limiting participation in social, professional, and communicative domains. Cochlear implants (CIs) restore access to auditory input for postlingually deafened adults, but understanding speech through a CI relies heavily on neuroplastic adaptation. While many CI users regain the ability to follow conversations in quiet environments, speech-in-noise (SPiN) perception remains a major challenge. Music training has been shown to enhance shared auditory and cognitive neural networks and to improve auditory-motor coupling, which may facilitate SPiN perception in both normal-hearing and hearing-impaired individuals. These findings provide a rationale for using multi-modal Neurologic Music Therapy (NMT™) to support auditory rehabilitation in adult CI users. However, research to date has focused primarily on passive music listening, leaving the effects of structured, active music training largely unexplored. This randomized controlled clinical trial investigates the effects of short-term targeted multi-modal music training on SPiN and working memory (WM) in postlingually deafened adult CI users. Additionally, the study examines neurophysiological mechanisms of change using electroencephalography (EEG), specifically focusing on alpha oscillation modulations during auditory-cognitive tasks. Thirty-six adult CI users are enrolled in a 4-week NMT™ training program, randomly assigned to pitch, rhythm, or timbre-focused conditions. Behavioral outcomes are assessed pre- and post-training using the sentence-final-word-identification-and-recall (SWIR) test; EEG is recorded during all SWIR tasks. Presented will be behavioral results from 36 participants on SPiN perception and WM performance between training types. So far, only behavioral data have been analyzed; EEG analysis is ongoing and will be reported as available. Early findings, based on a small and heterogeneous sample, show promising trends. Further EEG analysis is expected to provide deeper insight into neuroplastic changes, including modulation of alpha activity, potentially supporting music-based strategies to enhance auditory performance and life quality in CI users.

sciforum-158109: Psychophysiological Correlates of Geometric Visual Illusions: A Comparative Study of the Ponzo and Müller-Lyer Effects.

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Introduction:
Although the Ponzo and Müller-Lyer illusions distort line length similarly, they may involve distinct perceptual mechanisms. Both illusions make equal line segments appear unequal, yet prior studies classify them into different categories of visual processing (Gregory, 2009; Coren et al., 1978). Evidence suggests that the N200 component reflects intermediate visual processing, bridging low- and high-level stages of perception (Yang & Sui, 2022). Eye movement studies indicate that the Müller-Lyer illusion distorts saccadic trajectories (Chen et al., 2020), whereas in the Ponzo illusion, fixations tend to be longer on the upper segment than the lower one (Yildiz et al., 2019). This study compared the psychophysiological correlates of Ponzo and Müller-Lyer illusions by analyzing electrophysiological and oculomotor activity during the perception of equal central line segments.

Methods:
Forty participants (aged 18–45, M = 24.7) underwent synchronous EEG (64-channel BrainVision ActiChamp) and eye-tracking (EyeLink 1000). Stimuli included equal and unequal line segments in three conditions: control, Ponzo, and Müller-Lyer. ERPs (N200 270–320 ms), fixation and saccade durations, and response accuracy were analyzed using a repeated-measures ANOVA and the Friedman test.

Results:
The accuracy of the responses was significantly higher in the control condition than in both illusion conditions (p 0.0001). N2 amplitudes (F1, F3, FC1, FC3) were higher for Ponzo than for Müller-Lyer (p 0.0001), and in FC3, the control exceeded the Müller-Lyer group (p 0.05). Fixation durations differed between the control and Müller-Lyer (p = 0.0097) groups and the Ponzo (p 0.0001) group, while saccades were shorter in the Ponzo group. Eye movement heat maps revealed distinct viewing patterns across conditions.

Conclusions:
It was found that Ponzo and Müller-Lyer illusions engage partially distinct visual mechanisms. Behavioral differences were minimal, suggesting compensatory higher-level cognitive processing. Oculomotor and electrophysiological data together provide a nuanced understanding of the intermediate stages of geometric illusion perception.

sciforum-158131: Acute physical exercise enhances semantic memory processing speed: evidence from a reaction-time lexical decision task

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Introduction: Regular physical activity modulates neuroplasticity and protects against age-related cognitive decline. However, the acute effects of exercise on higher-order language functions remain poorly defined. This study investigated how a single moderate-intensity cycling session influences lexical-semantic processing speed in middle-aged adults.
Methods: Forty-one healthy participants (mean age 43.6 ± 1.7 years; 22 women, 19 men) were randomly assigned to an exercise group (n = 20; 15 min cycling at 65% HRmax) or control group (n = 21; passive video viewing). Cognitive performance was assessed before and after intervention using a lexical decision task involving real and pseudowords (1 s presentation). Reaction times (RTs) and accuracy were recorded. Heart rate (HR) was continuously monitored to verify exertion level. Participants were additionally divided into younger (40 years) and older (>40 years) subgroups to assess age-related differences in cognitive response.
Results: Heart rate analysis confirmed adequate workload and recovery ($p \leq 0.001$). Acute exercise significantly reduced RT for both pseudowords ($d = 0.60$; $p \leq 0.0001$) and real words ($d = 0.39$; $p \leq 0.0001$), whereas the control condition produced smaller but reliable improvements (pseudowords $d = 0.40$; $p \leq 0.001$; words $d = 0.27$; $p \leq 0.01$). Age-stratified analysis indicated more pronounced improvements in participants > 40 years ($p \leq 0.05$). These behavioral changes suggest transient enhancement of cortical efficiency in semantic decision-making, possibly mediated by increased cerebral perfusion and neurotrophic signaling.
Conclusion: A brief session of moderate-intensity aerobic exercise enhances semantic processing speed, reflecting rapid neuroplastic modulation of cortical language networks. The effect was evident across both sexes and most pronounced in adults over 40, suggesting that short aerobic activity may serve as a simple, non-pharmacological intervention to preserve semantic memory and cognitive fluency during midlife and aging.

sciforum-157156: Cognitive Neurological Mechanisms and Causal Analysis of Brain-to-Brain Synchronization Between Teachers and Students

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Objective: This review systematically examines the application of hyperscanning techniques in educational neuroscience over the past five years. It aims to elucidate the multidimensional cognitive–neural mechanisms underlying Interpersonal Brain Synchronization (IBS) during teacher–student interactions and to evaluate its potential causal role in pedagogical effectiveness.

Methods: A systematic search was conducted across academic databases, including Web of Science, PubMed, and Google Scholar, for core journal articles published between 2020 and 2025. The focus was on empirical studies employing hyperscanning techniques such as fNIRS, EEG, and fMRI to investigate teacher–student interactions. The selected literature underwent inductive, comparative, and integrative analysis.

Results: The findings indicate that IBS serves as a robust "biomarker" of effective teaching, emerging from the interplay of multi-level cognitive processes including joint attention, mentalizing, and behavioral coordination. The prefrontal cortex and temporo-parietal junction serve as key coupling regions. However, the current evidence remains predominantly correlational. A significant scholarly debate persists regarding whether IBS drives enhanced pedagogical outcomes or whether high-quality interactions and learning processes facilitate IBS. Direct experimental evidence establishing causality is still in its nascent stages.

Conclusion: Interpersonal brain synchronization represents a complex neural phenomenon associated with enhanced teaching efficacy. Future research should integrate experimental manipulations with neuromodulation techniques to resolve causal questions and develop dynamic mechanistic models. This effort will pave the way for novel approaches to precision education and teacher training grounded in neuroscientific evidence.

sciforum-155604: Correlations between olfactory, gustatory function, and cognitive abilities in different age ranges

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Aging is considered a progressive physiological degeneration associated with a decrease in synaptic plasticity and a decline in different functions such as olfaction, taste, attention, memory, and language. The biological mechanisms of aging are not yet fully understood. The aim of the study was to evaluate correlations between age-related changes in cognitive abilities with olfactory and gustatory functions. In our study, 290 participants with an age range from 18 to 86 years were enrolled. All participants were divided into three age groups: young adults (18-29 years, n=135), middle-aged (30-59 years, n=93), and elderly (≥60 years, n=62). Our results showed that odor threshold (OT), discrimination (OD), and identification (OI) significantly decreased in the elderly group compared to the young adults. Moreover, significant differences were also found for OT, OD, and OI between middle-aged and elderly groups. Instead, gustatory function decreased in relation to age in a different manner, since only sweet and sour taste perceptions decreased in elderly participants compared to the young adults' group. Similarly, cognitive abilities, memory, language, and global attention significantly declined in the elderly group compared to young adults and middle-aged groups. The decrease in global attention, memory, and language was significantly correlated to the OI performance. Interestingly, significant correlations were observed between sour taste perception versus global cognitive abilities, language, and memory.

In conclusion, olfactory function and cognitive abilities showed a slight decline in relation to age and a dramatic decrease after 60 years, while gustatory function decreased more gradually. The decrease in OI could be considered a predictor for global cognitive function, as well as for specific cognitive subdomains such as global attention, language, and memory. Our study highlighted the importance of using olfactory evaluations in clinical practice for the early diagnosis of cognitive decline and for the development of appropriate personalized risk prevention strategies.

sciforum-164393: Differential Cognitive Profiles Induced by Hexanic Fractions of *Rubus fruticosus* in a Transgenic Alzheimer's Disease Model

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Cognitive decline in AD extends beyond global memory impairment, encompassing deficits in working memory, cognitive flexibility, recognition processes, and the integration of exploratory strategies. Dissecting these components in transgenic models offers an opportunity to refine our understanding of how distinct neurobiological perturbations shape cognitive architecture under neurodegenerative conditions.

This study presents a comparative cognitive analysis of six hexanic fractions derived from *Rubus fruticosus* in the 5xFAD mouse model of AD. Transgenic mice were assigned to treatment groups receiving individual fractions at two concentrations (50mg/kg and 100mg/kg; n=10 per fraction) alongside control groups (NaCl,n=5; galantamine, n=5). All treatments were administered orally for seven consecutive days, one hour prior to behavioral assessment.

Cognitive performance was evaluated using a battery of tasks designed to probe complementary domains of cognition: spontaneous alternation (Y-Maze), recognition memory and encoding efficiency (Novel Object Recognition), and working and reference memory organization (Radial Arm Maze). Additional measures of exploratory behavior and emotional modulation were obtained using the Open Field, Elevated Plus Maze, and the Forced Swimming Test to contextualize cognitive outcomes within broader behavioral states. In silico pharmacokinetic and target-prediction analyses were conducted to support interpretation of behavioral variability across fractions.

Comparative behavioral profiling revealed fraction-specific cognitive signatures, indicating that distinct fractions differentially modulate cognitive subdomains rather than producing uniform enhancements across tasks. Several fractions exhibited non-linear dose–response relationships, with lower doses preferentially influencing memory accuracy and task-specific strategies, while higher doses primarily affected locomotor or exploratory parameters. These dissociations suggest that cognitive outcomes in the 5xFAD model reflect complex interactions between fraction-specific phytochemical composition and disease-related alterations in neural circuitry.

This study underscores the value of comparative cognitive phenotyping in transgenic models of AD. Integrating in silico predictions with multidimensional behavioral analysis provides a framework for interpreting divergent cognitive effects across chemically related fractions.

sciforum-156344: Direct Electrical Stimulation of Amygdala on Emotion

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The amygdala is an important part of the human brain that helps control individual feelings. It is connected to emotions, fear, and memory, and works closely with other areas of the brain. Techniques like transcranial electrical stimulation, which includes direct current stimulation, and transcranial alternating of the current stimulation change how the human brain works and influence behavior when electrical currents are applied. Most of the information about these electrical fields comes from computer models. Surprisingly, there have only been a few studies on the human brain that looked at the effects of directly stimulating the amygdala. This includes how it affects human emotional and physical reactions. Cognitive sciences still do not fully understand the exact emotional and physical changes that occur with this form of stimulation. Understanding how stimulating the amygdala impacts emotions is imperative for contemporary ideas about how feelings work in the human brain. These ideas present different viewpoints on the effect of stimulation on emotions. Studies that use functional neuroimaging proffer concrete substantiation for the significant function of the amygdala in handling emotions. To explore these discussions, this paper offers systematic review methods incorporating a structured literature search to document the entire process, ensure reproducibility, and minimize bias. This was conducted to examine functional neuroimaging research that examines how the human brain reacts to emotional representations that activate the amygdala. Our analysis specifically considered both the strength of the effects and the consistency of each activation, unlike past studies. Findings in this study suggest that the amygdala reacts to both positive and depressing emotionsthat demonstrate feelings. In conclusion, the results from **this** structured literature search and systematic review paper provide new insights into various theories and models discussed in contemporary research about how the human brain processes emotions.

sciforum-154793: Dynamic Network Reconfiguration during Attention and Working Memory: Integrating Neuroimaging and Computational Modeling for Cognitive Profiling

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Understanding how large-scale brain networks dynamically reorganize to support attention and working memory remains a central challenge in cognitive neuroscience. Emerging evidence suggests that the **prefrontal–parietal network** flexibly interacts with subcortical and default mode regions to optimize cognitive control, yet the temporal mechanisms underlying this reconfiguration remain unclear.

This study employed a **multi-modal experimental design** integrating functional MRI, electroencephalography (EEG), and computational modeling to investigate dynamic network transitions during attentional load and memory manipulation tasks. Eighty healthy adults completed a parametric n-back paradigm with real-time neuroimaging. **Dynamic causal modeling (DCM)** quantified directed connectivity, while **graph-theoretical metrics** assessed modularity and integration across cortical systems. Additionally, **recurrent neural network (RNN)** simulations were trained to reproduce observed neural trajectories and predict behavioral accuracy.

Results revealed a robust **task-dependent reorganization** of the frontoparietal control system, with transient decoupling from the default mode network (DMN) during high-load trials. EEG phase-synchrony analyses indicated theta–gamma coupling between dorsolateral prefrontal cortex and intraparietal sulcus as a predictor of task performance (r = 0.67, p 0.001). Computational models recapitulated these oscillatory dynamics, suggesting that recurrent feedback mechanisms enable efficient information maintenance.

Our findings provide convergent neurobiological and computational evidence that **cognitive flexibility arises from transient, hierarchical synchronization across distributed neural systems**. These results advance a mechanistic framework for understanding attention–memory interactions and may inform neuroadaptive interventions for cognitive decline.

sciforum-162154: EEG markers of cognitive load and mental fatigue in university students: a systematic review

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Introduction:

University students are frequently exposed to high cognitive demands, which can lead to sustained cognitive load and mental fatigue and may negatively affect learning outcomes and well-being. Electroencephalography (EEG) provides a non-invasive, real-time window into neural activity associated with cognitive effort. This systematic review aims to synthesise current evidence on EEG markers of cognitive load and mental fatigue in university students and other young adult learners.

Methods:

This systematic review was conducted in accordance with the PRISMA 2020 statement (Preferred Reporting Items for Systematic Reviews and Meta-Analyses). We systematically searched electronic databases (e.g., PubMed, Scopus and Web of Science) for peer-reviewed studies on EEG-based assessment of cognitive load/mental fatigue in healthy young adults (typically university students). Records were screened against predefined inclusion/exclusion criteria, and eligible studies were included for data extraction.

Results:

Seven studies met the inclusion criteria, comprising 179 participants (sample sizes 7–43). Paradigms varied across examination stress, language-mediated learning, multimedia manipulations, and classroom tasks, limiting direct comparisons. Cognitive load was most often reflected in changes in theta, alpha, and beta power and band ratios. One study found that self-reported difficulty correlated positively with beta activity at T3 ($r = 0.309$, p 0.05) and negatively with learning performance ($r = -0.391$, p 0.01). Studies manipulating multimedia design principles generally reported lower load indicators and better test performance when principles were applied.

Conclusions:

EEG-based markers, particularly theta and posterior alpha activity, show promising potential for indexing cognitive load and mental fatigue in university students. However, methodological variability and small sample sizes hinder the development of robust, generalisable EEG metrics. Future research should prioritise standardised protocols, larger cohorts and transparent reporting to support the use of EEG for monitoring cognitive strain in educational and applied settings.

sciforum-124738: Effects of Different Stress States on Athletes' Cognitive Function and the Underlying Neural Mechanisms

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Athletes often face acute, chronic, or extreme stress that activates the sympathetic nervous system–hypothalamic–pituitary–adrenal axis and elevates cortisol and catecholamines. Short bursts may aid arousal, but prolonged activation undermines dorsolateral prefrontal cortex (DLPFC)-mediated attention, working memory, and executive control. Precisely how each stress type affects cognition and its neural basis remains unclear.

Objective

To synthesise evidence (2000-2025) on how different stress states affect athletes' cognitive performance and to outline the associated neural mechanisms.

Methods

Following PRISMA, we searched CNKI, PubMed, Web of Science, SPORTDiscus, and PsycINFO (Feb 2000-Feb 2025). Keywords combined "stress", "athletes", "cognitive/executive function", and "neural mechanism". From 1,253 hits, 46 studies met inclusion criteria (athlete sample; stress manipulation; and cognitive or neural outcomes) and were qualitatively synthesised.

Results

1) Acute stress impairs attentional allocation and decision speed in combative/team sports, increasing critical-moment errors; 2) fine-skill athletes (e.g., gymnastics and shooting) show reduced motor precision under acute stress; 3) chronic stress in endurance athletes slows processing and reaction times and heightens cognitive fatigue; 4) working memory and attentional control prove most vulnerable, with marked capacity loss in high-pressure settings; 5) high acute stress disrupts sustained attention, causing lapses; 6) prolonged chronic stress erodes executive functions—planning and flexibility—and raises impulsivity; 7) stress lowers response inhibition, increasing interference errors; 8) imaging: acute stress down-regulates DLPFC and up-regulates amygdala/anterior cingulate cortex activity; 9) neurochemistry: elevated cortisol, norepinephrine, and dopamine weaken prefrontal control; 10) electroencephalography: diminished P300 amplitudes indicate reduced attentional resources; 11) psychological interventions (mindfulness and cognitive restructuring) ease anxiety and bolster attention/flexibility; 12) physiological techniques (breathing drills and progressive muscle relaxation) reduce heart rate and cortisol, improving adaptation; and 13) transcranial direct current stimulation shows promise for restoring attentional control and decision-making under stress.

Conclusion

Stress-related neuroendocrine shifts weaken prefrontal control, eroding athletes' attention, working memory, and executive skills. Future studies should track long-term and individual effects and develop targeted, multi-modal countermeasures.

sciforum-158225: Executive Functions and Decision-Making in Tunisian Pathological Gamblers

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Pathological gambling is a complex disorder characterised by a loss of control and the persistence of behaviour despite its negative consequences. It is frequently associated with executive deficits in cognitive flexibility, inhibition, and decision-making, which contribute to the maintenance of gambling behaviour and reduce adaptive capacities. A total of 120 participants aged between 16 and 80 years, all meeting the criteria for pathological gambling, were divided into three groups: adolescents, young adults, and older adults. Executive functions were assessed using the Wisconsin Card Sorting Test (WCST), the GoStop (Stop-Signal Task), and the Iowa Gambling Task (IGT). A one-way ANOVA revealed significant effects on cognitive flexibility (WCST) and inhibition (GoStop) but not on decision-making (IGT). Post hoc Tukey tests showed that older adults made more perseverative errors and completed fewer categories on the WCST than adolescents, while young adults occupied an intermediate position without significant differences. In the GoStop task, older adults exhibited poorer inhibitory control than adolescents. On the IGT, no significant group differences were found, although a trend towards lower scores was observed in older adults. Correlation analyses indicated that perseverative errors on the WCST were strongly and negatively associated with the number of categories completed and weakly related to the GoStop and IGT scores. These findings confirm the presence of executive deficits in pathological gamblers, emerging in young adulthood and worsening with age. They highlight the need for tailored interventions focusing on impulsivity management in younger adults and improving cognitive flexibility and inhibition in older individuals.

sciforum-163997: Long COVID-19 effects on the olfactory, gustatory, and cognitive functions

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The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has had a profound and lasting impact on global public health. Beyond the acute phase of infection, many people develop Long COVID-19, a condition characterized by the persistence or appearance of new symptoms weeks or months after recovery. Among the most common and debilitating symptoms are olfactory and gustatory disorders, which significantly impair quality of life weeks or months after recovery.

This study aimed to investigate the frequency and characteristics of self-reported symptoms across three distinct phases—pre-infection, acute COVID-19, and post-acute (Long COVID-19)—focusing on chemosensory disturbances and cognitive abilities. To this end, an anonymous online questionnaire was administered, incorporating validated instruments and specific questions regarding participants' clinical history. In addition, a subgroup of participants residing in Sardinia (Italy), specifically in the Cagliari area, underwent objective assessments of olfactory and gustatory functions using standardized tests integrating subjective reports with clinical experimental measures.

The results revealed a marked reduction in systemic symptoms from the acute to the post-acute phase; however, olfactory and gustatory impairments persisted in a substantial proportion of individuals, particularly among women and participants under 25 years of age. Our data show a significant correlation between chemosensory deficits and cognitive impairments. In conclusion, the results indicate that chemosensory dysfunction may serve as a potential clinical biomarker of Long COVID-19 and highlight the need for longitudinal studies to clarify the underlying pathophysiological mechanisms and guide the development of targeted therapeutic interventions.

sciforum-158380: Neuronal similarity in perceiving illusory and real rotation directional flips

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Introduction

Bistable perception occurs when stimuli present ambiguous sensory information that can be interpreted in two different ways, spontaneously changing in aware experience, and is widely used as an experimental approach in consciousness research. In the present study, we compared the neural activity induced by dynamic bistable stimuli that create illusory directional flips with that of rotating patterns changing direction.

Methods

Stimuli were spiral glass patterns of similar appearance. Six of them included a sequence of 1,000 different patterns, while the other six contained patterns rotating either clockwise or counterclockwise, changing direction unpredictably.

Thirty-six subjects participated in this study and had to press a joystick button whenever they perceived a change in the stimulus motion direction.

EEG was recorded at 26 positions. Epochs taken 1,000 msec before button presses were classified in two categories: illusory and real rotation directional flips. Representational similarity analysis (RSA) was applied using Pearson correlation. We examined the overall similarity within and across categories, its time course, and its spatiotemporal distribution (searchlight analysis).

Results

No significant differences in the overall neuronal similarity and its time course were found within or across categories, although real rotations showed the highest correlation. Category-specific information appeared earlier for illusory reversals than for real directional flips.

The searchlight analysis indicated differences in average correlation among brain regions and time windows for both types of directional flips' categories. Illusory flips induce lower similarity of neural activity but involved more brain regions than the real flips. Both types of flips displayed significant neural similarity across all electrodes approximately 250 milliseconds before the button press.

Conclusions

Directional transitions in bistable stimuli elicit neural activity that resembles real directional changes. However, illusory reversals recruit more widespread cortical regions and show distinct temporal dynamics, consistent with evidence that bistable perception engages distributed brain networks.

sciforum-158463: Neurotoxic effects of valproic acid in Wistar rats: transgenerational alterations after exposure of the first generation

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Introduction: Valproic acid (VPA) is a broad-spectrum anticonvulsant that is particularly effective against epilepsy, acting by increasing GABA (cerebral inhibitor) activity and blocking sodium channels. Despite its therapeutic benefits, VPA presents significant risks. **Objective:** This study aimed to evaluate the neurotoxic effects of VPA in Wistar rats. **Methods:** Forty-eight male and female Wistar rats were divided into six groups: (1 and 4) a control group, (2 and 5) a VPA 200 mg/kg group, and (3 and 6) a VPA 400 mg/kg group. The products were administered daily by gavage for 60 days. Wistar rats weighing between 100 and 300 g exposed to VPA and their offspring constituted our study population. Daily observations were made for three generations of Wistar rats. Behavioral, biochemical, cerebral histological, and reproductive parameters were evaluated, as well as 8-OH-dG dosage by PCR on one genetic region. **Results:** Observations revealed significant alterations across three generations. Wistar rats exposed to VPA exhibited brain alterations, reproductive dysfunction, and alopecia. In the second and third generations, our results showed brain atrophy, limb abnormalities, tumors, spina bifida, and histological alterations. Our results highlighted a significant decrease in biochemical parameters, stress, memory impairment, neurodevelopmental disorders, and reproductive system dysfunction. **Conclusion:** The abnormalities observed in the three generations of Wistar rats during our study reflect VPA-induced neurotoxicity at the doses studied after the exposure of the first generation. Our future work will consist of consolidating a neurotoxicity model based on APV.

sciforum-157036: Oxido-inflammatory changes in the amygdala exacerbates pain-related behaviours in chronically stressed diabetic rats: Ameliorative effect of *Sorghum bicolor* polyphenol extract

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Background: Chronic stress is a significant contributing factor to the advancement and complications of type 2 diabetes mellitus, notably the development of neuropathic pain. The amygdala, a key brain region regulating the emotional and affective aspects of pain, is susceptible to oxidative stress and inflammation. This study investigates the oxido-inflammatory changes in the amygdala and related pain behaviour in diabetic rats exposed to chronic unpredictable mild stress (CUMS) and the ameliorative effect of *Sorghum bicolor* polyphenol extract (SBPE).

Method: HFD/STZ-induced diabetic rats were subjected to CUMS and administered oral treatments of SBPE (200 mg/kg), metformin (250 mg/kg), or a combination of SBPE and metformin for a period of 28 days. Pain-related behaviours, inflammatory, oxidative stress and neurotransmitter-related parameters as well as histological changes in the amygdala were evaluated.

Results: Chronic stress exacerbates anxiety-like behaviour and neuropathic-like pain behaviour in diabetic rats, which was, however, reversed in SBPE and metformin-treated rats. SBPE ameliorated CUMS-induced significant elevation of TNF-α, IL-1β and myeloperoxidase in the amygdala brain tissue of diabetic rats. SBPE treatment significantly reduced malondialdehyde and nitrites and increased GSH, catalase, SOD, and GST in the amygdala. SBPE induced significant modulation of acetylcholinesterase, glutamic acid decarboxylase and arginase activities in the amygdala of diabetic rats. Distorted neuronal morphology of the amygdala in CUMS-diabetic rats was restored in SBPE-treated groups.

Conclusion: *Sorghum bicolor* polyphenol-rich extract relieves anxiety and neuropathic pain in chronically stressed diabetic rats possibly by inhibition of oxidative stress and inflammation in the amygdala.

sciforum-158160: Paradol Enhances Memory and Cognitive Function in an Amnesic Zebrafish Model

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Cognitive impairment is a defining feature of Alzheimer's disease (AD), a progressive neurodegenerative disorder characterized by deficits in memory, learning, and recognition. Developing natural compounds that can support or restore cognitive function is of critical importance in neuroscience research. Paradol, a bioactive compound derived from plants, exhibits potential neuroprotective properties that may influence cognitive performance. In this study, we investigated the effects of Paradol on cognitive function in adult zebrafish subjected to an Alzheimer-like model induced by okadaic acid (10 nM for 4 days). Zebrafish were randomly assigned to six experimental groups (n = 10 per group): control (dimethyl sulfoxide), galantamine (1 mg/L), okadaic acid alone, and okadaic acid with Paradol at 1, 3, or 6 µg/L. Paradol was administered via immersion every three days for seven days. Cognitive assessment focused specifically on processes relevant to Cognitive Neuroscience, including spatial memory measured with the Y-maze task and recognition memory evaluated with the Novel Object Recognition (NOR) test. Statistical analysis using one-way ANOVA with Tukey's post hoc test (p 0.05) revealed that okadaic acid significantly impaired both spatial and recognition memory. Treatment with Paradol at 3 and 6 µg/L significantly enhanced cognitive performance, increasing exploration of the novel arm in the Y-maze and preference for the novel object in NOR. These findings highlight the potential of Paradol to improve key cognitive processes in a preclinical model of AD, providing insights into memory restoration mechanisms and offering a promising avenue for future research in Cognitive Neuroscience.

sciforum-157320: Preliminary data regarding some cognitive effects of atypical antipsychotics in schizophrenia: monotherapy vesus polypharmaceutical approaches

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Introduction: Schizophrenia is a psychiatric disease characterized by deficiencies in multiple cognitive domains, such as attention, working memory, long-term memory, and learning, that are stable throughout the patient's life. Due to mainly focusing on typical symptomatology relief, in many cases, cognitive impairment is overlooked. However, the medication of choice often consists of antipsychotics, their combination being adjusted to the therapeutic response. Yet data regarding the cognitive effects of antipsychotics in schizophrenia is rather scarce and inconsistent. In this context, we aimed to investigate the impact of various combinations of atypical antipsychotics on the cognitive functions of schizophrenia patients. Methods: Thirty participants with schizophrenia were enrolled in the study (18 men and 12 women). The assessment of study participants was conducted by specialized medical personnel, and the application of the Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment Scale (MoCA) questionnaires aimed to verify cognitive parameters such as spatial–temporal orientation, attention, immediate and short-term memory, the capacity to perform concrete and abstract operations, motor skills, and language. Results: There were differences between the polypharmacy group and the monotherapy group in copying tasks, visuospatial orientation, and attention for both scales involved in this study. Conclusions: Our results suggested that the polytherapeutic approach based on a combination of atypical antipsychotics could lead to the significant improvement of cognitive functions, including spatial and visual orientation, general attention, and verbal memory.

sciforum-158551: The impact of age and sports experience on balance control: physiological and cognitive insights

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Introduction. Aging impairs the multisensory integration and cognitive control essential for posture regulation. Declining cerebellum–prefrontal cortex coordination disrupts attentional shifting, working memory, and executive flexibility, weakening balance responses, slowing adaptation, and increasing instability. The stabilometric target test on a force platform assesses cognitive–motor interaction by analyzing attentional load and sensorimotor updating during posture control. This study aimed to characterize age-related changes in cognitive–motor integration and evaluate whether athletic training mitigates cortical decline and optimizes sensorimotor–cognitive function.

Methods. Forty-four participants were divided by age (≤45 years, n = 24, median 36.5 years; >45 years, n = 20, median 55.5 years) and athletic experience (nonexperienced, n = 24; experienced, n = 20). All performed a stabilometric target test on the Stabilan-01-2 platform (OKB Ritm, Taganrog, Russia) on firm and soft surfaces, with stabilogram recording and frequency analysis. Statistical significance (p 0.05, q 0.1) was assessed using Student's t-test or the Mann–Whitney U test with FDR-BH correction. Athletic experience effects were analyzed via regression, adjusting for age, sex, and training.

Results. Age and athletic experience significantly explained the variation in stabilographic data (p 0.05 and p 0.01, respectively) on a firm surface. Age was negatively associated with stability parameters (r 0), while athletic experience showed positive associations (r > 0) during the stabilometric target test engaging attention and postural control. On a soft surface, no intergroup differences were observed, as support deprivation triggers adaptive mechanisms that maintain posture regardless of athletic training level. Regression analysis revealed that athletic experience was significantly associated with increased stability in both age groups.

Conclusion. Athletic experience enhanced stabilometric performance, improving visual–postural control and compensating for age-related decline. These results suggest stabilometric improvements are linked to training cognitive mechanisms, particularly attention and spatial orientation.

Session 5. Neurorehabilitation

sciforum-164381: The Efficacy of Telerehabilitation in Enhancing Motor Recovery and Quality of Life in Patients with Neurological Disorders: A Systematic Review

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Neurorehabilitation is undergoing a digital transformation, with telerehabilitation emerging as a critical tool for ensuring continuity of care. This systematic review aims to evaluate the efficacy of remote rehabilitation interventions compared to conventional in-clinic therapies in improving motor functions and quality of life for patients with neurological conditions, such as stroke and multiple sclerosis. A systematic search was conducted across PubMed, Scopus, and Web of Science for randomized controlled trials (RCTs) published between 2020 and 2025. The inclusion criteria focused on adult populations undergoing digital or remote neurorehabilitation interventions, utilizing outcomes such as the Fugl-Meyer Assessment (FMA), Berg Balance Scale (BBS), and various Quality of Life (QoL) metrics. Preliminary analysis of the selected studies indicates that telerehabilitation is at least as effective as face-to-face therapy in promoting neuroplasticity and motor recovery. Key findings suggest that digital platforms enhance patient adherence due to the convenience of home-based training. Furthermore, interventions incorporating biofeedback and synchronous monitoring showed significantly higher improvements in functional independence compared to non-monitored home exercises. Telerehabilitation represents a viable and evidence-based alternative to traditional methods, addressing geographical and mobility barriers. However, the heterogeneity of digital platforms and the need for standardized protocols remain challenges. This review highlights the potential of integrating telemedicine into routine clinical practice to optimize functional outcomes and long-term disability management.

sciforum-158276: Assessment of knee muscle performance in para-athletes with unilateral transtibial amputation

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Introduction. Unilateral lower-limb amputation often leads to long-term asymmetry in knee muscle function, which may persist despite rehabilitation. The magnitude and profile of impairment depend on rehabilitation timing and which muscle groups are most affected. Earlier prosthetic rehabilitation is typically associated with better functional preservation, whereas delays can lead to selective deficits – particularly in muscles that contribute to high-velocity movements. Flexors and extensors may recover differently, with consequences for joint stability and movement efficiency. We therefore aimed (I) to determine whether knee flexors or extensors show better functional preservation in unilateral transtibial para-athletes and (II) test whether time-to-prosthetic gait and movement velocity are related to inter-limb asymmetry (ILA).

Methods. Eight male unilateral transtibial para-athletes (classifications C2–C4; height 172.9±7.2 cm; body mass without prosthesis 68.9±7.9 kg; BMI 22.9±1.9) were tested. The mean interval from amputation to first prosthetic gait was 4.4±1.5 months (n=8). Concentric knee flexion/extension was assessed on an Isomed 2000 dynamometer at 60, 180, 240, and 300°·s^{-1}. Four athletes were tested bilaterally and four were tested on the intact limb only (retained to augment intact-limb reference values). Outcomes are listed: peak torque (PT, Nm; Nm·kg^{-1}), hamstrings-to-quadriceps ratio (H/Q, %), and inter-limb asymmetry (ILA, % = |PT_intact−PT_prosthetic|/PT_intact×100). Anthropometrics are reported as mean ± SD; isokinetic outcomes are reported as median [IQR]. The exploratory Spearman method tested the delay–ILA association.

Results. Prosthetic-side torque deficits were evident across all velocities. ILA was velocity-dependent, with minima at 240°·s^{-1} (flexion) and 300°·s^{-1} (extension), alongside pronounced individual outliers. H/Q was slightly higher on the prosthetic limb, consistent with relatively preserved flexors.

Conclusion. The profile indicates a selective extensor deficit and clear velocity specificity; prioritize early, targeted, velocity-specific extensor strengthening on the prosthetic side. From the results, earlier initiation of prosthetic gait was associated with lower asymmetry.

sciforum-124622: Artificial Intelligence- and Technology-Enhanced Neurorehabilitation: Innovations in Traumatic Brain Injury and HIV-Associated Cognitive Impairment

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Introduction:

Traumatic brain injury (TBI) and HIV-associated neurocognitive impairment (NCI) are the leading causes of long-term disability, with effects on cognitive, motor, and psychological functioning. Even with improved acute care and antiretroviral therapy, successful and individualized neurorehabilitation is a continuing need. New technologies, specifically artificial intelligence (AI), virtual reality (VR), and telerehabilitation platforms, present new potential to enhance outcomes through individualized interventions and decision-making in real-time.

Methodology:

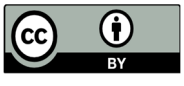
This review synthesizes evidence from recent clinical trials, AI model performance analysis, and neurorehabilitation trials for TBI as well as HIV-related NCI. The AI algorithms developed for TBI were compared on the basis of diagnostic accuracy, prognostic models, and rehabilitation optimization. The computational neurorehabilitation paradigms were evaluated for their capacity to merge the digital flow of data among patients, clinicians, and predictive systems. Cognitive rehabilitation devices, such as immersive games and distant training systems, were assessed regarding accessibility, customization, and compliance in HIV-impacted populations.

Results:

AI models exhibited up to 95.6% accuracy in mortality and functional outcome prediction in TBI. AI-enhanced neuroimaging enhanced diagnostic sensitivity. Non-invasive brain stimulation, VR, robot-assisted therapy, and computer-based training were partially effective in TBI rehabilitation. Technology-enabled cognitive rehabilitation enhanced adherence and motivation in HIV-related NCI, especially with gamified and immersive settings. Yet disparities in digital access and the absence of large-scale trials were observed.

Conclusion:

AI and computational neurorehabilitation have important potential to propel individualized therapy in TBI and HIV-related NCI. Satisfying ethical issues, interoperability of data, and availability will be crucial for their broader clinical deployment. Future research should emphasize solid, large-scale trials to prove and extend these technology-based interventions.



sciforum-164188: Brazilian adaptation of the “iHELP Stroke: Improving Health and Lifestyle Programme” and feasibility study

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Introduction: Recurrent stroke contributes to the high global burden of this disease. Low- and middle-income countries (e.g., Brazil) are disproportionately affected by stroke and have limited access to secondary prevention. A theoretically informed intervention for risk reduction post-stroke (“iHELP Stroke: Improving Health and Lifestyle Programme”) was recently co-designed in a high-income country (Ireland). Before implementation in Brazil, adaptations were required due to cultural, social, and economic differences. This study aims to describe the adaptation of the “iHELP Stroke” to the Brazilian context and assess its feasibility among Brazilians with stroke.

Methods: A mixed-methods study was conducted following ADAPT guidance. Phase I included two stakeholder panels (15 experts and 10 stroke survivors) to adapt the intervention for adequate reach, effectiveness, cost-effectiveness, adoption, implementation, and sustainability. Phase II involved a feasibility study with 10 Brazilian individuals post-stroke, evaluating recruitment, retention, attendance, acceptability and satisfaction, and clinical outcomes (achievement of behavior change goals). Descriptive statistics were applied.

Results: Phase I identified 11 contextual themes, leading to adaptations including translation into Brazilian Portuguese, renaming as “iVIDAVC: intervenção para melhorar a saúde e o estilo de vida pós-AVC”, and the addition of an educational session. Phase II showed a recruitment rate of 13.7%, high retention (80%) and attendance (88.9%), strong acceptability and satisfaction, and complete achievement of primary goals by half of participants. Adjustments are needed: sessions with guest professionals exceeded planned duration, one participant found the eight-week programme too short, and one group session included only one participant.

Conclusions: The “iHELP Stroke” program was successfully adapted to the Brazilian context, and the resulting “iVIDAVC” program demonstrated feasibility. The next phase (a pilot study), also with an appropriate sample size, should test the full 14-week programme, ensure repeated participation of invited professionals, include at least four participants per group, and refine outcome measures for comprehensive evaluation.

sciforum-158621: Determination of the Anaerobic Threshold Using Integrated Real-Time Analysis of Electromyographic, Oximetric, and Heart Rate Variability Data

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Modern methods for determining the anaerobic threshold (AT) are primarily based on laboratory exercise tests involving blood lactate concentration measurements, which limits their applicability in field training and clinical settings. The development of physiological signal recording technologies enables noninvasive and continuous monitoring of parameters associated with metabolic transitions, including muscle electrical activity, tissue oxygenation, and heart rate variability (HRV).

This study focuses on the second metabolic threshold (LT2/VT2), validated against lactate-defined LT2. The aim was to develop and evaluate a real-time software system for determining LT2 through integrated analysis of electromyographic (EMG), near-infrared spectroscopy (NIRS), and nonlinear HRV metrics (DFA-α1). Although DFA-α1 is commonly used to assess the first lactate threshold (LT1), in this study it was combined with EMG and NIRS features to detect the second threshold. Twenty-five healthy recreational and professional cyclists participated in the study. The developed system integrates data streams from Zephyr, Delsys, and Moxy Monitor devices, providing synchronous signal processing, dynamic index computation, and visualization within a unified interface. Data streams were time-aligned on the host system, artifact-suppressed, and averaged in a moving window to ensure real-time analysis.

Experimental results showed 85% agreement with lactate-defined LT2, with a model update interval of 30 seconds (i.e., the system processes accumulated data and updates the LT2 determination every 30 seconds), supporting feasibility and comparability of the proposed approach in comparison with standard lactate-based testing.

The proposed method offers promising applications in sports physiology, training monitoring, and particularly in neurorehabilitation, where personalized aerobic load adjustment is required but laboratory testing is often infeasible. The use of noninvasive sensors and automated signal processing may enhance the effectiveness of rehabilitation programs and accelerate patient recovery.

sciforum-157395: From Brain Surgery to Back Pain: Exploring the Role of Probiotics in Quality of Life and Pain Relief

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Probiotics are live microorganisms that, when administered in adequate amounts, confer a health benefit on the host. Their use has become increasingly common as they help maintain a balanced gut microbiota and support immune function. In recent years, attention has also turned to the communication pathways between the gut and the brain, which suggest that probiotics might influence not only digestion and immunity but also inflammation, pain, and recovery after neurological injury.

The aim of this review was to explore how probiotic supplementation affects quality of life in patients after brain surgery and whether it plays a role in reducing lower back pain. We searched for scientific studies in the international databases PubMed, ScienceDirect, MEDLINE (EBSCOhost), CINAHL Ultimate, and Wiley Online Library. The search and selection process was shown using a PRISMA flow diagram, and the quality of the studies was assessed according to an eight-level hierarchy of evidence. A thematic synthesis was then carried out.

Altogether, eleven studies met the inclusion criteria—five dealing with lower back pain and six focusing on patients after brain surgery. The results show that probiotics may have some positive effects, such as reducing inflammation and infection risk, shortening hospital stay, and helping bowel function recover faster. However, the evidence for improving overall quality of life or reducing back pain is still limited and inconsistent.

In conclusion, probiotics show potential as supportive agents in improving recovery and general health, but more well-designed clinical studies with larger samples, defined strains, and standardized evaluation methods are needed to better understand their role in pain management and postoperative recovery.

sciforum-156347: Perineural Injection Treatment - Dextrose for Chronic Migraine: A Case Study Analysis

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Objective: To explore the effectiveness of perineural injection treatment with dextrose (PITD) as a cost-effective and accessible alternative for managing refractory chronic migraine in low- and middle-income countries (LMICs).

Case Report: A single-patient case study was conducted involving a 30-year-old male diagnosed with chronic migraine complicated by medication-overuse headache, based on the International Classification of Headache Disorders, 3rd edition (ICHD-3). The patient had a one-year history of right-sided daily headaches refractory to over-the-counter analgesics and conventional prophylactic therapy.

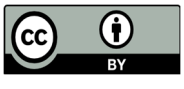
Comprehensive neurological and physical examinations were performed, followed by diagnostic investigations including electroencephalography (EEG), magnetic resonance imaging (MRI), and transcranial Doppler (TCD), all of which showed no structural or vascular abnormalities.

The therapeutic intervention consisted of biweekly perineural injection treatment with 5% dextrose (PITD), administered bilaterally to the greater and lesser occipital nerves (GON and LON). Each session involved 5 cc of 5% dextrose per side, guided by anatomical landmarks and palpation of tender points.

Concurrent pharmacological management included topiramate (50 mg twice daily) and low-dose amitriptyline (12.5 mg at bedtime). A rescue regimen of paracetamol and oral diazepam was provided for breakthrough attacks. The treatment period lasted three months, with progress assessed using the Numerical Pain Rating Scale (NPRS) at each follow-up visit.

Discussion: The patient reported significant improvement in pain severity, with the Numerical Pain Rating Scale (NPRS) decreasing from 9 to 5 over the three-month treatment period. Sustained relief was achieved by the fifth PITD session, with stabilization of symptoms and no structural abnormalities detected on follow-up MRI.

Conclusions: PITD may offer a viable, low-cost treatment for chronic migraine in resource-constrained settings, addressing both peripheral and central mechanisms of pain. This approach could reduce reliance on expensive treatments and improve patient outcomes in LMICs. Further studies are needed to validate its efficacy and long-term benefits in larger populations.



sciforum-164189: Prioritizing components for a healthy lifestyle intervention post-stroke: a cross-country Patient and Public Involvement-based descriptive analysis

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Introduction: Adopting healthy lifestyle behaviors is vital for stroke secondary prevention. However, low- and middle-income countries often lack specific guidelines. High-income countries, such as Ireland, have more extensive research and resources, whereas Brazil, which has a higher stroke burden, frequently relies on external evidence. Understanding the perspectives of individuals post-stroke is essential for implementing contextually relevant interventions. Therefore, this study aims to identify similarities and differences in how individuals post-stroke participating in Patient and Public Involvement (PPI) panels in Ireland (high-income) and Brazil (middle-income) prioritize components to be included in an intervention designed to support the adoption of healthy lifestyle behaviors.

Methods: A descriptive, cross-sectional study was conducted with two PPI panels (five individuals post-stroke from each country), who rated core components across six behaviors (healthy diet, medication adherence, mood management, physical activity participation, safe alcohol consumption, and smoking cessation) as "definitely important", "maybe important", or "not important". Frequencies of responses were calculated per behavior. A cross-country similarity was defined when a majority (>50%) from both panels rated a component the same ("definitely important" or "not important"); otherwise, perspectives were considered different. Descriptive statistics were used.

Results: Most components were rated "definitely important" by >50% in both panels, except for healthy diet. No component was largely deemed "not important". Differences emerged: only the majority of the Irish PPI panel rated "Have medications review with pharmacist/general practitioner" as "definitely important"; only the Brazilian panel rated all healthy diet, physical activity participation, and smoking cessation components as "definitely important". Only in Ireland did most PPI members rate 18-50% of the components across all behaviors as "maybe important".

Conclusions: Similarities point to core priorities for post-stroke lifestyle interventions across contexts, while differences highlight the need for cultural adaptation when transferring interventions from high- to middle-income countries.

sciforum-164175: Telehealth intervention involving the HEARTS Technical Package and the additional use of an activity monitor to increase physical activity level post-stroke: a feasibility randomized controlled trial

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Introduction: Physical inactivity remains common post-stroke. Although behavior change interventions improve physical activity, many require home visits or Internet access. Telephone calls and activity monitors are low-cost telehealth options, but evidence for monitor use post-stroke is limited. Integrating monitors into multifaceted strategies (e.g., the 5As brief intervention from the HEARTS Technical Package) may enhance outcomes. This study aimed to assess the feasibility of the 5As brief intervention with versus without a physical activity monitor and to explore changes in physical activity levels to inform a future randomized controlled trial (RCT).

Methods: This phase 1 feasibility RCT, with concealed allocation and blinded assessments, was conducted in a university laboratory (Belo Horizonte, Brazil). Twenty-four individuals in the chronic phase post-stroke were included (12 per group) and received the 5As brief intervention from the HEARTS Technical Package. The experimental group (EG) additionally used an activity monitor (Mi Band 7® Smartwatch). Outcomes included feasibility indicators and physical activity level, analyzed descriptively.

Results: Trial feasibility was supported by recruitment (36.4%), retention (83.3% in both groups), and high attendance (99.1% in the EG and 97.0% in the CG). No adverse events were reported. Attrition remained a challenge, with 66.7% of EG participants (n=8) and 50.0% of CG participants (n=6) completing all follow-up assessments. Main costs were related to transportation for in-person assessments. Physical activity levels increased clinically in both groups, with a large within-group effect in the EG (Cohen's d = 1.01) and a moderate effect in the CG (Cohen's d = 0.76).

Conclusions: Both interventions were feasible and resulted in clinical improvements in physical activity levels post-stroke, with greater effects observed in the EG. These findings support the design of a future, larger RCT, with attention to retention strategies, follow-up procedures, and formal evaluation of the added value of physical activity monitor use.

Session 6. Neurotechnology and Neuroimaging

sciforum-157434: Differentiation of subtypes of voluntary movements

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Introduction: Emergency department providers may be challenged by the presentation of patients who exhibit movement abnormalities that could indicate conditions requiring interventions with potential morbidity and mortality. Crucially, interventions for potential neurological diseases that require immediate intervention (e.g. stroke) are contraindicated in patients exhibiting movements that may be voluntary (e.g. emotional expressions and fabricated symptoms) or functional (e.g. functional movement disorders such as functional tremor or psychogenic nonepileptic seizures). This diagnostic challenge is amplified when evaluating patients who are unable to provide symptom history, so information provided by accompanying persons requires confirmation by corroborating sources. Since accurate and rapid classification of these movement subtypes is essential for proper diagnosis and treatment of patients, we hypothesize that technology incorporating the neural underpinnings of voluntary movements provides a foundation for differentiating subtypes of movements. This difference and these signatures may be quantifiable: Voluntary movements, reflecting complex and specific planning, demonstrate spatial and temporal precision and accuracy, motor smoothness, and longer preparation and execution time.

Methods: We propose that kinematic analysis using three-dimensional position data from three sensors on the thumb, little finger, and wrist can effectively capture these unique temporal and spatial characteristics, allowing differentiation in subtypes of voluntary movements. This protocol and device setup are being implemented to generate preliminary data.

Results: We propose that an algorithm for voluntary movements may appear as follows:

Voluntary movements = The presence of at least one of the following: + malingering + factitious disorder + ululation + applause + ...

Conclusion: Our protocol examines the relationship between the neural underpinnings of motor planning in quantifiable kinematic differences among voluntary movements. This distinction, captured by simple, multi-sensor analysis, may provide a foundation for objective and novel diagnostic aid in complex movement disorders and symptoms. This protocol may be a valuable tool to monitor patients participating in clinical trials.

sciforum-163877: Learning-Dependent Modulation of Corticospinal Excitability During TMS-Based Neurofeedback

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Transcranial magnetic stimulation (TMS) provides a direct and reliable method for probing corticospinal excitability via motor evoked potentials (MEPs). While neurofeedback (NF) approaches using EEG or fMRI have demonstrated learning-related neural modulation, it remains unclear whether repeated NF based on TMS-elicited MEPs can induce stable, learning-dependent changes in corticospinal output across multiple training sessions.

In this study, we investigated longitudinal changes in corticospinal excitability during TMS-based neurofeedback training. Twenty-two healthy adults were randomly assigned to a feedback (FB) group or a control (CON) group. Participants performed motor imagery-based mental practice over six training days (three sessions per week for two weeks). During each trial, a single TMS pulse was delivered over the left primary motor cortex at a fixed timing, and MEPs were recorded from the right first dorsal interosseous muscle. In the FB group, trial-by-trial normalized MEP amplitudes were visually presented to participants as neurofeedback, whereas the CON group performed identical training without feedback.

Repeated-measures analysis revealed a significant interaction between group and training day for normalized MEP amplitudes. The FB group exhibited a consistent increase in corticospinal excitability from Day 2 through Day 6 relative to baseline, indicating early acquisition and maintenance of neurophysiological modulation. In contrast, the CON group showed a significant increase in MEP amplitude on only a single training day, suggesting limited learning without feedback.

These findings demonstrate that TMS-based neurofeedback can facilitate learning-dependent modulation of corticospinal excitability across repeated sessions. The results highlight the potential of MEP-guided neurofeedback as a powerful experimental framework for studying motor system plasticity and neural self-regulation, and as a methodological foundation for future neurophysiological and brain-stimulation-based research.

sciforum-165926: Machine Learning in Neuroscience Research: A Systematic Review of Predictive and Mechanistic Models

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The application of machine learning in neuroscience has expanded markedly in recent years, propelled by advances in data acquisition technologies and the proliferation of large-scale neural, imaging, and behavioral datasets. Machine learning techniques have become indispensable for capturing complex, high-dimensional patterns inherent in brain data; however, their deployment varies significantly in the extent to which they prioritize predictive performance over mechanistic insight. This systematic review offers a comprehensive synthesis of ML-based approaches in contemporary computational neuroscience, with a particular focus on the conceptual and methodological distinction between predictive models, aimed at optimizing the decoding or forecasting of neural and behavioral outcomes, and mechanistic models, which seek to elucidate the computational principles and biological architectures underpinning brain function. A systematic review of the peer-reviewed literature was conducted, encompassing studies that applied machine learning techniques to the analysis of neural data derived from neuroimaging, electrophysiological recordings, cellular-level measurements, and multimodal experimental paradigms. The extant literature reveals a strong dominance of predictive modeling, typically utilizing supervised learning and deep neural networks to classify brain states, decode experimental conditions, or predict cognitive and behavioral phenotypes. Conversely, mechanistic machine learning models, often grounded in computational neuroscience traditions such as dynamical systems theory, probabilistic inference, and network modeling, remain comparatively underrepresented, yet they offer essential explanatory value by linking algorithmic components to biophysical mechanisms and system-level dynamics. Emerging hybrid approaches aim to reconcile predictive accuracy with mechanistic transparency, though they remain methodologically heterogeneous and are often limited by insufficient validation. This review delineates critical challenges confronting machine learning in neuroscience, including the opacity of complex models, limited reproducibility, and difficulties in cross-scale integration. It concludes by advocating the development of integrative modeling frameworks that transcend the prediction–explanation dichotomy, thereby fostering a more unified, biologically grounded understanding of brain function.

sciforum-164191: Normative Convolutional Neural Network Modelling of Structural MRI for Personalised Neuroimaging

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Introduction

Deep learning methods in neuroimaging have largely focused on population-level classification and group-based inference, limiting their applicability for individualised clinical interpretation. Convolutional neural networks (CNNs) are well suited to modelling complex spatial patterns in structural MRI; however, their use for single-subject analysis remains limited. This study introduces a technically novel normative CNN framework designed to enable personalised, single-subject interpretation of T1-weighted structural MRI without reliance on diagnostic group comparisons or case–control statistics.

Methods

A three-dimensional convolutional autoencoder was implemented to learn normative neuroanatomical representations from healthy adult T1-weighted MRI data. Approximately 100 scans were selected from the publicly available IXI dataset to construct the normative reference model. Images underwent standard pre-processing including skull stripping, spatial normalisation, and intensity standardisation. The trained CNN was subsequently applied to individual subjects to generate voxel-wise reconstruction error maps, representing deviations from learned normative structure. Quantitative regional deviation scores were computed by aggregating voxel-level errors within anatomically defined brain regions, enabling subject-specific neuroanatomical profiling.

Results

The proposed framework produces continuous, voxel-wise deviation measures across the entire brain volume from a single T1-weighted MRI scan. Reconstruction error maps provide spatially localised representations of neuroanatomical variability relative to normative structure learned from the IXI reference cohort. Regional aggregation yields quantitative subject-specific deviation profiles, supporting interpretable individual-level assessment. Preliminary analyses demonstrate stable model training and consistent deviation patterns across healthy reference scans, indicating the technical feasibility of CNN-based normative modelling for single-subject inference.

Conclusions

This work presents a single-subject CNN framework for personalised structural MRI analysis. By shifting deep learning applications from group-level classification to normative individual-level deviation mapping, the proposed approach supports personalised medicine and precision neuroimaging paradigms. The framework provides a foundation for future large-scale validation, longitudinal modelling, and clinical translation of personalised neuroimaging methodologies.

sciforum-160206: Protocol for Thalamocortical Functional Connectivity and Loss of Consciousness Investigation in Children with Epilepsy Using Stereo-EEG Data

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Introduction:

Seizure-related loss of consciousness (LOC) in pediatric epilepsy significantly impacts safety, cognitive development, and quality of life. The thalamus is believed to play a central role in sustaining consciousness via its integrative cortical connectivity. Disruption of thalamocortical networks may underlie impaired awareness during seizures, yet mechanistic insights remain limited. Stereo-electroencephalography (sEEG) offers high-resolution intracranial recordings that enable in vivo exploration of these neural dynamics. Here, we present a protocol designed to systematically evaluate thalamocortical functional connectivity during seizures with and without LOC in pediatric patients with drug-resistant epilepsy. This protocol serves as a foundation for future hypothesis-driven investigations and network-targeted therapeutic strategies.

Methods:

Postoperative CT scans are co-registered with preoperative MRI to localize and label electrode contacts. sEEG data are exported from the Natus system into Brainstorm for preprocessing and quantitative analysis. Recordings include at least 10 minutes of pre-ictal, ictal, and post-ictal data. Seizures with and without LOC are identified and time-marked using synchronized video-EEG. LOC severity is scored independently by two experienced reviewers. Functional connectivity is assessed via Coherence (Coh) and maps are generated to visualize thalamocortical network dynamics.

Results:

Fourteen pediatric patients underwent thalamic sEEG implantation. Etiologies included focal cortical dysplasia, periventricular nodular heterotopia, neonatal HIE, and non-lesional epilepsy. Pre-implant hypotheses included temporal, frontal, insular, and multifocal seizure onset. Thalamic sampling included anterior and pulvinar nuclei. Interictal thalamic abnormalities were present in most of the patients. A protocol of the analysis was created and will be presented as a flow chat with the major analysis steps in the poster.

Conclusions:

This protocol-based approach enables rigorous investigation of thalamocortical connectivity and its role in LOC during epileptic seizures. Ongoing analyses will evaluate frequency-specific functional connectivity associated with altered awareness. Findings from this study may enhance patient-specific neuromodulation strategies and advance our understanding of consciousness disruption in epilepsy.

sciforum-144451: Pulsed arterial spin labeling MRI insights: Sevoflurane's effects on moyamoya disease and cerebral ischemia

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Background: Few studies have explored whether sevoflurane impacts cerebral vascular reactivity (CVR) or increases postoperative cognitive dysfunction in ischemic moyamoya disease (IMMD). We aimed to evaluate the effects of sevoflurane anesthesia on cerebral blood flow (CBF) and CVR in IMMD.

Methods: This prospective cohort study recruited 15 patients with IMMD and 17 with intraspinal space-occupying lesions, as healthy controls (HCs), between September 2023 and March 2024 in Peking University International Hospital. CBF in the whole brain and anterior (ACA), middle (MCA), and posterior (PCA) cerebral arteries was measured using 3.0 T magnetic resonance imaging (MRI) with pulsed arterial spin labeling (PASL) sequences. Measurements were taken in the awake and sevoflurane-anesthetized states (sedation depth BIS 40-60). CVR was then calculated. PASL data were processed using SPM8 and ASLtbx. Continuous variables were compared using t-tests; categorical variables were compared using chi-squared tests.

Results: Under sevoflurane anesthesia, CBF and CVR decreased in IMMD (P0.05) in the whole brain and bilateral ACA, MCA, and PCA regions. Whole-brain CBF was higher in IMMD than in the HCs pre-anesthesia and decreased post-anesthesia (P 0.05); there was little change pre- and post-anesthesia in the HCs (P > 0.05). CVR differed pre- and post-anesthesia in the IMMD group (P0.05) in all arteries except the left MCA. No significant difference in CVR was observed in the HCs pre- and post-anesthesia.

Conclusions: PASL-MRI is a feasible, non-invasive, quantitative tool for assessing global and regional CBF changes in mechanically ventilated patients under anesthesia. Sevoflurane anesthesia may further reduce CVR in IMMD, potentially increasing cerebral ischemia risk during and after surgery. Inhalational anesthetics should be carefully selected for these patients. Alternative drugs, or techniques that optimize CBF regulation, should be considered to improve postoperative recovery and long-term outcomes.

Session 7. Systems Neuroscience

sciforum-142241: The Tri-Electrical Axis: Integrating Heart, Brain, and Gut Electrophysiology in the Age of AI

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The heart, brain, and gastrointestinal tract have long been examined as separate electrophysiological domains, each with its own clinical tools and diagnostic language. However, emerging evidence points to a deeper, bioelectrically coordinated relationship among these systems—one that has remained largely unexplored in both research and practice. This work introduces the **Tri-Electrical Axis (TEA)**, a novel physiological framework proposing that the cardiac, cerebral, and enteric systems form a synchronized regulatory network governed by shared electrical patterns. Rather than treating ECG, EEG, and electroenterographic (EEnG) signals as isolated modalities, TEA emphasizes their dynamic interactions and latent harmonies. Initial analyses of co-registered cardiac and cerebral signals reveal reproducible phase coupling and feedback loops suggestive of an integrated bioelectrical substrate. These findings support the hypothesis that multi-organ synchrony may play a role in homeostasis, adaptive responses, and even early disease manifestation. The upcoming inclusion of enteric electrophysiology is expected to complete this triadic structure, offering a richer model of systemic physiology. More than a methodological innovation, TEA represents a conceptual shift: it challenges organ-specific silos and reimagines human physiology as an interdependent, signal-driven system. Such a model holds transformative potential for preventive medicine, enabling earlier detection of dysfunctions that manifest first in cross-organ desynchronization rather than isolated pathology. By uncovering this hidden electrical axis, the TEA framework offers a foundation for future diagnostics, dynamic monitoring tools, and integrative research across cardiology, neurology, and gastroenterology. It is not only a new way to analyze signals—but a new way to understand life.

sciforum-161849: A Systems Biology Approach to unravel the common genes and pathways in Amyotrophic Lateral Sclerosis and Traumatic Brain Injury

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Background: Amyotrophic lateral sclerosis (ALS) and traumatic brain injury (TBI) are distinct neurological disorders that share overlapping molecular and cellular mechanisms, including neuroinflammation, oxidative stress, and synaptic dysfunction.

Methods: This study employs a systems biology approach to identify common genes and pathways linking ALS and TBI. Differentially expressed genes (DEGs) from publicly available transcriptomic datasets (GSE89866 for TBI and GSE153960 for ALS) were integrated and analyzed through network-based and enrichment analyses. Protein–protein interaction (PPI) networks were constructed to highlight key hub genes and regulatory modules shared between both conditions.

Results: A total of 3885 DEGs were identified in ALS (GSE153960) whereas 449 DEGs were obtained in TBI (GSE89866). Total number of common overlapping DEGs between the two datasets were 20, out of which 13 common DEGs were upregulated in both the cases, while 7 DEGs were downregulated. Important upregulated genes were MAFB, IL13RA1, PLBD1, CYP1B1, FBP1, TLR5, and RAB20. Similarly, key downregulated genes were ZNF541, DNAI2, NYAP1, TRIM17, and TRPV6. The functional enrichment analysis indicated that these key genes are involved largely in the innate immune system, muscle contraction, aldosterone synthesis and secretion, cellular responses to stimuli, cytosolic DNA-sensing pathway and cellular senescence.

Conclusion: These findings provide novel insights into shared molecular mechanisms and may contribute to identifying potential therapeutic targets for neuroprotection and disease modification in ALS and TBI.

sciforum-159252: Interactive Brain Interface for Multimodal EEG Visualization and Disease-Specific Neural Dynamics

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Understanding how brain activity changes across neurological and neurodevelopmental disorders requires tools that can reveal patterns hidden in complex EEG data. Conditions such as epilepsy, Alzheimer's disease, dementia, and autism often produce distinct alterations in neural oscillations and connectivity, but these signatures can be difficult to interpret in real time. In this work, we present an interactive brain interface designed to visually explore disease-specific EEG dynamics through integrated spectrograms, topographic maps, and connectivity graphs.

Our system combines classical signal-processing techniques with computational modeling to generate a multi-layer representation of ongoing brain activity. EEG segments are analyzed to extract spectral features, inter-electrode coherence, and spatial activation patterns. The interface simulates key biomarkers for each condition, including epileptic spike–wave discharges, Alzheimer-related reductions in alpha power, dementia-associated slowing, and atypical connectivity profiles observed in autism. A dedicated seizure module models the rapid synchronization that occurs during ictal events, highlighting propagation pathways across the scalp. All visual components, EEG waveforms, frequency-band power, scalp topomaps, and graph-based networks, update continuously, allowing users to observe how brain states evolve over time.

Initial results demonstrate that the interface effectively captures meaningful differences between disorders, making high-dimensional EEG patterns easier to understand and compare. Epileptic simulations display strong bursts and dense network coupling, while neurodegenerative modes show weakened connectivity and spectral slowing. These visualizations offer an intuitive yet rigorous way to explore neural dynamics.

Overall, the project illustrates how computational neuroscience, mathematical modeling, and interactive visualization can be combined to create an accessible tool for research, education, and potential clinical support. This interface provides a flexible platform for studying how neural circuits behave across diverse brain conditions and how their dynamics relate to cognition and behavior.

sciforum-158301: Recurrence Network Analysis Uncovering Biomarkers of Depression from Nonlinear Dynamics underlying EEG Signals

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Introduction: Major Depressive Disorder (MDD) is one of the most prevalent and severe mental health condition, affecting millions of individuals worldwide and often leading to significant cognitive and emotional dysfunction. MDD is characterized by persistent low mood, loss of interest or pleasure, and impaired cognitive and physical functioning. Our objective is to apply advanced complexity and recurrence network analysis approaches to investigate underlying changes in functional brain connectivity using electroencephalographic (EEG) signals.

Methods: We used the multi-modal open dataset for mental-disorder analysis (MODMA), consisting of 128-channel resting-state EEG recordings of individuals with MDD and those of healthy controls. We have particularly focussed on channels in the frontal brain region as it is known to play a crucial role in emotional and cognitive processing. After pre-processing, power spectral density (PSD) and Lempel–Ziv (LZ) complexity analysis were applied to distinguish MDD subjects from healthy controls. We performed t-tests to identify significant channels. We then constructed recurrence networks to capture the nonlinear temporal dynamics of EEG activity in these significant frontal channels. Then, we computed various network measures, including Recurrence Quantification Analysis (RQA), recurrence rate (RR), determinism (DET), and laminarity (LAM), to characterize the resulting recurrence network. Further, we performed functional cartography of nodes based on modularity to quantify network hubs.

Results: The results revealed frontal asymmetry with several frontal channels exhibiting significant variations in oscillatory power and signal complexity in MDD. The recurrence networks represent the complex interrelationships among recurring patterns of EEG brain activity over time. We observed that functional cartography applied for hub classification in the constructed recurrence networks provides a powerful framework for quantitative analysis and unveiling network-based signatures underlying the nonlinear dynamics of EEG signals.

Conclusions: The identification of such EEG-based network markers could facilitate early detection and prediction of MDD severity.

sciforum-158088: Sensorimotor adaptation in virtual environments: The critical role of individual cognitive style

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Introduction: It has been established that the cognitive style of field dependence/field independence (FD/FI) significantly influences sensory reliance in postural control. Individuals with FD appear to rely more on vision, whereas those with FI prioritize vestibular and proprioceptive inputs. The current study aims to investigate the impact of this cognitive style on postural adaptation in complex tasks involving sensory deprivation and virtual reality.
Methods: A total of 41 participants aged 19–26 years were classified as either FD or FI based on the Gottschaldt Figures Test. Their postural stability was assessed using stabilometry (Stabilan-01-2) under various conditions: eyes open (EO), eyes closed (EC), virtual reality goggles (VR), hard surface (HS), and soft surface (SS).
Results: All participants showed impaired postural control during complex tasks. Individuals with FI showed enhanced stability when faced with either simple visual deprivation or proprioceptive limitation. In contrast, individuals with FD demonstrated increased destabilization in response to virtual reality on a firm surface, with a prolonged recovery time after visual deprivation. However, when virtual reality was combined with a soft surface, both groups exhibited immediate destabilization. Interestingly, FI individuals showed a slower recovery after the test, while FD individuals displayed a faster adaptation rate.
Conclusion: It has been shown that FI individuals use more effective strategies in sensory-deprived conditions. However, their strong reliance on internal cues makes them more susceptible to the combined effects of VR and proprioceptive deficits. It is crucial to consider a patient's cognitive style when prescribing VR-based therapy, as this can be critical in minimizing fall risks and optimizing rehabilitation protocols.
This study was supported by the Russian Science Foundation (grant №25-15-20048).

sciforum-160911: Task-dependent modulation of aftereffects during visuomotor adaptation

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Introduction Implicit motor adaptation is widely described as having a fixed ceiling of 12–15° that does not scale with perturbation size when explicit strategies are suppressed. We examined how this ceiling changes when participants experience two different visuomotor rotation magnitudes (24° and 48°) under gradual versus error-clamped schedules.

Methods Forty-one participants adapted to 24° and 48° rotations in counterbalanced order across two sessions. Perturbations were introduced either gradually (stepped increments designed to minimize the awareness of the visuomotor perturbation) or via error-clamp (constant clamped feedback that eliminates explicit aiming). Immediate aftereffects were measured during the first four no-feedback trials after each exposure.

Results In the gradual groups, aftereffects scaled strongly with the perturbation size (first exposure: 12.7° after 24° vs. 24.8° after 48°, t(19)=6.06, p0.001; second exposure: 24.4° vs. 16.3°). In the error-clamped groups, aftereffects remained ~11–14° regardless of whether participants had just experienced a 24° or 48° clamp (all ps>0.18).

Conclusions When visuomotor adaptation occurs gradually, implicit aftereffects are flexibly modulated based on the rotation size. When the adaptation occurs under the error-clamped condition, aftereffects are fixed at approximately 13° irrespective of the rotation size. These results indicate that the “fixed ceiling” effect known to follow implicit adaptation may be task dependent.

sciforum-155341: WM/GM ratio of mammalian spinal cord: insights from African hedgehog (*Atelerix albiventris*)

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Introduction. The mammalian spinal cord consists of myelinated axons forming the white matter (WM) and neurons forming the gray matter (GM). The ratio of WM to GM areas (WM/GM) is a well-known macroanatomical characteristic of the spinal cord. This ratio is lower within cervical and lumbar enlargements and higher between them (in the thoracic region). It also varies significantly across species: for example, in the upper lumbar segments, it is lower in rats but higher in horses. We hypothesized that this species-dependent variation might be (at least partially) explained by the degree of spinal cord ascension.

Methods. We assessed the level of SC ascension and WM/GM ratio of the spinal cord segments in the African hedgehog. Based on the literature data from other mammals (goat, European hedgehog, horse, human, mouse, opossum, pig, rabbit, rat, rhesus macaque, and mole), we performed phylogenetic correlations between the WM/GM ratio of the L1–L5 lumbar segments and the level of ascension, defined as the number of the vertebra where the 29th spinal cord segment was located.

Results. We found that in African hedgehog, similarly to European hedgehog, the spinal cord is highly ascended, and the WM/GM ratio in all lumbar segments is less than those in other mammalian species. The phylogenetic correlation was significant for all lumbar segments studied (L1 and L5: p 0.05; L2–L4: p 0.01).

Conclusion. The WM/GM ratio of the lumbar spinal cord segments is significantly correlated with the degree of spinal cord ascension: the higher the ascension, the lower the WM/GM ratio. This relationship may be explained by the fact that a longer spinal cord requires a relatively larger volume of conducting pathways to exchange the same amount of information with the brain.

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