Count: 1
Abstract ID: 148
subject: Cognition: Learning and Memory
Presentation Type: Poster

Coenzyme Q10 ameliorates anxiety and depression-like behavior associated with chronic opioid use and increases GDNF expression in the hippocampus of morphine-dependent rats

Submission Author: Somayeh Nazari

Somayeh Nazari[1]

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Background and Aim : Opioid dependence is strongly associated with moderate to severe depression and anxiety. The primary objective of this investigation was to determine whether coenzyme Q10 (CoQ10) has the capacity to increase the level of glial cell line-derived neurotrophic factor (GDNF), with the aim of ameliorating anxiety- and depression-like behaviors in morphine (MOP)-dependent rats

Methods : In this study, 40 male Wistar rats were randomly divided into five experimental groups: Oil group, MOP+Oil group, MOP+Q10-100 group, MOP+Q10-200 group, and MOP+Q10-400 group. Rats received escalating doses of MOP (25 to 100 mg/kg, s.c.) once daily. After 21 days of drug dependency, CoQ10 was administered orally at doses of 100, 200, and 400 mg/kg once daily for four weeks. Behavioral assessments were conducted using the open field test, elevated plus maze, and forced swim test

Results : GDNF expression in the hippocampus was evaluated using immunohistochemistry. Four weeks of CoQ10 treatment significantly improved anxiety- and depression-like behaviors induced by MOP administration. Furthermore, CoQ10 significantly increased GDNF expression in the hippocampus. Oral administration of CoQ10 at doses of 100, 200, and 400 mg/kg over four weeks significantly reduced depressive- and anxiety-related behaviors associated with prolonged MOP exposure

Conclusion : These behavioral improvements were accompanied by increased hippocampal GDNF expression

Keywords : coenzyme Q10, depression, anxiety, morphine, opioid-related disorders, addiction

Count: 2
Abstract ID: 559
subject: Cognition: Learning and Memory
Presentation Type: Poster

Effect of prolonged exposure to different lighting conditions on MPTP-induced Q2 motor dysfunction and cognitive performance in male rats

Submission Author: Hamideh Rahmati

Hamideh Rahmati[1], Raheleh Rafaiee[2], Seyedeh Masoumeh Seyedhosseini Tamijani[3]

1. my project
2. co-respondent
3. co- respondent

Background and Aim : Circadian rhythm disruption has been increasingly implicated in neurodegenerative diseases, including Parkinson's disease (PD). While motor impairments in PD are well-established, the impact of chronic circadian misalignment on both motor and cognitive dysfunctions remains 15 underexplored. This study investigates the effect of prolonged exposure to different lighting conditions on MPTP-induced motor and cognitive deficits in a rat model.

Methods : A total of 100 adult male Wistar rats were divided into five groups with different lightdark cycle conditions: standard 12-h light/12-h dark (12 L:12D), constant light (24 L), constant darkness (24D), 20-h light/4-h dark (20 L:4D), and 4-h light/20-h dark (20D:4 L). After 60 d of 20 exposure to these conditions, each group was further divided into Parkinsonian (MPTP-treated) and non-Parkinsonian (saline-treated) subgroups. MPTP hydrochloride (25 μg/kg) was administered intranasally to induce Parkinsonism. Behavioral assessments, including the beam balance test, open field test, Y-maze test, and shuttle box test, were conducted to evaluate motor coordination and cognitive function. Immunohistochemical analyses of α-synuclein (α-syn) accu- 25 mulation in the substantia nigra (SNc) and tyrosine hydroxylase (TH) expression in the suprachiasmatic nucleus (SCN) were performed. Data were analyzed using t test, one-way ANOVA and Tukey's post hoc test.

Results : MPTP administration significantly impaired motor function, as evidenced by increased crossing time in the beam balance test ($p < 0.001$) and reduced locomotor activity in the open field 30 test ($p < 0.01$). Prolonged exposure to different lighting conditions further exacerbated these deficits, particularly in the 24 L and 20 L:4D groups ($p < 0.0001$). Cognitive assessments revealed that MPTP-treated rats exhibited marked deficits in working memory (Y-maze, $p < 0.05$) and associative learning (shuttle box, $p < 0.01$), with the most severe impairments observed in groups subjected to extreme light-dark alterations. Immunohistochemical analyses demonstrated signifi- 35 cantly increased α-syn accumulation in the SNc ($p < 0.0001$) and decreased TH

Conclusion : Chronic photoperiod disruption exacerbates MPTP-induced motor dysfunction, cognitive impairment, and neurodegeneration. Targeting circadian regulation may offer a potential Q9 therapeutic approach for mitigating neurodegenerative progression in PD.

Keywords : Circadian; photoperiod disruption; Parkinson's disease; MPTP; motor dysfunction; cognitive impairment; α-synuclein; tyrosine hydroxylase

Count: 3
Abstract ID: 16
subject: Cognition: Learning and Memory
Presentation Type: Poster

Neuroprotective potential of alpha-pinene on cognitive impairment in 3-nitropropionic acid–induced Huntington's disease

Submission Author: Paria Hashemi

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Background and Aim : Cognitive dysfunction is a key feature of Huntingtons disease, a progressive neurodegenerative disorder. 3-nitropropionic acid (3-NP) is commonly used to induce Huntington's disease –like symptoms in animal models, particularly memory deficits, by impairing mitochondrial function. Alpha-pinene, a naturally occurring monoterpene found in essential oils, exhibit antioxidant, anti-inflammatory, and neuroprotective effects. The present study aimed to evaluate the protective effects of alpha-pinene on cognitive deficits induced by 3-NP in rats, and to investigate its influence on neurotrophic signaling pathways in the hippocampus and cortex.

Methods : Cognitive dysfunction is a key feature of Huntingtons disease, a progressive neurodegenerative disorder. 3-nitropropionic acid (3-NP) is commonly used to induce Huntington's disease –like symptoms in animal models, particularly memory deficits, by impairing mitochondrial function. Alpha-pinene, a naturally occurring monoterpene found in essential oils, exhibit antioxidant, anti-inflammatory, and neuroprotective effects. The present study aimed to evaluate the protective effects of alpha-pinene on cognitive deficits induced by 3-NP in rats, and to investigate its influence on neurotrophic signaling pathways in the hippocampus and cortex.

Results : Alpha-pinene significantly improved cognitive performance in rats treated with 3-nitropropionic acid, as evidenced by increased alternation behavior in the Y-maze and reduced escape latency in the MWM. Molecular analysis showed increased expression of BDNF, TrkA, TrkB, and CREB in the hippocampus and cortex of treated rats compared to 3-NP in a dose-dependent manner.

Conclusion : These findings suggest that alpha-pinene alleviates cognitive deficit in a rat model of Huntington's disease, possibly through enhancement of neurotrophic signaling pathways. Alpha-pinene may be considered a promising candidate for further research in neuroprotective therapies targeting cognitive deficits in neurodegenerative disorders.

Keywords : Alpha-pinene, Huntington's disease, 3-nitropropionic acid, Cognitive deficit, BDNF, CREB

Count: 4
Abstract ID: 473
subject: Cognition: Learning and Memory
Presentation Type: Oral

Bicuculline-Mediated Modulation of Neuroinflammation and Remyelination Enhances Cognitive Recovery in a Rat Model of Hippocampal Demyelination

Submission Author: Fereshteh Pourabdolhossein

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Background and Aim : Introduction: Gamma-aminobutyric acid type A (GABA A) receptors play a crucial role in hippocampal inhibitory neurotransmission, but their involvement in demyelination-induced cognitive deficits remains poorly understood. Building upon previous findings showing that bicuculline, a GABA A receptor antagonist, improves spatial memory in a lysophosphatidylcholine (LPC)-induced hippocampal demyelination model, this study investigates the underlying mechanisms with focus on neuroinflammation and remyelination.

Methods : Methods: Adult male Wistar rats (200-220 gr) were subjected to hippocampal demyelination via stereotaxic injection of lysophosphatidylcholine (LPC) (3 µL each side). Animals were randomly assigned to control, LPC, LPC+vehicle (saline), LPC+bicuculline (BM), and LPC+muscimol (MS) groups. Bicuculline (0.05 µg/2 µL per rat) and muscimol(0.1 µg/2 µL per rat) were administered intracerebroventricularly following LPC injection per day for 21 days. Behavioral assessments of cognition included the Y-maze spontaneous alternation test for short-term spatial memory and the passive avoidance task for learning and memory retention. Post-behavioral analyses involved immunofluorescent staining for myelin basic protein (MBP) and immunohistochemistry (IHC) for GFAP to assess myelin integrity and astrocyte activation, respectively. Quantitative PCR was used to measure expression levels of myelin markers (Olig2, PLP), inflammatory cytokines (NF-κB, TNF-α, IL-1β), anti-inflammatory cytokine IL-10, and iNOS.

Results : Results: Behavioral assays including Y-maze and passive avoidance tests confirmed that bicuculline restored LPC-induced memory deficits, significantly improving spontaneous alternation percentage and passive avoidance performance, while muscimol showed no effect.

Myelin integrity assessed by MBP immunofluorescence revealed severe reduction after LPC treatment, which was markedly reversed by bicuculline but not muscimol. qPCR analysis showed downregulation of myelin-associated genes Olig2 and PLP in LPC and vehicle groups, which were significantly upregulated by bicuculline administration. Inflammatory mediators NF-κB, TNF-α, IL-1β, and iNOS were elevated following LPC injection but significantly reduced by bicuculline, whereas IL-10 expression was increased exclusively by bicuculline treatment. GFAP immunostaining demonstrated pronounced astrocyte activation in LPC and vehicle groups, which was attenuated after bicuculline but unaffected by muscimol.

Conclusion : Conclusions: These findings suggest that bicuculline mitigates neuroinflammation and promotes remyelination, thereby reversing cognitive impairments in the LPC-induced hippocampal demyelination model. Thus, suggesting a promising therapeutic strategy for cognitive dysfunction associated with demyelinating diseases.

Keywords : Demyelination; Hippocampus; Inflammation; Memory deficit; Bicuculine; Mocimol

Count: 5
Abstract ID: 930
subject: Cognition: Learning and Memory
Presentation Type: Oral

The effect of agmatine on molecular and behavioral indices of male rat offspring under maternal

Submission Author: FATEMEH Mohammadi

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Background and Aim : Physical stress experienced by a mother during gestation is often associated with serious behavioural and cognitive deficits in newborns. Investigations of protective agents, which could prevent the adverse outcomes of prenatal stress (PS), are warranted. Agmatine is a neurotransmitter putatively involved in the physiological response to stress, and exogenous administration of agmatine has been shown to produce a variety of neuroprotective effects.

Methods : In this study, we aimed to assess whether agmatine exposure could ameliorate behavioural and cognitive deficits in offspring born to prenatally stressed rats. Pregnant wistar rats were exposed to physical unpredictable mild stress for 10 days from the 11th to 21th days of gestation. Agmatine (40 mg/kg, i.p.) was administrated in pups on postnatal days 14 for 14 consecutive days. The offsprings were assessed using a variety of behavioral tests and molecular assays on postnatal days 50 to 52.

Results : Agmatine attenuated impairments in anxiety-like behaviour. Furthermore, agmatine reduced cognitive deficits induced by prenatal stress. Agmatine treatment affected the level of hippocampal brain-derived neurotrophic factor (BDNF).

Conclusion : Taken together, our findings highlighted the protective effects of administered agmatine on PS-mediated behavioural and cognitive deficits of the offsprings. Future studies are needed to elucidate the underlying mechanisms, which could allow for more targeted prenatal treatments.

Keywords : Learning, Memory, Anxiety

Count: 6
Abstract ID: 420
subject: Cognition: Learning and Memory
Presentation Type: Poster

Interaction of Memantine with Homocysteine on the Apoptosis in the Rat Hippocampus cells

Submission Author: Amin Ataie

Amin Ataie[1], AMN ATAIE[2]

1. Babol university of Medical science

Background and Aim : It has been hypothesized that elevated plasma Homocysteine (Hcy) plays a role in the pathogenesis of Alzheimer's disease (AD) and age-related cognitive decline. The mechanism of Hcy neurotoxicity in the brain is controversial as well Hcy is a ligand of NMDA receptor. Memantine, an uncompetitive antagonist of N-methyl- D-aspartate (NMDA) receptors approved for the treatment of moderate to severe Alzheimer's disease. Memantine could antagonize NMDA receptors in mid brain.

Methods : Hcy was injected 0.5 µmol/µl in the hippocampus of the rat brain and Memantine hydrochloride was injected 10mg/kg intraperitoneally 1 hour prior to Hcy injection. After five days, rats were killed and whole brain were taken out, fixed, and embedded in paraffin. The slices of the rat brain were prepared and immunohistochemical analysis was done to reveal the protein expression of Bax, Bcl-2, and the activation of Caspase 3 in the rat hippocampus layers. Results showed significant increase of Bax and Caspase-3 immunoreactivity in hippocampus of rat brain in Hcy group. Also an increase in Bax/Bcl-2 ratio in rat hippocampus cells .Memantine pretreatment could not change the levels of Bax, Bcl-2, Caspase-3 significantly in rat's hippocampus cells

Results : These findings suggest that Memantine could not antagonize Hcy – induced Apoptosis. Hcy may induce apoptosis via the other oxidative stress mechanism in the rat brain. potential. It may therefore be interesting that The barberry fruit extracts has the unique capacity to quench free radicals

Conclusion : Our results suggested that Hcy may induce apoptosis and cell death in rat hippocampus. Hcy my induce oxidative stress and produce ROS that attack all biological macromolecules (e.g. proteins,DNA and lipids). It is suggested that Hcy may be a risk factor for AD and PD. Also Memantine could not antagonize Hcy neurotoxicity and not improve hippocampus cell death in our experiment.

Keywords : Homocysteine, apoptosis, memantine, alzheimer's disease, hippocampus, NMDA receptor, oxidative stress

Count: 7
Abstract ID: 964
subject: Cognition: Learning and Memory
Presentation Type: Poster

Post-Stress Forced Running Wheel Exercise in Ovariectomized Rats: Effects on Recognition Memory and BDNF in the Hippocampus and Prefrontal Cortex

Submission Author: SeyedehRose Jamali

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Background and Aim : Single prolonged stress (SPS) degrades recognition memory and suppresses neurotrophic support in the hippocampus and prefrontal cortex (PFC). Estrogen loss after ovariectomy (OVX) exacerbates these deficits and may dampen responsiveness to interventions. We investigated whether post-stress, moderate-intensity forced running wheel (FRW) exercise can rescue recognition memory and brain-derived neurotrophic factor (BDNF) signaling, and whether its efficacy depends on ovarian status

Methods : Adult female Wistar rats were studied in a 2×2×2 design: ovarian status (Sham vs. OVX) × stress (no-SPS vs. SPS) × activity (Sedentary vs. FRW). After SPS and a 14-day incubation, animals completed a 4-week FRW program (acclimation, then moderate speed, fixed duration, five sessions per week). Recognition memory was assessed with the Object Recognition test (discrimination index). BDNF was quantified in the hippocampus and PFC by ELISA. Analyses tested main effects and interactions among stress, ovarian status, and exercise.

Results : SPS and OVX each impaired recognition memory and reduced BDNF in both the hippocampus and the PFC. FRW improved memory performance and elevated BDNF across conditions. However, ovariectomy moderated these benefits: although FRW remained effective after SPS in OVX animals, the magnitude of recovery in memory and BDNF was smaller than in sham counterparts. Patterns in the PFC mirrored those in the hippocampus. The overall profile supports strong main effects of stress and ovarian status, a clear benefit of exercise, and meaningful interactions among these factors.

Conclusion : This study showed that moderate-intensity forced wheel running (FRW) exercise after experiencing severe stress can partially restore cognitive memory and increase neurotrophic factors in critical areas of the brain responsible for cognition.. This finding demonstrates that structured aerobic exercise remains an effective rehabilitation strategy even after experiencing severe stress, which can often lead to cognitive deficits. However, the presence of hypoestrogenic status, such as during menopause or due to certain medical conditions, constrains the full potential of recovery. This suggests that estrogen depletion not only diminishes the brain's overall plasticity reserve but also blunts the responsiveness to exercise interventions, thereby limiting cognitive and neurotrophic gains. Consequently, these insights highlight the importance of developing personalized, mechanism-based interventions tailored to individual hormonal status. In both premenopausal and postmenopausal settings, combining aerobic exercise with estrogenic strategies such as hormone replacement therapy may prove essential in optimizing neurotrophic tone, enhancing neural plasticity, and improving cognitive outcomes. Future research should focus on elucidating the precise mechanisms by which estrogen interacts with exercise-induced neuroplasticity and on designing integrated treatment protocols that consider hormonal status to maximize recovery and cognitive resilience

Keywords : post-stress exercise; forced running wheel; ovariectomy; single prolonged stress; recognition memory; hippocampus; prefrontal cortex; BDNF

Count: 8
Abstract ID: 3
subject: Cognition: Learning and Memory
Presentation Type: Oral

Investigation of topological changes of the links in the brain functional network during false memory formation

Submission Author: Parisa Khoorahe

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Background and Aim : This study aims to examine and compare the organization of the brain functional network during the formation of false and true memories using structural balance theory and its associated parameters.

Methods : A causal-comparative research design was employed, using data from a previous study involving 38 participants aged 18 to 30 from Beheshti University. Participants performed a false memory task based on the DRM (Deese-Roediger-McDermott) paradigm. Functional connectivity matrices corresponding to true and false memory events were extracted, and structural balance theory-informed parameters were extracted from the functional connectivity matrices before being submitted to comparative analysis.

Results : The statistical analysis of the nine parameters of structural balance showed differences in four parameters in the beta II frequency band (15–18 Hz) between false memories and true memories. The parameters are the number of strong unbalanced triads (|T2|), the number of strong balanced triads (|T3|), the degree of balance energy (Un), and the tendency to form positive hubs (TMHp).

Conclusion : The decrease in the balance energy of the functional brain network observed for false memory formation corroborates the hypothesis of this study. This change was, however, limited to the beta II frequency band only. Previous studies have also investigated the involvement of the beta band in false memory formation. Furthermore, comparisons of functional brain network parameters between patients with psychological or cognitive disorders and healthy individuals have reported greater differences across different frequency bands, suggesting that the disorders may induce more basic changes in the structure of brain networks than those corresponding to state-dependent differences in healthy individuals.

Keywords : Electroencephalography;False memory;Signed links;Balance of the Network;Structural Balance Theory;Triads Association

Count: 9
Abstract ID: 190
subject: Cognition: Learning and Memory
Presentation Type: Poster

Neuroplasticity-Based Mechanisms of fMRI-Guided rTMS and TBS in Mild Cognitive Impairment (MCI)

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Conclusion : Mild Cognitive Impairment (MCI) is a widespread disorder among the aging population, an intermediary state between normal aging and Alzheimer's disease. Early intervention is imperative, but effective pharmaceutical choices are limited. Non-invasive brain-stimulating procedures, especially repetitive transcranial magnetic stimulation (rTMS) and theta burst stimulation (TBS), with the guidance of functional MRI (fMRI), hold promise for stimulating neuroplasticity throughout large-scale networks in the brain. This narrative review set out to examine the neuroplasticity-based mechanisms and cognitive benefits of fMRI-guided rTMS and TBS in MCI. A structured literature search was conducted in Web of Science, Scopus, and PubMed for studies between 2019 and 2025, including randomised controlled trials, pilot studies, and systematic reviews/meta-analyses evaluating rTMS/TBS protocols applied to brain areas identified by fMRI connectivity analysis as involved in MCI. Selected studies revealed that high-frequency rTMS and intermittent TBS (iTBS) over the dorsolateral prefrontal cortex (DLPFC) enhanced memory, executive function, and total cognition. Neuroimaging results revealed the modification of hippocampal function and boosted DMN (Default Mode Network), CEN (Central Executive Network), and intrahippocampal connectivity. Dual-site stimulation (DLPFC-hippocampal networks) and multi-session protocols (10 sessions or more) elicited larger, longer-lasting cognitive improvements. Finally, adding cognitive training with rTMS resulted in neuroplastic effects with supra-additive effects. Weaknesses included a few subjects, heterogeneity for the parameters of the stimulations, and variability for the neuroimaging approaches. These results indicate that fMRI-guided TBS and rTMS are safe, effective, and potentially compelling non-pharmacological therapies for cognitive enhancement in MCI with effects on the network level, where neuroplasticity may be modulated, preventing the progression to dementia. More comprehensive sham-controlled studies that are large-scale and multi-centered are necessary to regulate the protocols for optimal stimulation, thereby supporting personalized treatment approaches.

Keywords : Mild Cognitive Impairment; rTMS; Theta Burst Stimulation; fMRI; Neuroplasticity; Cognitive Function

Count: 10
Abstract ID: 480
subject: Cognition: Learning and Memory
Presentation Type: Poster

MiR-532-5p ameliorates learning and memory impairment induced by polystyrene microplastics by activation of Wnt/β-catenin and PI3K/Akt pathways

Submission Author: Leila Mohammadi

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Background and Aim : Microplastics are environmental pollutants that can accumulate in the brain and induce significant neurotoxic effects. Exposure to polystyrene microplastics (PS-MPs) can impair cognitive functions such as learning and memory. The Wnt/β-catenin and PI3K/Akt signaling pathways are essential for the survival, development, and homeostasis of the nervous system. Another important function of these pathways is the maintenance of synaptic plasticity, which is essential for learning and memory. MiR-532 is a microRNA that plays a role in a variety of biological processes, particularly in neurobiology, and exhibits neuroprotective properties.

Methods : In this study, PS-MPs were orally administered to male rats over eight weeks to induce neurotoxicity. Subsequently, the intraventricular (ICV) injection of miR-532-5p was administered for the purpose of treatment. Levels of Wnt, β-catenin, PI3K and AKT in hippocampal tissue were measured using ELISA.

Results : PS-MPs were found to cause a decline in the levels of Wnt, β-catenin, PI3K and Akt. However, subsequent treatment with miR-532-5p increased their levels.

Conclusion : Overall, the results of this study indicate that PS-MPs can impair learning and memory by inactivating the PI3K/Akt and Wnt/β-catenin pathways. In contrast, restoring memory and learning appears to be possible by activating these pathways with the help of miR-532-5p.

Keywords : polystyrene microplastics, miR-532-5p, learning and memory, Wnt/β-catenin, PI3K/Akt.

Count: 11
Abstract ID: 546
subject: Cognition: Learning and Memory
Presentation Type: Oral

The effect of exercise on increasing intelligence and students cognition

Submission Author: Sima Abazari

Sima Abazari[1]

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Conclusion : Introduction: Exercise and regular exercise (such as aerobics) have a good effect on brain function and cognition. Exercise and regular exercise (such as aerobics) have a good effect on brain function and cognition. Exercise enhances intelligence, math and verbal skills. Meta-analysis studies show that aerobic exercise training increases attention, memory, processing, speed, and executive Function. Evidence suggests that aerobic-based physical activity causes structural changes in the brain. Exercise improves cognitive performance by affecting the frontal. Also During exercise, increase the brain's hippocampal blood flow and hippocampal activity. Brain volume increases significantly in both white matter and gray matter due to aerobic exercise which results in faster neurotransmission between areas of the brain that are important for cognitive control. In particular, the size of the inner hippocampus increases as a result of physical exercise, which is associated with spatial memory. Physical activity increases oxygen consumption resulting in increased blood flow to the brain and leads to brain cell reproduction in specific areas of cognitive function. Aerobics causes short and long-term changes in brain areas that are important for learning and memory and increase blood flow. In addition, aerobic exercise increases the levels of neurotransmitters, which in turn increases neuronal tissue and synaptic flexibility. Generally research has shown that physical activity has a significant impact on academic performance and increases cognition. Exercise affects intelligence and social development. Objective: The purpose of this review study is to investigate the effects of exercise on increasing intelligence and cognition. Method: For this purpose, were studied numerous articles and researches from Valid scientific sites, including PubMed, science direct, springer, wielly, sage, and were used 30 articles related to this research. Results and Conclusion: So according to the results of this study, exercise can have a good effect on increasing intelligence and cognition and attention, so this method can be used to rehabilitate students with cognitive impairment.

Keywords : Exercise, Rehabilitation, Cognition, intelligence, Students

Count: 12
Abstract ID: 606
subject: Cognition: Learning and Memory
Presentation Type: Poster

Risperidone and Sertraline Ameliorate Behavioral Deficits in a Rat Model of Sodium Valproate-Induced Autism Spectrum Disorder

Submission Author: Golnoosh Yadollahi

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Background and Aim : Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by deficits in social communication, repetitive behaviors, and restricted interests. Prenatal exposure to sodium valproate (Na-VPA) disrupts neurodevelopment, causing excitatory–inhibitory imbalance, structural brain changes, and neuroinflammation, resulting in autism-like behaviors in animal models. This study evaluated the therapeutic potential of risperidone (RIS) and sertraline (SER) in ameliorating Na-VPA-induced behavioral deficits.

Methods : Forty male Wistar rats were divided into five groups (n=8/group): Control (saline-exposed), VPA-only (prenatal Na-VPA 400 mg/kg), RIS (Na-VPA plus risperidone 1 mg/kg postnatally), SER (Na-VPA plus sertraline 10 mg/kg postnatally), and RIS+SER (Na-VPA plus combined risperidone and sertraline). Offspring treatments were administered from postnatal day (PND) 21 to 35. Behavioral assessments conducted 24 hours after the last treatment included the Social Interaction Test (SIT), Y-maze, and Elevated Plus Maze (EPM) to evaluate social behavior, repetitive behavior, and anxiety, respectively.

Results : Prenatal Na-VPA exposure induced ASD-like behaviors: impaired social interactions, increased repetitive behaviors, and heightened anxiety. RIS treatment significantly reduced repetitive behaviors and anxiety. SER administration decreased anxiety and partially improved behaviors. Combined RIS+SER treatment resulted in the most pronounced improvements across all behavioral domains, surpassing single-drug effects.

Conclusion : Prenatal Na-VPA exposure induces ASD-like behaviors in rats, which are effectively attenuated by risperidone and sertraline. Combination therapy demonstrates superior efficacy, indicating a promising approach for managing ASD-related behavioral symptoms.

Keywords : Autism Spectrum Disorder, Sodium valproate, Risperidone, sertraline, Behavioral test

Count: 13
Abstract ID: 612
subject: Cognition: Learning and Memory
Presentation Type: Poster

Evaluation of the Efficacy of Neurocognitive Games on Cognitive Performance in Children with Specific Learning Disabilities Referred to the Educational and Rehabilitation Center for Learning Problems

Submission Author: Nahid Barati

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Background and Aim : Learning disorders are neurological conditions characterized by a delay or reduction in students' performance in academic skills such as reading, writing, spelling, speaking, and arithmetic. These disorders create a significant gap between students' potential abilities and their actual performance. This study aimed to evaluate the efficacy of neurocognitive games on cognitive functions, including verbal reasoning, auditory discrimination, visual-spatial processing, and verbal working memory, in children with specific learning disabilities referred to an educational and rehabilitation center for learning problems in Arak.

Methods : This research employed a quasi-experimental design with a control group. The statistical population consisted of all second and third-grade elementary school students referred to learning disorder centers in Arak during the 2021–2022 academic year. Based on Cohen's formula and accounting for potential attrition, 30 participants were selected and randomly assigned to either an experimental group (n = 15) or a control group (n = 15). Research instruments included the Wechsler Intelligence Scale for Children—Fourth Edition (to assess verbal reasoning; Wechsler, 1949), the Wepman Auditory Discrimination Test (Wepman, 1987), the Stanford–Binet Test (for visual-spatial processing; Gale H. Roid, 2003), and a researcher-designed neurocognitive games protocol.

Results : The mean age of participants was 8.23 ± 1.46 years, and each group consisted of five girls and ten boys. The results showed that neurocognitive games significantly improved the total score of cognitive functions, as well as verbal reasoning, auditory discrimination, visual-spatial processing, and verbal working memory, in the intervention group (80.40 ± 18.07, 18.40 ± 3.54, 17.06 ± 4.28, 20.20 ± 5.72, and 59.33 ± 20.63, respectively), compared to the control group (56.13 ± 13.64, 13.46 ± 2.35, 11.33 ± 3.28, 14.73 ± 4.07, and 47.73 ± 11.08, respectively), among children with specific learning disabilities.

Conclusion : The findings indicate that children who participated in the neurocognitive games program demonstrated significantly higher post-intervention scores across all cognitive domains compared to the control group. This suggests that structured cognitive play effectively

enhances core neuropsychological processes underlying academic performance. Overall, the results highlight the potential of neurocognitive games as an evidence-based adjunct in educational and therapeutic interventions, contributing to improved cognitive and academic outcomes among children with specific learning disabilities.

Keywords : Neuropsychological games; special learning disorder; verbal reasoning; auditory discrimination; visual spatial processing; verbal working memory.

Count: 14
Abstract ID: 730
subject: Cognition: Learning and Memory
Presentation Type: Oral

Consuming high-fat, high-carbohydrate diet from pre-pregnancy to the end of lactation selectively impaired short-term recognition memory in lactating rats

Submission Author: Fariba Azarkish

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Background and Aim : The metabolic and cognitive functions of mothers during and after pregnancy are strongly affected by their diet. High-fat, high-carbohydrate (HFHC) diets can induce systemic oxidative stress and affect hippocampal-dependent memory. This study aimed to investigate the impact of HFHC diet from pre-pregnancy until the end of lactation on metabolic parameters, oxidative stress, locomotion, and recognition memory in nursing rats.

Methods : Female rats were fed either a standard diet (control) or HFHC diet starting from two weeks before mating and continuing through gestation and lactation. At the end of lactation, dams underwent behavioral assessments and then were fasted for approximately 12 hours, anesthetized with isoflurane, and their trunk blood were collected following decapitation. Plasma was separated for determining the triglycerides (TG) and total cholesterol (TC) levels and oxidative stress markers (Malondialdehyde [MDA], catalase [CAT], glutathione [GSH]). Oxidative stress was also evaluated in hippocampus and prefrontal cortex (PFC). Behavioral assessments included Open Field test and Novel Object Recognition (NOR) for short-term and long-term memory. During NOR familiarization, animals were exposed to two identical objects. Short-term memory was assessed 2 hours later by replacing one familiar object with a

novel one, and long-term memory was assessed 24 hours later by introducing a different novel object while retaining the familiar one. Recognition memory was assessed using the discrimination index (DI), recognition index (RI), and the ratio of time spent exploring the novel versus familiar object (Novel/Familiar Ratio). Statistical analyses were performed using GraphPad Prism version 8.0. An unpaired t-test was applied to compare the two groups.

Results : HFHC-fed rats exhibited elevated plasma TG and TC levels. Plasma oxidative stress was pronounced, with marked increases in MDA level and decreases in CAT activity and GSH level. In the hippocampus, MDA content showed a moderate, non-significant increase ($p \approx 0.08$), while CAT activity and GSH content remained unchanged. No significant differences were found in oxidative stress markers within the PFC. HFHC-fed rats demonstrated greater locomotor activity, with increased distance moved and velocity in the Open Field test. Also, they showed significantly impaired short-term recognition memory in the NOR test, with decreases in DI, RI and Novel/Familiar Ratio. In contrast, long-term memory performance did not differ significantly between groups, however according to DI and RI results both groups showed weak long-term memory retention.

Conclusion : Maternal HFHC diet from pre-pregnancy to the end of lactation induces systemic oxidative stress and metabolic disturbances, leading to hyperactivity and selective impairment of short-term recognition memory in nursing rats. Moderate reductions in long-term memory indices observed in both groups may reflect the influence of maternal hormonal changes and/or behavioral test constraints. Overall, these results highlight the critical impact of maternal diet on cognitive function, locomotion, and oxidative balance during lactation, emphasizing the importance of the dams dietary interventions.

Keywords : High-fat high-carbohydrate diet; maternal brain; oxidative stress; recognition memory; locomotion

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Presentation Type: Poster

Effects of Probiotic and High-intensity Interval Training on Cognitive Function in Male Rats Consuming a High-Fat Diet

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Background and Aim : High fat diets are linked to impaired cognitive function. Several studies reported that high-fat diets are associated with metabolic disorders that can negatively impact brain health through mechanisms like insulin resistance, oxidative stress, and inflammation in the brain. We investigated whether a combined regimen of Lactobacillus rhamnosus and High-Intensity Interval Training (HIIT) could counteract the adverse effects of a High-Fat Diet (HFD) in rats. The measured outcomes included cognitive performance, systemic metabolic parameters, and oxidative stress.

Methods : Forty rats were randomly assigned to four groups (n=10 per group): a High-Fat Diet (HFD) control group, HFD + probiotic Lactobacillus rhamnosus GG, HFD + High-Intensity Interval Training (HIIT), and a combination HFD + L. rhamnosus GG + HIIT group. All rats consumed HFD daily for 16 weeks. During this period, treatments (probiotic: 1×10 10 CFU orally, and HIIT protocol) were administered four times weekly on alternating days. At the end of the 16-week intervention, memory function using the Y-maze task, and oxidative stress markers and lipid profiles using standard assay kits were measured.

Results : Both Lactobacillus rhamnosus and HIIT alone or in combination improved memory dysfunction in HFD-fed rats. In addition, both Lactobacillus rhamnosus and HIIT alone or in combination increased superoxide dismutase, and catalase levels, and decreased the serum levels of triglyceride, cholesterol, low-density lipoprotein (LDL), and increased serum high-density lipoprotein (HDL) in HFD-fed rats.

Conclusion : The combination of the probiotic Lactobacillus rhamnosus and HIIT improved memory dysfunction, oxidative stress, and metabolic parameters in the HFD-fed rats.

Keywords : Probiotic, exercise, memory, high fat diet

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subject: Cognition: Learning and Memory
Presentation Type: Poster

Effects of Fingolimod on Scopolamine-induced Memory Impairment and Neuroinflammation in rats

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Background and Aim : Dementia is a syndrome with progressive impairments on cognition and behavior beyond the normal course of aging that affects a significant number of people around the world, and its prevalence is projected to reach 153 million people by 2050. Current therapies for dementia have limitations. Therefore, it is need of today to evaluate an effective drug to treat dementia. Fingolimod (FTY720), a drug commonly used to treat multiple sclerosis, has shown promising efficacy against various neurological disorders. Fingolimod exerts its neuroprotective effects by targeting multiple cellular and molecular processes, such as apoptosis, oxidative stress and neuroinflammation. Scopolamine, a muscarinic receptor antagonist, cause impairment in memory and provide a dementia model. This study aimed to assess the effects of fingolimod on neurobehavioral and biochemical parameters in scopolamine-induced amnesic rats.

Methods : 30 male Wistar rats were randomly assigned to 3 groups (n=10 for each group): Scopolamine group (Rats received scopolamine (1mg/kg. intraperitoneal injection, for 14 days)), scopolamine+ fingolimod group (Rats received scopolamine (1mg/kg, intraperitoneal injection, for 14 days) and fingolimod (1 mg/kg, orally, from the seventh day to the fourteenth day), and control group (Rats received normal saline, for 14 days). After 14 days, Using Barnes maze and shuttle box tests learning and memory were evaluated. After completing the behavioral assessments, the rats were decapitated, and their brains were carefully removed from the skull. Then, the hippocampus was quickly separated from the brain for biochemical assessments. In addition, Interleukin (IL)-6 and tumour necrosis factor alpha (TNF-α) by ELISA kits, Iba-1 expression (activated microglia marker) by immunohistochemistry, and acetylcholinesterase (AChE) by western blot were evaluated.

Results : Scopolamine significantly impaired memory function in rats compared to control group ($p<0.05$). It also increased IL-6 and TNF-α levels ($p< 0.05$), Iba-1 protein expression ($p< 0.05$), and AChE protein expression ($p< 0.05$) in hippocampus. The data showed that fingolimod significantly improved scopolamine-induced learning and memory impairments in Barnes maze and shuttle box tests. In addition, it decreased IL-6 and TNF-α levels ($p< 0.05$), Iba-1 protein expression ($p< 0.05$), and AChE protein expression ($p< 0.05$) in hippocampus.

Conclusion : The present study highlights the neuroprotective effects of fingolimod, noting that it improves learning and memory impairments, and decreased IL-6, TNF-α, AChE and microglia activity in the hippocampus.

Keywords : Fingolimod, memory, neuroinflammation

Count: 17
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subject: Cognition: Learning and Memory
Presentation Type: Poster

Neurocognitive Effects of Postbiotics from Native Lactobacillus and Bifidobacterium Strains in a Mouse Model of Chronic Stress

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Background and Aim : Postbiotics, comprising bioactive metabolites like short-chain fatty acids and cell wall components, offer enhanced stability and safety compared to live probiotics, making them a promising therapeutic tool for cognitive enhancement. These compounds may improve memory by modulating neuroplasticity and reducing neuroinflammation via the gut-brain axis. The gut-brain axis plays a pivotal role in modulating cognitive function, particularly under chronic stress conditions. This study evaluates the effects of postbiotics derived from Bifidobacterium longum and Lactobacillus rhamnosus strains on learning and memory in male BALB/c mice subjected to chronic variable stress (CVS).
Methods : In a randomized controlled trial, mice were assigned to two major groups: naïve and CVS which each has four sub-groups (control, Bifidobacterium longum (Bif), Lactobacillus rhamnosus (Lac) and Mixed treatment), receiving oral postbiotics (109 CFU/mL supernatant containing bacterial debris) or placebo for 8 weeks. Cognitive performance was assessed using the Morris Water Maze (MWM) for spatial memory, Shuttle Box for passive avoidance memory, and Novel Object Recognition (NOR) test for recognition memory. Hippocampal expression of learning- and memory-related genes (AKT, Arc) was quantified via qRT-PCR.
Results : CVS impaired memory performance across all behavioral tests. However, treatment with postbiotics specially those derived from Bifidobacterium longum significantly enhanced cognitive performance across. In the MWM, postbiotic-treated mice showed reduced escape latency and mean distance to platform compared to CVS group. The NOR test revealed an improved discrimination index, and the Shuttle Box test indicated increased latency to enter the dark compartment, reflecting enhanced recognition and inhibitory memory performance, respectively. qRT-PCR analysis demonstrated upregulated AKT and Arc expression in postbiotic groups, suggesting enhanced synaptic strengthening and memory-related signaling molecules.
Conclusion : These findings indicate that postbiotics preserve learning and memory in chronic stressed condition. This study underscores the therapeutic potential of postbiotics for cognitive impairments associated with chronic stress and neurodegenerative disorders, emphasizing the need for further investigation into their mechanisms and clinical applications.
Keywords : postbiotics ; chronic stress ; gut-brain axis ; hypothalamus

Count: 18
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subject: Cognition: Learning and Memory
Presentation Type: Poster

Quantifying Visual Working Memory Deficits in Multiple Sclerosis via Python-Based Behavioral Modeling

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Background and Aim : Cognitive dysfunction is one of the most frequent symptoms in multiple sclerosis (MS), often affecting processing speed and working memory. However, while verbal and executive memory have been extensively studied, visual working memory (VWM) deficits remain poorly characterized. Traditional tasks usually measure accuracy in a binary way (correct/incorrect) and fail to capture subtle distortions in memory precision. To address this, we developed a Python-based continuous-report paradigm and probabilistic modeling approach to quantify distinct sources of VWM errors in relapsing-remitting MS (RRMS).

Methods : Thirty-nine RRMS patients and thirty-nine age- and sex-matched healthy controls (18–55 years) completed a color recall task implemented in PsychoPy. Each participant performed 60 randomized trials in which two colored squares were briefly shown and, after a short delay, one was recalled from a circular color wheel. Error angles (in degrees) were analyzed using probabilistic mixture modeling to decompose (1) precision error — variability around the target color, (2) misreport error — reporting the distractor color instead of the target, and (3) random guessing — uniform responses unrelated to either stimulus. All patients were diagnosed according to the 2017 McDonald criteria at Bouali Hospital, Qazvin, Iran.

Results : MS patients showed significantly greater mean absolute error (35.61° ± 15.13) than controls (25.93° ± 6.26; $t(76)=5.53$, $p<0.001$). Mixture modeling revealed higher distractor misreporting ($p=0.001$), greater guessing probability ($p=0.01$), and reduced precision ($p=0.001$) in the MS group. The simple model provided a better fit (lower AIC/BIC) than the full model. Participants with diplopia as their first symptom exhibited lateralized performance deficits despite normal visual acuity.

Conclusion : These results suggest that MS patients not only show reduced memory precision but also increased feature-binding errors, indicating potential disruption in attentional control and integration processes within visual working memory networks. Such computational modeling provides a sensitive framework for detecting subtle cognitive impairments beyond conventional neuropsychological measures.

Keywords : Multiple Sclerosis, Visual Working Memory, Cognitive Impairment, Error Analysis, Probabilistic Modeling

Count: 19
Abstract ID: 924
subject: Cognition: Learning and Memory
Presentation Type: Oral

The Relationship Between Flexibility and Inference in Reward Learning

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Background and Aim : Our interactions with objects are often guided by memories of their past rewards. Our recent study has shown that reward learning relies on two complementary systems: a fast, flexible system that quickly adapts to new information, and a slow, stable system that integrates experience over time. There is another dichotomy in reinforcement learning mechanisms that has to do with the degree of inference: model-free mechanisms store values from direct experience, whereas model-based mechanisms compute values via cognitive maps. In this study, we have adapted our previous design used to establish the presence of a multi-rate processes to explore how fast and slow systems may differ across the inference axis.

Methods : we trained macaque monkeys to associate visual objects with direct rewards (first-order objects) and with those predictive of them (second-order objects). After reversing the values of first-order objects, we examined how the flexible and stable systems updated their representations of the second-order objects. If a process is model-free, its second-order value should remain unchanged by the reversal of the first-order rewards.

Results : Behavioral data and computational modeling revealed that the flexible system exhibited model-based updating, while the stable system behaved in a model-free manner. Neural recordings in prefrontal cortex further supported this distinction: early neural responses reflected stable, model-free value signals, whereas later responses reflected flexible, model-based adjustments.

Conclusion : Together, these findings suggest that the stable, model-free system drives initial orienting responses to objects, providing a rapid but inflexible guide to behavior. The flexible, model-based system refines this response later in time, integrating contextual information to support adaptive decision-making. This dual-process framework highlights a temporal and computational division of labor in the brain’s reward learning mechanisms.

Keywords : Reward; Model-Based; Model-Free; Fast system; Slow system

Count: 20
Abstract ID: 933
subject: Cognition: Learning and Memory
Presentation Type: Poster

ER Stress–Mediated Disruption of Inflammatory, MAPK, and Insulin Signaling Pathways Following Thapsigargin Exposure in the Brain

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Background and Aim : The accumulation of misfolded or unfolded proteins within the endoplasmic reticulum (ER) initiates a cellular stress response known as ER stress, a process increasingly implicated in the pathogenesis of Alzheimer's disease (AD). Impairments in central insulin signaling have also been linked to AD-related cognitive deficits. Despite these connections, the interplay between ER stress, neuroinflammation, MAPK pathway activation, and hippocampal insulin resistance remains poorly understood. This study aimed to explore the mechanistic relationship between brain ER stress and memory impairment and to establish a reliable animal model to study ER stress–mediated cognitive dysfunction.

Methods : To induce central ER stress, thapsigargin (TG), a well-known inhibitor of the sarco/endoplasmic reticulum Ca^{2+}-ATPase, was administered intracerebrally to rats. Spatial learning and memory performance were evaluated using the Morris Water Maze (MWM) test. To confirm the induction of ER stress, the hippocampal mRNA levels of unfolded protein response (UPR) markers were quantified. Western blot analysis was then used to assess the protein expression of ER stress markers (BiP, CHOP), apoptotic regulators (Bax, Bcl-2, caspase-3), inflammatory cytokines (TNF-α), and signaling molecules associated with MAPK and insulin pathways.

Results : Behavioral assessments revealed that TG administration significantly impaired spatial learning and memory performance. Molecular analyses demonstrated a pronounced upregulation of hippocampal UPR markers, along with increased levels of pro-apoptotic proteins (CHOP, Bax, caspase-3) and inflammatory mediators (TNF-α). Furthermore, TG treatment enhanced p38 MAPK activation and disrupted insulin signaling in the hippocampus, indicating an interconnection between ER stress and metabolic dysfunction.

Conclusion : The present findings indicate that thapsigargin-induced ER stress leads to hippocampal dysfunction, inflammation, apoptosis, and insulin signaling impairment, culminating in cognitive deficits. These results establish TG-induced ER stress as a pharmacological model for studying memory impairment and provide mechanistic insights into the contribution of ER stress to neurodegenerative and metabolic brain disorders.

Keywords : Alzheimer's disease; endoplasmic reticulum stress; apoptosis; inflammation; MAPK; insulin resistance; spatial memory impairment

Count: 21
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subject: Cognition: Learning and Memory
Presentation Type: Poster

Pancreatic Tumors Hijack Genes Involved in Learning, Memory, and Hope: A GEO-Based Transcriptomic Analysis

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Background and Aim : Subject: Neurogenetics, Learning, Memory, Psychological Resilience, and Neural Plasticity in Pancreatic Cancer Objective: Pancreatic cancer is one of the most aggressive and lethal malignancies, in which unknown molecular pathways contribute to growth and survival. This study aimed to investigate the expression patterns of genes associated with learning, memory, and hope in tumor and normal pancreatic tissues, to evaluate the tumor's ability to utilize these genes for its own adaptability and survival.

Methods : Transcriptomic data were obtained from GEO datasets, including GSE15471, GSE28735, GSE60979, GSE62165, and GSE62452. Genes involved in both hope/resilience and learning/memory were selected. Differential gene expression analysis between tumor and normal tissues was performed using the GEO2R tool and R statistical software.

Results : The results revealed that genes related to psychological resilience, such as OXTR, BDNF, and NR3C1, exhibited stable and significant expression patterns across most datasets, whereas genes involved in learning and memory, such as ARC, FMR1, NRG1, and EGR1, showed variable, context-dependent alterations. Up regulation of OXTR, BDNF, ERBB4, NRG1, and ARC indicated activation of adaptive and survival-related pathways in tumor cells, while down regulation of SLC6A4 and NR3C1 may suggest disruption of stress regulation and loss of normal hormonal feedback within the tumor microenvironment. Integrated data analysis suggested that tumor cells exploit neuroadaptive molecular programs associated with neural resilience to promote their own growth and resistance.

Conclusion : Genes associated with emotional resilience exhibit stable expression patterns and play a more direct role in the biology of pancreatic tumors; while learning and memory-related genes show variable, environment-dependent effects. These findings suggest that cancer cells hijack neurobiological programs originally designed for learning and hope to enhance their survival and adaptability. The results may provide a basis for identifying new biomarkers and therapeutic targets in pancreatic cancer.

Keywords : Pancreatic cancer, Neurocognitive genes, Memory, Learning, Hope, Gene expression, GEO, Psychological resilience

Count: 22
Abstract ID: 305
subject: Cognition: Working Memory
Presentation Type: Poster

Investigating the Face Working Memory and Perception in Patients with Specific Learning Disorders

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Background and Aim : Working memory impairments represent a core cognitive limitation in adolescents with specific learning disorders (SLD), yet little is known about how emotional valence modulates face working memory (FWM) in this population.

Methods : The present study employed a morph-based delayed-reproduction paradigm to compare recall precision, directional error bias, and reaction times between 30 adolescents with SLD and 29 typically developing (TD) peers. Emotional faces (sad, neutral, happy) were used to examine the interaction between executive and affective processes.

Results : Results revealed that the SLD group exhibited significantly larger recall errors and slower responses across all valence conditions. Both happy and sad faces produced systematic drift toward neutrality, indicating “valence flattening,” while subgroup analyses showed that dyslexia was associated with the most severe bias, particularly for neutral faces. Emotion classification accuracy was consistently lower in SLD participants, with the greatest deficits for sad and neutral expressions.

Conclusion : These findings suggest that emotional load exacerbates working memory inefficiency in SLD, disrupting executive–affective filtering and diminishing perceptual precision. Emotion-modulated FWM error metrics may therefore provide sensitive diagnostic markers and inform targeted interventions aimed at strengthening cognitive–emotional regulation in learning-disabled adolescents.

Keywords : Specific Learning Disorders. Working Memory, Perception, Emotion, Face

Count: 23
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subject: Cognition: Working Memory
Presentation Type: Poster

Cogniran: A School-Based Conceptual Model for Working Memory Rehabilitation in Iran

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Background and Aim : Working memory (WM), a core component of executive functions, enables the temporary storage and manipulation of information essential for reasoning, learning, and self-regulation. According to Baddeley's multicomponent model (2012), WM supports higher-order cognition through domain-specific buffers coordinated by a central executive. Recent evidence emphasizes its strong association with academic outcomes, particularly in mathematics, reading comprehension, and science learning (Peng & Kievit, 2020; Giofrè et al., 2021). Deficits in WM are linked to poor educational performance, reduced attentional control, and emotional difficulties. Neuroscientific studies show that WM training engages the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), parietal cortex, and hippocampus, strengthening both cognitive and academic functions (Baniqued et al., 2022; Kassai et al., 2023). Despite international efforts such as Cogmed or Lumosity, evidence indicates their limited ecological validity and lack of cultural adaptation (Sala & Gobet, 2020; Holmes et al., 2021). In Iran, no indigenous, school-based WM rehabilitation model currently exists.

Methods : This study employed a conceptual design approach in four stages: 1. Literature review (2020–2023): Synthesizing evidence on WM, executive functions, and digital training. 2. Neurocognitive target identification: Selection of brain regions most involved in WM—DLPFC, ACC, parietal cortex, hippocampus. 3. Contextual analysis: Identifying the absence of culturally adapted WM tools in Iran. 4. Model development (Cogniran): A three-layer framework with: o Inputs: Curriculum-based digital tasks in math, physics, chemistry, biology. o Processing: Training of updating, inhibition, sequencing. o Outputs: Cognitive, academic, and emotional improvements. A defining principle is adaptivity: tasks scale in complexity as students' progress, ensuring sustained engagement and preventing task automatization.

Results : Cogniran integrates curriculum-embedded gamified exercises with adaptive difficulty. • Input Layer: Academic tasks designed to enhance ecological validity. • Processing Layer: Explicit engagement of updating, inhibition, sequencing, mapped to DLPFC, ACC, parietal cortex, hippocampus. Adaptive progression fosters experience-dependent plasticity and strengthens executive processes. • Output Layer: Anticipated outcomes include increased WM span, improved problem-solving and comprehension, reduced test anxiety, and long-term transfer to daily functions. Prototype scenarios illustrate the framework. Math Quest progressively challenges updating and inhibition with multi-step equations and distractors.

Chemistry Lab Memory enhances sequencing and hippocampal-prefrontal coordination by recalling and ordering experimental steps under interference. These examples highlight the adaptive and domain-integrated nature of Cogniran.

Conclusion : Cogniran represents a neuroscience-informed, adaptive, and culturally contextualized model for WM rehabilitation in schools. Evidence supports the effectiveness of adaptive training (von Bastian & Oberauer, 2020), curriculum integration (Holmes et al., 2021; Katz et al., 2022), and neuroplastic changes in prefrontal and parietal networks (Fellman et al., 2020; Soveri et al., 2021). By embedding WM training within school curricula, Cogniran addresses a critical gap in Iran and provides a scalable, evidence-based pathway for enhancing cognitive capacity, academic achievement, and emotional resilience. Future studies should empirically test this framework in Iranian schools to confirm its educational and neurocognitive impact.

Keywords : Working Memory; Executive Functions; Cognitive Rehabilitation; School-Based Intervention; Educational Neuroscience; Cogniran

Count: 24
Abstract ID: 1030
subject: Cognition: Working Memory
Presentation Type: Poster

Investigating the reciprocal effects of crocin and morphine on state-dependent memory in male mice

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Background and Aim : State-dependent memory (SDM) occurs when a subject forms memories under specific conditions, such as pain, emotions, or drug influence, and those memories are best recalled when the same mental or drug conditions are recreated. Evidence suggests that morphine plays a role in state-dependent memory. Additionally, studies indicate that crocin enhances memory. However, the interaction between crocin and morphine in improving SDM has not been fully explored.

Methods : A total of 180 male NMRI mice were used in this study. The rats were anesthetized and cannulated in the left ventricle using a stereotaxic apparatus during brain surgery. After recovery, drugs were administered peritoneally, and tests for state-dependent memory in step down apparatus.

Results : Showed that crocin administration at a dose of 10 mg/kg exhibited an enhanced SDM in the rats. Intraventricular injection of morphine (10 µg/mouse) on the day of training or testing did not significantly impaired SDM. However, administering the same dose on both the training and testing days increased SDM. Simultaneous intraperitoneal injection of crocin (5 mg/kg) with intraventricular naloxone (2 µg/mouse) and morphine (5-10 µg/mouse) on both training and testing days demonstrated that naloxone nullified the SDM enhancement induced by crocin and morphine. Furthermore, intraperitoneal crocin injections (1-5-10 mg/kg) combined with naloxone confirmed that naloxone negates crocin's memory-enhancing effects. Hematoxylin-eosin staining revealed that the injections of crocin, morphine, and naloxone affected cell appearance and the number of dead cells in the prefrontal cortex and CA1 region. Morphine increased the number of dead cells in these regions, whereas naloxone and crocin significantly reduced cell death compared to the morphine group.

Conclusion : The conclusion suggest that morphine, crocin, and naloxone individually influence SDM in male mice. Furthermore, the simultaneous administration of morphine and crocin showed an interactive effect on SDM processes. The present study highlights the synergistic effect of crocin and morphine in enhancing SDM in male mice.

Keywords : (State-dependent memory; Morphine; Crocin; Naloxone; Step down; Mice)

Count: 25
Abstract ID: 194
subject: Cognition: Working Memory
Presentation Type: Poster

The Effectiveness of Emotional Working Memory Training on Smartphone Addiction: A Systematic Review

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Conclusion : This systematic review, conducted under the PRISMA 2020 guidelines, evaluates the effectiveness of emotional working memory training (EWMT) as a cognitive intervention for addressing smartphone addiction, also known as problematic smartphone use (PSU). Affecting approximately 20-30% of adolescents and young adults globally, PSU is characterized by compulsive smartphone use that disrupts daily functioning, leading to psychological issues such as anxiety, depression, and impaired cognitive capacities like attention and working memory. EWMT, utilizing tasks such as affective dual n-back to enhance the processing of emotional stimuli in working memory, aims to improve emotional regulation and executive functions, potentially reducing the impulsivity and emotional dependence associated with PSU. The review synthesizes evidence from 12 peer-reviewed studies, including randomized controlled trials, reviews, and observational studies, identified through comprehensive searches of databases like PubMed, Scopus, and Google Scholar from 2015 to 2025. Focusing on participants aged 12-40, the studies primarily explore EWMT's impact on problematic internet use (PIU), a closely related construct to PSU, alongside outcomes like inhibition, attention, and cognitive emotion regulation. Key findings indicate that EWMT, typically delivered over 20 sessions, significantly reduces addiction symptoms (e.g., 40-60% decrease in Internet Addiction Test scores, $p<0.001$) and enhances cognitive control (effect sizes $\eta^2>0.80$). While direct PSU studies are limited, the overlap with PIU suggests EWMT's potential to mitigate compulsive smartphone use by strengthening prefrontal cortex activity and reducing maladaptive behaviors. The review highlights EWMT's promise as a non-invasive, scalable intervention for PSU, particularly for youth, but notes gaps in direct evidence and long-term data. It recommends integrating EWMT with behavioral therapies like mindfulness or cognitive-behavioral therapy and calls for further randomized controlled trials to confirm efficacy across diverse populations. This work contributes to psychology and cognitive science by offering insights for clinicians, educators, and policymakers tackling the rising challenge of smartphone addiction in the digital age.

Keywords : Emotional Working Memory Training; Smartphone Addiction; Emotional Regulation

Count: 26
Abstract ID: 119
subject: Cognition: Working Memory
Presentation Type: Poster

Pupil linked modulation of Fronto-Temporal Beta synchrony During Working Memory Maintenance in a Delayed Match-To-Sample Task

Submission Author: Eghlima Razeghian

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Background and Aim : Pupil diameter, aside from its basic role in light reflex, is increasingly recognized as a sensitive marker of neuromodulatory activity, particularly reflecting arousal, cognitive load, and attentional allocation through the locus coeruleus–norepinephrine (LC-NE) system. In parallel, beta-band synchrony between the frontal eye fields (FEF) and inferotemporal cortex (IT) has been identified as a neural signature of successful working memory maintenance. Moreover, theta–beta phase-amplitude coupling (PAC) is thought to facilitate hierarchical information integration. Despite these insights, it remains unclear how fluctuations in internal neuromodulatory states, as indexed by tonic pupil diameter, influence beta synchrony and cross-frequency coupling during working memory.
Methods : We investigated this relationship in non-human primates performing a delayed match-to-sample (DMS) task. Simultaneous local field potential (LFP) recordings were obtained from FEF and IT, alongside continuous pupilometry to track tonic pupil diameter throughout the delay period. Beta-band phase-locking value (PLV) and theta–beta PAC were computed within and between regions. Linear mixed-effects models were employed to assess both linear and nonlinear (inverted-U) associations between pupil-linked states and neural synchrony.
Results : Preliminary analyses suggest that tonic pupil diameter during the delay period systematically modulates synchrony measures. Specifically, moderate-to-high pupil diameters were associated with stronger beta PLV between FEF and IT, as well as enhanced theta–beta PAC. In contrast, extreme pupil sizes—either very small or very large—were linked to reduced synchrony, consistent with an inverted-U relationship between neuromodulatory state and network coordination.
Conclusion : These findings demonstrate that pupil-linked internal states play a critical role in shaping large-scale neural coordination during visual working memory. By showing that optimal synchrony emerges at intermediate levels of arousal and cognitive engagement, this work highlights the mechanistic bridge between LC-NE neuromodulation, fronto-temporal beta synchrony, and memory maintenance.
Keywords : Beta synchrony; Working memory; Locus coeruleus–norepinephrine (LC-NE) system; Neuromodulation; Phase-locking value (PLV); Theta–beta phase-amplitude coupling (PAC)

Count: 27
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subject: Cognition: Working Memory
Presentation Type: Poster

The Role of Eye Movements and Replay Strategies in Spatial Working Memory

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Background and Aim : Spatial working memory enables the temporary storage and manipulation of spatial information, which is essential for everyday tasks such as navigation, object tracking, and visually guided decision-making. Eye movements have been shown to play a key role in memory encoding and retrieval, suggesting that visual exploration supports accurate reconstruction of spatial layouts. In addition, memory replay—the mental retracing of previously observed sequences—has been found to improve recall, whether forward or backward. However, it remains unclear how the availability of eye movements interacts with replay strategies and how task difficulty influences the reliance on these strategies. This study investigates the effects of eye movements and memory replay on performance in a visual spatial memory task, examining both accuracy and cognitive load under varying conditions.

Methods : Thity healthy adults with normal or corrected vision participated in the study. Participants were required to memorize the locations of yellow target cells in a hexagonal grid and subsequently select them from memory, with target numbers ranging from four to thirteen and arranged in either dense or distributed patterns. The study included two conditions: in the “With Saccades” (WS) condition, participants could freely move their eyes during encoding, while in the “Without Saccades” (WOS) condition, participants were required to maintain central fixation, preventing voluntary eye movements. Performance was measured in terms of accuracy and completion time, and eye-tracking metrics such as saccade patterns and pupil diameter were recorded to assess cognitive load. Replay strategies were identified by comparing the sequence of eye movements during encoding with the order of selections during recall, classifying trials as forward, backward, or no replay. Generalized Linear Mixed Models (GLMM) and multinomial logistic regression were used to analyze the effects of spatial features, eye movement condition, and replay strategy on task performance.

Results : The results showed that allowing eye movements significantly improved accuracy and reduced task completion time, whereas restricting saccades led to higher error rates and increased cognitive effort. Accuracy declined as the number of targets increased, and spatial features such as target clustering and distance from the center affected performance, particularly under the WOS condition. Selection sequences aligned with prior encoding saccades enhanced recall, while later-selected targets were more likely to be missed, indicating memory decay. Replay strategies, whether forward or backward, improved performance compared to trials without replay, although there was no difference between forward and backward replay. As task difficulty increased, participants were less likely to employ replay strategies, suggesting that higher cognitive load or complexity reduced reliance on memory-based sequencing.

Conclusion : This study demonstrates that both eye movements and memory replay strategies are crucial for spatial working memory. Eye movements facilitate accurate and efficient recall by allowing active visual exploration, while replay strategies enhance performance by mentally retracing previously encoded sequences. Task difficulty influences both overall performance and the likelihood of employing replay strategies, with higher difficulty reducing the use of memory-based sequences. These findings highlight the interaction between visual exploration and memory processes and have important implications for designing adaptive cognitive systems that support spatial memory under varying task demands.

Keywords : Spatial working memory;Visual memor;Eye movement;Task difficulty;Adaptive games

Count: 28
Abstract ID: 19
subject: Cognition: Working Memory
Presentation Type: Oral

"Validation and Reliability of the Persian Abbreviated Word Auditory Recognition and Recall Measure (WARRM) for Auditory Working Memory Assessment"

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Background and Aim : To develop and validate the Persian abbreviated version of the Word Auditory Recognition and Recall Measure (WARRM) for use with normal-hearing Persian speakers.

Methods : Fifty-nine monolingual Persian-speaking young adults (18–25 years) with normal hearing participated. Experts selected Persian words considering phonetic and semantic factors to ensure lexical appropriateness. The test's face and content validity were evaluated by a panel of 10 specialists. Participants completed the Persian WARRM and standardized forward and backward digit span tests to assess convergent validity. Test-retest reliability was examined over a two-week interval in 30 participants using Intraclass Correlation Coefficients (ICC).

Results : The Persian WARRM showed strong face and content validity. Internal consistency was acceptable overall ($\alpha = 0.72$), highest in the recall subtest ($\alpha = 0.87$). Test-retest reliability was excellent for the total score (ICC = 0.88) and recall (ICC = 0.89), moderate for recognition (ICC = 0.48). Significant positive correlations with digit span tasks (forward: $r=0.65$; backward: $r=0.43$; $p < 0.001$) confirmed its construct validity.

Conclusion : The Persian abbreviated WARRM is a reliable and valid auditory working memory measure. It provides a valuable tool for neuroscience research and clinical assessment of Persian-speaking populations, facilitating investigations into cognitive mechanisms of speech perception.

Keywords : Auditory working memory; Speech perception; Word Auditory Recognition and Recall Measure (WARRM);

Count: 29
Abstract ID: 327
subject: Cognition: Working Memory
Presentation Type: Poster

Auditory Working Memory Performance in Patients with Specific Learning Disorders

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Background and Aim : Specific Learning Disorders (SLD) are frequently linked to deficits in executive functions, particularly working memory and metacognitive regulation. Although impairments in visual and verbal working memory are well documented, auditory working memory (AWM)—a key mechanism for phonological decoding, language comprehension, and verbal reasoning—remains insufficiently explored in this population.

Methods : This study examined AWM performance and confidence monitoring in adolescents with dyslexia, dyscalculia, and dysgraphia compared to typically developing peers. Participants performed a delayed auditory comparison task involving parametric sound intensity discrimination, followed by confidence ratings. Behavioral outcomes were analyzed using generalized and linear mixed-effects models, and latent decision dynamics were assessed with drift diffusion modeling.

Results : Results indicated that adolescents with SLD showed reduced perceptual sensitivity, higher decision thresholds, and slower evidence accumulation relative to controls. Confidence ratings varied systematically with stimulus intensity and response accuracy, but distinct profiles of metacognitive calibration emerged across SLD subtypes.

Conclusion : These findings demonstrate that AWM deficits in SLD extend beyond perceptual limitations to include disruptions in self-monitoring processes. By linking auditory processing with metacognitive awareness, the study identifies potential cognitive markers that may inform both diagnostic assessment and targeted educational interventions.

Keywords : Auditory Working Memory, Specific Learning Disorders, Executive Function

Count: 30
Abstract ID: 380
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Poster

The relationship between executive functions and anxiety/depression: the mediating role of cognitive emotion regulation strategies in patients with MS.

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Background and Aim : Multiple sclerosis (MS) is a chronic, autoimmune disease of the central nervous system that, in addition to physical problems, is associated with cognitive and emotional disorders such as anxiety and depression. Executive functions play an important role in psychological adjustment, and deficiencies in this area can predispose to mood disorders. On the other hand, cognitive emotion regulation strategies have been proposed as a key factor in explaining the relationship between cognitive weaknesses and emotional symptoms. The present study aimed to investigate the relationship between executive functions and symptoms of anxiety and depression with the mediating role of cognitive emotion regulation strategies in MS patients.

Methods : The study was descriptive-correlational and the statistical population included patients referred to medical centers in Tehran in 1404. The instruments included the Anxiety and Stress Questionnaire (DASS-21), the Cognitive Emotion Regulation Strategies Questionnaire (CERQ), and neuropsychological tests (Wechsler, Stroop, and Tower of Hanoi scales). The data were analyzed using structural equation modeling.

Results : The findings showed that the relationship between executive functions and testing (β=-0.105, sig=0.369) and direct harm (β=0.046, sig=0.544) was not significant. However, the mediating role of cognitive emotion strategies was assessed: for testing (β=0.193, t=1.983, sig=0.048) and for depression (β=0.180, t=2.019, sig=0.044). In other words, patients using maladaptive strategies (such as rumination and self-blame) experienced higher levels of anxiety and depression, while adaptive strategies had a protective effect.

Conclusion : The results showed that cognitive emotion regulation is the main link between executive functions and mental health in patients with MS. Therefore, clinical interventions should simultaneously focus on strengthening executive abilities and teaching emotion regulation strategies to improve patients' psychological quality of life.

Keywords : Multiple sclerosis, executive functions, anxiety, depression, cognitive emotion regulation.

Count: 31
Abstract ID: 135
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Oral

Chronotype Modulates Risky Decision-Making: Behavioral Evidence from a Monetary Gambling Task

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Background and Aim : Decision-making under risk is a fundamental cognitive process that shapes daily choices from trivial matters to decisions with profound personal and societal consequences. Understanding the psychological factors that contribute to risky decision-making is therefore essential. One emerging factor is chronotype, the personal trait of morningness or eveningness, which has been linked to impulsivity, reward sensitivity, and self-control. Previous research on chronotype and risky decision-making has produced paradoxical findings, with some studies reporting greater risk preference in evening-types, while others found no differences or even the opposite pattern. This inconsistency highlights the need for more direct behavioral investigations. The present study investigated whether chronotype modulates behavioral responses during risky decision-making.

Methods : Thirty-nine healthy adults (20 morning-types, 19 evening-types; aged 18–31) were recruited from Tabriz University, Iran, following screening with the Morningness–Eveningness Questionnaire (MEQ) and Munich Chronotype Questionnaire (MCTQ). High-anxiety participants were excluded to minimize confounding effects. Participants completed a modified monetary gambling task that provided gain-correct, gain-error, loss-correct, and loss-error feedback. Behavioral measures included the percentage of risky decisions and reaction times (RTs) across feedback conditions.

Results : Evening-types displayed significantly higher percentages of risky choices than morning-types, particularly after gain-correct and loss-error feedback. This suggests that evening-types were more likely to continue risk engagement after both rewarding and punishing outcomes, whereas morning-types adopted a more conservative strategy. In terms of RTs, evening-types responded faster overall, with the largest difference emerging after error feedback. Reduced post-error slowing in evening-types indicates weaker behavioral adjustment following mistakes, consistent with greater impulsivity. Together, these findings suggest that evening-types display a behavioral profile marked by increased risk preference, faster decision-making, and diminished sensitivity to negative outcomes.

Conclusion : Eveningness is associated with a greater tendency toward risk-taking behavior, as well as faster and more impulsive responses. These results provide converging behavioral evidence that chronotype is a trait-linked modulator of risky decision-making, but our findings also help explain paradoxical results reported in prior studies. A key contribution of this study is clarifying the feedback-specific mechanisms underlying evening-types' risk bias. After loss-error feedback, evening-types continued to choose risky options, reflecting insensitivity to negative consequences and aligning with their higher sensation-seeking tendencies. This reduced aversion to punishment suggests that negative feedback does not effectively deter risk behavior in evening-types. Conversely, after gain-correct feedback, evening-types showed an increased tendency to pursue further risky decisions, consistent with a stronger drive for immediate reward attainment and heightened sensitivity to short-term gains. Morning-types, in contrast, demonstrated slower, more deliberate responses and greater caution following negative feedback, indicating stronger feedback-based adjustment. Taken together, these results suggest that evening-types are simultaneously more reward-driven and less punishment-sensitive, contributing to a distinct decision-making style with implications for understanding susceptibility to maladaptive real-world risk behaviors, including gambling, unsafe driving, and impulsive choices.

Keywords : Chronotype; Risky decision-making; Impulsivity; Feedback processing

Count: 32
Abstract ID: 207
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Poster

Hemispheric Alpha Coherence as a Neural Marker of Divergent and Convergent Thinking

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Background and Aim : Background and Aim: People approach problem-solving in various ways. Some generate a wide range of creative ideas, a process known as divergent thinking, while others focus on identifying a single correct solution, referred to as convergent thinking. Brain activity, particularly the coherence of alpha waves, indicates how different regions of the brain communicate during these processes. By studying how the left and right hemispheres coordinate during divergent and convergent thinking tasks, we can gain a deeper understanding of the neural foundations of creative and analytical problem-solving. This study aimed to examine the differences in alpha coherence between the two hemispheres during divergent and convergent thinking tasks, compared to a resting state, in healthy adults.

Methods : Method: The study involved 36 right-handed adults (18 males and 18 females) who were recruited through convenience and voluntary sampling from various university faculties. After excluding two participants due to insufficient task engagement and EEG artifacts, a total of 36 participants were included in the analysis. EEG signals were recorded utilizing a 19-channel Mitsar system, with a band-pass filter set between 0.1 and 70 Hz, while ensuring electrode impedances stayed below 5 kΩ. To assess divergent thinking, participants completed the Alternative Uses Task (AUT), where they were tasked with generating as many creative and unusual uses as possible for common objects. Their responses were evaluated based on fluency (the number of responses), flexibility (the number of categories), and originality (the novelty of the responses). Convergent thinking was measured using the Remote Associates Test (RAT), which required participants to identify a word that connects three provided stimulus words through either semantic or compound associations.

Results : Results: Overall, alpha-band coherence was found to be higher during divergent thinking compared to convergent thinking. When examining differences between the hemispheres, the left hemisphere exhibited greater alpha coherence during divergent thinking

than both during convergent thinking and the resting state. Follow-up analyses using Least Significant Difference (LSD) tests confirmed a significant difference between divergent and convergent thinking in the left hemisphere ($p < .001$, $d = 4.82$). However, coherence during divergent thinking did not significantly differ from the resting state ($p = .55$, $d = -0.875$). Similarly, in the right hemisphere, alpha coherence was also higher during divergent thinking compared to both convergent thinking and the resting state. LSD analyses revealed a significant difference between divergent and convergent thinking in the right hemisphere ($p < .001$, $d = 9.95$), but no significant difference was found between convergent thinking and the resting state ($p = .668$, $d = -0.629$).

Conclusion : Conclusion: This study demonstrates that divergent thinking is linked to higher alpha-band coherence compared to convergent thinking. This indicates a greater engagement of distributed neural networks. The increased coherence observed in both hemispheres suggests that creative problem-solving depends on coordinated activity across the hemispheres, highlighting alpha coherence as a potential neural marker of creativity.

Keywords : Keywords: Divergent and Convergent thinking, Alpha coherence, Hemispheric differences

Count: 33
Abstract ID: 160
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Oral

Exploring Creative Cognition: Hallucinatory VR Alters Frontal Alpha Dynamics in Creative Thinking

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Background and Aim : Creativity is a complex cognitive ability defined as the capacity to generate novel and contextually appropriate ideas. Recent research links creativity to altered states of consciousness, such as those induced by psychedelics, which place the brain in a more flexible mode. In such states, default-mode processes temporarily relax, reducing top-down constraints and allowing novel information and associations to emerge. Studying these altered states in controlled environments offers insights into the mechanisms underlying creativity. Virtual reality (VR) provides a promising tool for this purpose, as it can induce specific states of consciousness through perceptual modification and immersive experience. Hallucinatory Visual Virtual Experiences (HVVEs) alter perception in ways similar to dream-like and hallucinatory states. A key method is the Deep Dream algorithm, which transforms visual input into dreamlike, hallucinatory imagery. Prior studies show that Deep Dream exposure enhances cognitive flexibility, a core process in creativity measured by Alternative Uses Test (AUT). However, the neural activity underlying this creative thinking under Deep Dream conditions has not been investigated. The present study examines the temporal dynamics of frontal alpha-band, an established marker of creative ideation, during creative thinking following exposure to a Deep Dream–based virtual environment.

Methods : Thirty participants (13 males, 17 females; age 18–30, M = 23.43, SD = 9.35) were randomly assigned to control or experimental groups. Both underwent 2-minute resting-state EEG (eyes open/closed) before seven minutes in VR. Controls viewed a 360° courtyard video; the experimental group saw the same video altered with the Deep Dream algorithm. Participants then completed the Alternative Uses Test (AUT) while EEG was recorded during 15-second idea generation. A second 2-minute resting-state EEG followed.

Results : During the 15-second idea generation, the control group showed a gradual increase in frontal alpha power from 0–12 s, followed by slight suppression. In contrast, the experimental group exhibited an initial alpha suppression from 0–3 s, followed by a gradual increase peaking around 12 s, and then a subsequent suppression. Significant differences during the initial 3-second window were observed at F3 ($t = -2.41$, $p = 0.023$), Fz ($t = -2.28$, $p = 0.031$), and Fp2 ($t = -2.09$, $p = 0.046$). Overall, alpha showed a gradual increase followed by suppression, with the experimental group exhibiting an initial early suppression before this pattern.

Conclusion : These findings highlight distinct temporal patterns of frontal engagement during creative thinking after Deep-Dream exposure. Prior studies link increased frontal alpha during creative ideation with internal attention and reduced external distraction,—a form of inhibitory control over attention for inward search. Meanwhile, alpha suppression has been described as releasing this inhibitory control. Unlike the control group, which showed the typical gradual alpha increase during idea generation, the Deep Dream group first exhibited marked alpha suppression followed by a gradual rise. This pattern suggests that exposure to a hallucinatory environment via Deep-Dream videos can modulate creative thinking by briefly shifting toward bottom-up attention before engaging inward focus for creative associations, reflecting a more exploratory and flexible mode of thinking rather than reliance on default top-down processes.

Keywords : Creativity; Cognitive flexibility; Hallucinatory Visual Virtual Experiences; Deep-Dream; VR; Frontal alpha-band

Count: 34
Abstract ID: 320
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Oral

Cognitive Flexibility and Its Impact on Rumination: An Objective Evaluation Through 4 Cognitive Flexibility tests

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Background and Aim : Rumination is a style of response to low mood, characterized by repetitive thoughts that are focused on self, depressive symptoms, and the causes and consequences of mood. The importance of rumination is its role as an important risk factor of depression. On the other hand, cognitive flexibility is a cognitive concept, defined as flexible adjustment of thoughts and behaviors to changing environmental demands. The repetitive nature of rumination suggests a theoretically justifiable negative relation between rumination and cognitive flexibility. Since the field of studying the relation between cognitive flexibility and rumination, consists of conflicting results, the main purpose of this study was to further investigate the relation between rumination and cognitive flexibility by using different measures of cognitive flexibility. Also, a challenge in the field of cognitive flexibility, is the drastically different measures of cognitive flexibility, and a few studies have shown that the different measures of cognitive flexibility, seem to have insufficient correlations with each other; Therefore, we investigated whether the result scores from the cognitive flexibility measures load on the same factor, or they load on different factors.

Methods : 90 participants were selected from students of the psychology department of Tehran University, using convenience sampling. Participants were either healthy or only had major depressive disorder. Measures was Ruminative Response Scale (RRS), Beck Depression Inventory - second edition (BDI-II), and 4 behavioral tasks for cognitive flexibility: a subtype of Switch Task (ST), Wisconsin Card Sorting Test (WCST), Probabilistic Reversal Learning Test (PRLT), and a new type of probabilistic task with continuous responses, which we named Continuous Probabilistic Reversal Learning Test (CPRLT). We used reinforcement learning models to further investigate the data from probabilistic tests. We used exploratory factor analysis (EFA) to identify underlying factors of the cognitive flexibility test scores. We used

Lasso regression to investigate the role of cognitive flexibility test scores in predicting rumination and depression scores, due to low sample size and high number of predictors.

Results : We found 3 underlying factors for the cognitive flexibility test scores. Lasso regression was used 5 times, each time with all cognitive flexibility test scores as predictors, and one of the questionnaire scores (BDI-II score, RRS total score, and RRS subscale scores) as outcome variable. In all 5 models, lasso returned null model (all coefficients equal to zero), indicating no single or combination of cognitive flexibility test scores provided a sufficient signal-to-noise ratio to outperform a simple intercept-only model in predicting the rumination or depression.

Conclusion : We believe that the insignificant correlation between cognitive flexibility and rumination, can be due to low sample size, nature of the sample (students of psychology department), the concept of "sampling error" (a result by chance), and the concept of far transfer. The later explanation refers to the fact that when comparing assessments of cognitive flexibility and rumination, the distance between these two domains is definitely considerable, and therefore, the probability that high performance in cognitive flexibility tests can result in lower levels of rumination, according to the concept of far transfer, is low.

Keywords : Rumination; Cognitive flexibility; Probabilistic reversal learning; Task-switching; far transfer

Count: 35
Abstract ID: 720
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Poster

Trait Dominance Shapes Leaders' Reaction Times Following Prior Feedback and Adapts to Current Risk

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Background and Aim : Effective decision-making under uncertainty requires more than simply reacting to outcomes, it depends on the ability to anticipate potential errors and prepare adaptive responses in advance. This anticipatory regulation, often described as proactive control, allows individuals to bias attention and strategy toward optimal choices based on prior experience and expected outcomes. Recent evidence suggests that such anticipatory adjustments can be observed directly in behavior. For example, people tend to slow down and deliberate more carefully after negative feedback or in situations where choices are ambiguous and outcomes carry greater risk. By studying these behavioral dynamics, particularly how prior outcomes and decision stakes shape subsequent choices, we can better understand the strategies individuals use to adaptively regulate their decisions under uncertainty.

Methods : Twenty-eight corporate leaders (mean age = 40.80, SD = 6.53) participated in the study. Participants completed a computerized gambling task designed to simulate real-world decision-making under risk. On each trial, they chose between options associated with probabilistic gains or losses, with varying stake levels. Behavioral measures included reaction times (RTs) and choice patterns following different feedback conditions. Analyses focused on how prior outcomes (e.g., positive vs. negative feedback) influenced decision behavior on subsequent trials.

Results : Behavioral data revealed systematic adjustments based on prior feedback and decision context. Participants showed slower reaction times following missed gains or negative feedback, indicating heightened caution and increased conflict monitoring. Similar slowing

was observed in ambiguous decision contexts, where outcome probabilities were less predictable. In contrast, positive feedback tended to reduce decision latencies, suggesting a shift toward more automatic or confident responding. Decisions following low-stake outcomes were also faster, whereas high-stake trials elicited more deliberative behavior. These findings suggest that prior outcomes and decision stakes dynamically shape anticipatory decision strategies, with participants engaging in more cautious, controlled processing in contexts associated with higher uncertainty or regret.

Conclusion : This study demonstrates that behavioral adjustments in decision-making reflect underlying anticipatory control processes. Slower responses following negative outcomes or ambiguous conditions indicate enhanced conflict monitoring and proactive engagement of cognitive control before outcomes occur. These results provide behavioral evidence for anticipatory regulation in high-stakes decisions and offer insights into how humans adaptively allocate cognitive resources under uncertainty. Future work will extend this approach to neural measures, exploring how proactive error-related signals and individual motivational traits contribute to these adaptive mechanisms. Such insights have implications for leadership performance, risk management, and training strategies in complex decision environments.

Keywords : Decision-making; Leadership;Trait dominance; Reaction Time; Prior feedback; Risk

Count: 36
Abstract ID: 579
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Poster

Effects of Visuomotor Delay on Perceptual Decision-Making, Mental Demand, and Embodiment in Virtual Reality

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Background and Aim : The brain maintains a coherent sense of the body through continuous integration of vision, proprioception, and motor signals. Virtual Reality (VR) allows controlled disruption of this visuomotor loop, such as introducing a temporal delay between action and its visual feedback. Such delays provide a way to test how embodiment, agency, performance, and perceived effort are shaped by sensorimotor prediction. The present study examined how visuomotor delay in VR influences perceptual decision-making performance, sense of embodiment, sense of agency, and cognitive workload.

Methods : Thirty-five participants completed a three-dimensional Random Dot Motion (RDM) task in VR using controller-free hand tracking. Participants indicated perceived motion direction through “shooting” gestures under two visuomotor feedback conditions: synchronous (0 ms delay) and delayed (190 ms delay). Task difficulty was manipulated through motion coherence levels. Objective measures included reaction time and accuracy. Subjective measures included Sense of Embodiment (SoE), Sense of Agency (SoA), and perceived workload (NASA-TLX subscales). Data were analyzed with mixed models for performance and paired-samples t-tests for embodiment, agency, and workload.

Results : Objective performance was resilient to delay: reaction times, accuracy, and angular errors did not significantly differ between synchronous and delayed conditions ($p > .05$). In contrast, subjective experience was clearly altered. Paired t-tests revealed that Sense of Embodiment significantly decreased under the delayed condition ($p < .05$), with participants reporting a reduced feeling of ownership over the virtual hand. Similarly, Sense of Agency declined significantly ($p < .05$), suggesting that temporal misalignment disrupted the feeling of control over actions. Furthermore, participants reported significantly higher Mental Demand and Workload under delay ($p < .05$). Many participants qualitatively described their hands as feeling “heavier” and movements as requiring “more effort,” despite no actual physical force being added.

Conclusion : These findings demonstrate a dissociation between motor performance and subjective experience during visuomotor delay in VR. Even though task performance was maintained, embodiment and agency weakened, and participants experienced the task as more mentally demanding, often describing an illusory heaviness in their hands. This suggests that the brain compensates for sensory prediction errors not by abandoning motor performance, but by recalibrating internal body models, attributing discrepancies to increased effort and reduced ownership. Temporal precision thus plays a critical role in sustaining embodiment and agency, and its disruption imposes hidden cognitive costs even when behavior appears unaffected. These results have implications for the design of VR interfaces, neurorehabilitation tools, and human-computer interaction systems, emphasizing the importance of aligning visuomotor feedback to support both performance and subjective bodily experience.

Keywords : Virtual Reality; Visuomotor Delay; Embodiment; Agency; Effort; Workload; Random Dot Motion; Perceptual Decision Making

Count: 37
Abstract ID: 697
subject: Cognition: Executive Function (Decision Making, Reasoning, Problem Solving)
Presentation Type: Poster

Effectiveness of the game-based history education package on the executive functions and self-esteem of children with ADHD.

Submission Author: Hengameh Yaghoubi

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Background and Aim : The present study was conducted to determine the effectiveness of the game-based history education package on the executive functions and self-esteem of children with attention deficit hyperactivity disorder.

Methods : The statistical population of this research included all children suffering from attention deficit and hyperactivity disorder under education in Tehran primary schools in the academic year of 2024, and 20 of them who met the criteria for entering the study were selected using the available sampling method as a sample. They were randomly divided into experimental and control groups. The experimental group was subjected to the game-based training of the history educational package based on the protocol proposed by Labadi and Ronoghi (2009), while the control group remained waiting. Data were collected using Coolidge executive functions and Cooper Smith self-esteem questionnaires. Descriptive and inferential statistics were used in data analysis.

Results : The research results indicated that the history educational package based on the game on the executive functions of children with hyperactivity and attention deficit is effective and can be used along with other effective activities to rehabilitate children with ADHD.

Conclusion : The research results indicated that the history educational package based on the game on the executive functions of children with hyperactivity and attention deficit is effective and can be used along with other effective activities to rehabilitate children with ADHD.

Keywords : educational package of history, executive functions, self-esteem, game

Count: 38
Abstract ID: 800
subject: Cognition: Cognitive Aging
Presentation Type: Poster

Protective role of Spirulina against memory impairments and oxidative stress in an experimental model of aging

Submission Author: Neda Ebrahimi

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Background and Aim : Aging is an irreversible, and gradual loss of the function of each cell and organ tissue in the body. One of the most common problems among aging individuals is cognitive dysfunction. Oxidative stress pathways have a pivotal role in aging-induced cognitive disorders pathophysiology. Recently, various studies have evaluated the effects of various nutritional supplements in preventing age-related disorders. It has been reported that Spirulina as an antioxidant supplement had beneficial effects in neurodegenerative disease such as Alzheimer diseases. Hence present study was performed to evaluate the effect of Spirulina to prevent memory impairments, and oxidative stress in an animal model of aging in rats.

Methods : This experimental study was performed on 30 male Wistar rats. Studied groups (n=10) were including control group (fed a routine diet+ normal saline, subcutaneously), D-galactose group (fed a routine diet+ D-galactose at 300 mg/kg, subcutaneously injection, for 6 weeks), D-galactose+ Spirulina group (fed a routine diet containing 5% spirulina powder+ D-galactose at 300 mg/kg, subcutaneously injection, for 6 weeks). At the end of the sixth week, memory function was determined by the passive avoidance test. Finally, the rats were decapitated, and their brains were carefully removed from the skull. Then, hippocampal malondialdehyde (MDA) levels and superoxide dismutase (SOD) activity as oxidative stress biomarkers were evaluated by colormetric assay and ELISA kits, respectively.

Results : The D-galactose group showed less step-through latency than control group. However, D-galactose+ Spirulina group compared to D-galactose group showed more step-through latency. In addition, D-galactose injection led to elevated MDA levels, and decreased SOD activity in the hippocampus compared to control group. Notably, diet containing Spirulina effectively reversed these deleterious effects compared to D-galactose group.

Conclusion : These findings indicated that diet containing Spirulina may prevent memory impairments in an animal model of aging induced by D-galactose, suggesting its promise as a treatment for age-related neurodegenerative diseases.

Keywords : Aging, memory, oxidative stress, rats

Count: 39
Abstract ID: 476
subject: Cognition: Cognitive Aging
Presentation Type: Oral

The effectiveness of the stop signal task on problem solving in the elderly

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Background and Aim : Besides disability and physical problems, elderly is accompanied by impairment in many cognitive functions including working memory, problem solving, and impulsivity, which affects her quality of life. We investigate the effectiveness of the stop sign task on problem solving in the elderly.

Methods : Thirty older adults were purposively selected and randomly assigned to either the experimental group or the control group (n = 15, in each group). The experimental group received 12 sessions of cognitive rehabilitation based on the Stop Signal Task, while the control group received no intervention, then problem solving. Data were analyzed using paired t-tests for within-group comparisons and mixed analysis of covariance (MANCOVA) to assess the effects of the intervention

Results : The results showed that the Stop Signal Task had a significant effect on improving problem-solving in older adults ($p < 0.05$). Comparison of posttest scores revealed significant improvements in all variables in the experimental group compared to the control group

Conclusion : The findings suggest that cognitive rehabilitation based on the Stop Signal Task can be an effective intervention to improve cognitive functions in older adults. These results may serve as the basis for designing rehabilitation programs for elderly individuals with cognitive impairments

Keywords : stop signal task: problem solving:

Count: 40
Abstract ID: 478
subject: Cognition: Cognitive Aging
Presentation Type: Oral

new approach about the neuroprotective effect of opioid drugs in the treatment od alzheimer,s disease

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Background and Aim : the role of opioids in AD were controversial. in this paper some new studies were investigated. The deposition of intracellular and extracellular b-amyloid peptide (Ab) in the brain is a pathologic feature of Alzheimer's disease (AD), a prevalent neurodegenerative disorder.

Methods : in this review article we investigate new studies about neuroprotective effects of opioids

Results : Some studies showed neuroprotective effect of moerphine and dextrometrphan

Conclusion : morphine via NO and glial cells interact in beta amyloids metabolism This suggests that a defi ciency of basal NO or endogenous morphine may trigger drastically reduced levels of basal NO. The outcome is chronic vasoconstriction and brain hypoperfusion and eventual neuronal death. This novel theorized mechanism for AD supports an increasingly-accepted vascular pathological hypothesis for the disease.

Keywords : opioid, morphine, neuroprotective, Alzheimer, NO, Amyloid beta

Count: 41
Abstract ID: 345
subject: Cognition: Consciousness
Presentation Type: Poster

Eliciting Awe in Virtual Reality Using Complex Fractal Structures

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Background and Aim : Watching a sunset, gazing at a starry night sky, solving mathematical problems, or observing vast natural landscapes can evoke a shared human emotion known as awe. Awe is a complex emotion that integrates both negative and positive feelings, is multidimensional, and can expand the boundaries of perception. Awe arises from the perception of vastness or complexity and requires cognitive accommodation. Psychological studies have shown that awe has psychological benefits. Consequently, inducing awe is not only a fascinating cognitive phenomenon but also an important factor for mental health. Virtual reality (VR) provides a unique platform for eliciting awe by offering immersive, multisensory experiences under controlled experimental conditions. VR allows individuals to encounter scenarios otherwise difficult or impossible to access in daily life, such as space travel, observing vast natural wonders, or exploring imaginary worlds. Such experiences can more profoundly activate perceptions of vastness and complexity, increasing the likelihood of awe. Among potential stimuli, fractal patterns—with their self-similar structures—may also elicit awe. This study examined the potential of fractal patterns to elicit awe in Virtual reality

Methods : Using a within-subjects design, 24 participants (12 males and 12 females) viewed three different 360-degree 4K videos in virtual reality. While their heart rate and skin conductance were recorded using physiological sensors, participants listened to a neutral piece of music through headphones to facilitate immersion. After each video, participants completed the Awe Experience Scale

Results : The results demonstrated that fractal patterns can effectively elicit awe experiences. Physiological data indicated that mere exposure to the virtual reality environment increased arousal compared to the resting condition. However, analysis of the low-frequency component of the blood volume pulse—primarily associated with sympathetic activity—revealed that activity in this band was significantly lower in the fractal condition than in the control condition.

Conclusion : This study is the first to investigate how complex patterns can induce awe. Fractals, as complex and self-similar visual patterns, were found to elicit awe. While virtual reality in general elevated physiological arousal compared to rest, the quality of this arousal differed depending on the stimulus. Specifically, fractal stimuli were associated with reduced sympathetic activity, producing a form of arousal that was more balanced, adaptive, and less stressful.

Keywords : Awe; Virtual Reality; VR; Fractal; complexity

Count: 42
Abstract ID: 361
subject: Cognition: Consciousness
Presentation Type: Oral

Enhancing Interoceptive Function via Insular Neuromodulation: A Connectivity-Based EEG Study

Submission Author: Alireza Fallahi

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Background and Aim : Interoception refers to the perception and interpretation of internal physiological signals from organs such as the heart, lungs, and gastrointestinal system. It underlies homeostasis, emotional awareness, and adaptive decision-making. The insular cortex plays a central role in integrating sensory and intracortical information, regulating emotions, and linking bodily states to cognition. Functional disturbances in this region are associated with various psychiatric and neurological disorders, including anxiety and depression. Investigating how electrical stimulation affects EEG connectivity in the insula is essential for understanding stimulation-induced network dynamics and their impact on interoceptive processes. Such insights can guide the design of targeted neuromodulation protocols aimed at improving emotional regulation and treating insula-related disorders. This study seeks to provide evidence to enhance the effectiveness of brain stimulation in promoting interoception and supporting the development of personalized therapeutic interventions.

Methods : Nineteen healthy volunteers (10 women, 9 men; age range 20–25 years) participated in this study, conducted at the Cognitive Neuroscience Laboratory, Department of Biomedical Engineering, Hamadan University of Technology. Each participant underwent two EEG recording sessions: (1) a pre-stimulation session with sham stimulation to mimic the sensation of stimulation and prevent bias, and (2) a post-stimulation session with real transcranial direct current stimulation (tDCS) applied for 20 minutes at 2 mA. EEG was recorded for 5 minutes in each session using a 19-channel Neurosurve system following the international 10–20 montage (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T3, T4, T5, T6, Fz, Cz, Pz). A Neurostim device delivered the tDCS. Brain connectivity changes were assessed using Pearson correlation, and statistically significant differences were identified using false discovery rate (FDR) correction.

Results : Insular stimulation induced notable alterations in brain network connectivity. Significant changes were observed between the P4–T4, P4–P3, P3–T3, and P3–O1 connections ($p < 0.05$, FDR-corrected). Analysis of the connectivity matrices revealed that temporal and posterior channels exhibited the most pronounced modulations. This pattern suggests that insular stimulation selectively influences networks involved in cognitive and sensory processing, reshaping inter-regional communication pathways.

Conclusion : This study demonstrates that targeted electrical stimulation of the insula can significantly modulate EEG-based brain connectivity, particularly in networks associated with cognitive and sensory functions. By enhancing the strength of inter-regional interactions, insular stimulation shows promise as a neuromodulatory tool for cognitive enhancement and as a potential therapeutic intervention for neurological and psychiatric conditions involving disrupted interoceptive or affective processing.

Keywords : Interoception, tDCS, Connectivity, EEG

Count: 43
Abstract ID: 77
subject: Cognition: Consciousness
Presentation Type: Oral

How does the brain generate consciousness?

Submission Author: Majid Beshkar

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Background and Aim : The problem of consciousness is one of the most difficult problems in science, which has remained unsolved despite several decades of research. To solve the problem of consciousness, empirical evidence alone is not sufficient; we must also have an appropriate theory that can organize and integrate diverse empirical findings to reveal a hidden pattern. In this context, the QBIT theory is an attempt toward solving the puzzle of consciousness with pieces of evidence collected from different scientific disciplines including quantum mechanics, biology, information theory, and thermodynamics.

Methods : This is a multidisciplinary theoretical investigation into the problem of consciousness. Here, the basic assumptions and subsequent implications of the QBIT theory serve as a framework for explaining what consciousness is and how the brain generates it.

Results : The following is a list of basic assumptions and propositions of the QBIT theory: (1) When information-theoretic uncertainty of the brain about an external stimulus decreases below a critical threshold, the brain becomes conscious of that stimulus. Therefore, the emergence of consciousness in the brain is critically dependent on the minimization of uncertainty. (2) Minimization of uncertainty is an information-theoretic (or computational) process that must be performed (or realized) by a physical substrate. Minimization of uncertainty at a computational level corresponds to the minimization of thermodynamic entropy at a physical level. (3) Microtubules are the physical substrate by which the brain computes. At a fundamental level, sensory information processing in the brain is performed by the microtubules inside the neurons. (4) Due to the correspondence between uncertainty and entropy, minimization of uncertainty in the brain corresponds to the reduction of entropy in neuronal microtubules. Imagine an ensemble of microtubules in a population of cortical pyramidal neurons that are responsible for the processing of an external stimulus. Reduction of the entropy in this ensemble of microtubules is equivalent to minimization of uncertainty of the brain about that stimulus. (5) Thermodynamic entropy is deeply related to quantum coherence. A quantum coherent state is characterized by the minimum possible entropy. (6) The emergence of consciousness in the brain is associated with the spontaneous emergence of

coherence in a population of microtubules located in the axon initial segment of cortical pyramidal neurons. (7) The spontaneous emergence of coherence in an ensemble of microtubules is equivalent to the spontaneous decline of entropy in that ensemble, which, in turn, is equivalent to minimization of uncertainty of the brain about the stimulus being processed by that ensemble.

Conclusion : From the perspective of the QBIT theory, the emergence of consciousness in the brain requires a specific substrate, a suitable environment, and a particular mechanism. The substrate is the microtubule, the environment is the axon initial segment, and the mechanism is the spontaneous emergence of coherence.

Keywords : Consciousness, Microtubule, Axon initial segment

Abstract ID: 292
subject: Cognition: Consciousness
Presentation Type: Oral

Individual differences in response to virtually induced hallucinations on object recognition task with moderating role of neuroticism

Submission Author: Nooshin Moghadam

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Background and Aim : Human perception is a dynamic process where the brain actively interprets noisy sensory inputs to form better models. this system continuously generates predictions and minimizes "prediction error" to reduce uncertainty of environment. This study utilizes an immersive virtual reality technology that uses DeepDream algorithms to systematically alters state of consciousness by noisy visual input. This disruption of normal perception is thought to engage higher-order cognitive processes, as the brain must update its internal models to make sense of the novel and uncertain sensory information. This study aimed to investigate how this induced altered state of consciousness effects object recognition and how this process is moderated by the personality trait of neuroticism.

Methods : Forty four participants were randomly assigned to an intervention (DeepDream-altered 360° “hallucination machine”) or control (original VR video) group and completed a fragmented ambiguous-object recognition task before and after the exposure. to measure interactions between the intervention, time, and neuroticism and also to account for the random effects of participants and trials mixed-effect models were used. linear mixed model was used for reaction time, a cumulative link (ordinal) mixed model for confidence ratings(Likert scale from 1 to 7), and a generalized linear mixed model for the binary response data. To investigate the moderating role of neuroticism, neuroticism subscale of NEO questionnaire was added as a continuous fixed effect predictor to these models.

Results : Analysis of reaction times revealed a significant Group × Condition interaction, indicating that the intervention group became significantly faster after the exposure compared to control group (β = -0.061, p = .025), though the effect size was small (ηp^2 = 0.0013). Additionally, a significant Condition × Neuroticism interaction was observed (β = 0.0019, Std. Coef. = 0.10, p = .011), where individuals with lower neuroticism demonstrated greater speed improvements over time, irrespective of their group. Furthermore, the intervention induced a significant shift in response bias. The odds of a ‘yes’ response for the intervention group increased by 32% (OR = 1.32, p = .011). While the intervention had no significant effect on confidence ratings, a main effect of neuroticism personality trait was found, where higher neuroticism was independently associated with lower confidence across all conditions, representing a medium to large effect (β = -0.025, Std. Coef. = -0.73, p = .012).

Conclusion : The hallucination machine exposure resulted to faster reaction time and increase in recognition rate of ambiguous objects, which moderated by neuroticism trait without altering participants' confidence in their judgments. This dissociation between performance gain and unchanged confidence might suggest that the intervention might not effect mechanisms involving metacognitive process and needs further investigation.These findings collectively advance our understanding of how induced altered states can systematically modulate the brain's predictive processes to enhance perceptual adaptation.

Keywords : Hallucination Machine; Object recognition; Altered States of Consciousness; Neuroticism; Virtual Reality

Count: 45
Abstract ID: 202
subject: Cognition: Attention
Presentation Type: Poster

The Neurophysiological Signature of Attentional Bias in Irritable Bowel Syndrome: An ERP Study

Submission Author: Reyhaneh Akbari

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3. Assistant Professor
4. Associate Professor

Background and Aim : Irritable Bowel Syndrome (IBS) is a common disorder of gut-brain interaction. While attentional bias towards illness-related stimuli is a recognized cognitive feature of IBS, its underlying neurophysiological mechanisms remain poorly understood. This study aimed to investigate the neural correlates of attentional bias in IBS patients compared to high-anxiety individuals and healthy controls using event-related potentials (ERPs).

Methods : Three groups of participants: patients with IBS (n=15), high-anxiety individuals (n=15), and healthy controls (n=15) were recruited. All participants completed a modified dot-probe task with bodily (bloating, pain) and facial (anger, disgust) threatening stimuli. Continuous electroencephalogram (EEG) was recorded during the task. We analyzed ERP components (P200, N200, P300, and LPP).

Results : Behavioral results partially confirmed attentional bias towards bodily stimuli in the IBS group. Neurophysiological findings revealed a distinct pattern. ERP Findings: The IBS group showed significantly enhanced amplitudes of P200, N200, P300, and LPP in response to bodily stimuli, particularly over parietal and fronto-temporo-parietal regions.

Conclusion : The findings provide strong neurophysiological evidence for the attentional bias hypothesis in IBS. The observed ERP and ERSP patterns suggest that IBS is not merely a disorder of the gut, but also involves a specific pattern of central nervous system processing characterized by enhanced automatic attention (P200), sustained emotional engagement (LPP) in response to bodily symptoms. This distinct neural signature differentiates IBS from general anxiety and underscores the importance of a comprehensive brain-gut axis model. These results pave the way for developing targeted neuro-behavioral interventions and utilizing these neurophysiological markers as objective indices for treatment efficacy.

Keywords : Irritable Bowel Syndrome, Attentional Bias, ERP, Gut-Brain Axis, Neurophysiology, Dot-Probe Task

Count: 46
Abstract ID: 328
subject: Cognition: Attention
Presentation Type: Poster

Effects of Infra-Low-Frequency Neurofeedback on Behavioral Outcomes in Children with Autism

Submission Author: Fatemeh Samadkhah

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Background and Aim : Children with autism spectrum disorder (ASD) often exhibit deficits in selective attention. Neurofeedback has shown promise in improving attentional control, but the effects of Infra-Low-Frequency (ILF) neurofeedback on both behavior and brain activity require further exploration.

Methods : Nine children aged 4–8 years participated in a single-group experimental design. Behavioral assessments included standardized measures of autism symptoms and selective attention, alongside EEG recordings. Participants underwent 15 sessions of ILF neurofeedback over five weeks. Post-intervention and short-term follow-up evaluations were conducted.

Results : Participants showed improvements in autism-related behaviors and selective attention following ILF neurofeedback. EEG analyses indicated changes in brain activity patterns, consistent with behavioral improvements. The findings suggest a link between neurofeedback-induced neural changes and enhanced attentional control.

Conclusion : ILF neurofeedback may serve as a complementary intervention for improving behavioral outcomes in children with ASD. While these preliminary results are promising, caution is warranted due to the small sample size and lack of a control group. Further studies with larger samples are needed to confirm these effects.

Keywords : Autism spectrum disorder; ILF neurofeedback; selective attention; EEG; behavioral intervention

Count: 47
Abstract ID: 637
subject: Cognition: Attention
Presentation Type: Oral

A Network Approach to Eye-Gaze Data for Assessing Attentional Bias in Depression

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Background and Aim : Available data regarding attentional biases in depression are less conclusive than for memory and interpretation biases. One potential reason is that group differences in attention allocation to emotional stimuli may be subtle and difficult to detect using traditional fixation metrics such as fixation duration and count, which provide static summaries of attention but fail to capture its dynamic nature. We propose that network-based measures, such as degree centrality—which captures the relational dynamics of attention deployment—can more sensitively index attentional bias.

Methods : In this study, individuals with (N = 29) and without (N = 28) depression completed the Persian Scrambled Sentences Task while their eye movements were recorded. Attentional bias was assessed using both classic fixation metrics (frequency and duration) on positive and negative words, and a network approach whereby degree centrality was computed from gaze transition networks between defined areas of interest.

Results : Both traditional metrics of fixation count and fixation duration and the network analysis of fixation points revealed a similar pattern of attention allocation to emotional stimuli in depressed and non-depressed groups. However, the network metric degree centrality demonstrated a significant correlation with rumination within the depressed group, unlike fixation duration and count. This finding reveals a nuanced aspect of attentional bias closely linked to emotion dysregulation in depression, a relationship undetected by traditional fixation analyses.

Conclusion : The dissociation between degree centrality's inability to differentiate diagnostic groups and its significant association with rumination suggests that degree centrality may capture clinically relevant attentional dynamics tied to emotion dysregulation rather than serve merely as a marker of diagnostic status. This underscores the value of network-based gaze metrics in identifying subtle attentional patterns relevant to the emotional processes underlying depression.

Keywords : attentional bias; depression; eye-tracking; network analysis

Count: 48
Abstract ID: 708
subject: Cognition: Attention
Presentation Type: Poster

Chronic Constriction Injury and Cognitive Behaviors: Investigating the Neuroprotective Role of Progesterone

Submission Author: Fariba Akhondzadeh

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Background and Aim : Chronic pain disrupts sensory processing and may also impair cognitive and social behaviors. Progesterone has been proposed as a neuroprotective agent with potential analgesic effects, but its broader behavioral consequences remain incompletely understood.

Methods : In this study, 16 adult male rats were randomly assigned to four groups: Sham + Vehicle, Sham + Progesterone, CCI + Vehicle, and CCI + Progesterone. Neuropathic pain was induced using the chronic constriction injury (CCI) model, while sham-operated animals underwent nerve exposure without ligation. Progesterone (15 mg/kg) dissolved in sesame oil, or vehicle alone, was administered intraperitoneally once daily for 14 days after CCI surgery. Mechanical hyperalgesia was assessed using the paw withdrawal threshold test. On day 14 post-surgery, the novel object recognition (NOR) test, the object-based attention test (OBAT), and the three-chamber sociability assay were conducted to evaluate memory, attention, and social interaction, respectively.

Results : Mechanical hyperalgesia was significantly greater in the CCI + Vehicle group compared with the Sham + Vehicle group. Progesterone treatment (CCI + Progesterone) significantly reduced pain responses on days 7 and 14. In the NOR test, exploration time

differed between the CCI and Sham groups, but this difference did not reach significance. Progesterone treatment reduced exploration time in the CCI group compared with CCI + Vehicle, though the effect was not significant. In the OBAT, recognition index scores showed a non-significant increase. In the sociability test, social novelty time was reduced in the CCI group compared with Sham, but the difference was not significant. Progesterone administration did not significantly alter social novelty time.

Conclusion : These findings demonstrate that CCI reduced sociability but did not significantly affect memory or attention. Although progesterone produced a robust analgesic effect in the CCI model of neuropathic pain, it had no significant influence on attention or sociability and appeared to impair recognition memory. These results suggest that the therapeutic benefits of progesterone may be largely restricted to nociceptive processing rather than extending to broader cognitive or social domains.

Keywords : Progesterone, Chronic constriction injury, Cognition, Social behavior

Count: 49
Abstract ID: 493
subject: Cognition: Attention
Presentation Type: Oral

Visual attention modulation by the dopaminergic system in the medial prefrontal cortex (mPFC)

Submission Author: Amirhossein Darvish

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Background and Aim : Visual attention is a core cognitive ability, and its disruption is strongly linked to several psychiatric and neurodegenerative conditions, including ADHD, neglect syndromes, Alzheimer's disease, and schizophrenia. Among the key modulators of attention, dopamine plays a central role. Dopaminergic neurons originating in the midbrain send projections to the prefrontal cortex (PFC), where they shape neuronal activity and regulate attentional processes. In this study, we investigated how dopamine contributes to the regulation of membrane potential in prefrontal neurons, and how these mechanisms may underlie the modulation of visual attention.

Methods : Eight-week-old male and female mice were anesthetized with urethane and prepared for cranial surgery targeting the medial prefrontal cortex (m-PFC). To explore dopaminergic modulation of visual attention, we combined in vivo whole-cell patch-clamp recordings with pharmacological and stimulation approaches in both anesthetized and awake conditions. Specifically, we assessed the impact of ventral tegmental area (VTA) stimulation, local inhibition of the PFC using Muscimol, and blockade of dopaminergic signaling via flupentixol. The latter, a non-selective dopamine receptor antagonist, was employed to clarify whether the observed effects were mediated through prefrontal dopamine receptors.

Results : Our experiments revealed that inhibition of the prefrontal cortex markedly impaired visual attention, as reflected by a decline in response accuracy during attentional tasks. In contrast, stimulation of the ventral tegmental area (VTA) suppressed neuronal firing activity, which also contributed to diminished attentional performance. Furthermore, blockade of dopamine receptors with flupentixol reduced both neuronal spike rates and behavioral accuracy, underscoring the critical role of dopaminergic signaling in the modulation of attention.

Conclusion : Taken together, these findings emphasize the multifaceted role of dopamine in shaping visual attention and highlight the prefrontal cortex as a key regulator of this process. Elucidating the interaction between dopaminergic pathways and prefrontal activity may offer valuable insights into the mechanisms underlying attention-related disorders and could inform future therapeutic strategies.

Keywords : Dopaminergic system, Visual attention , Prefrontal cortex , in vivo whole-cell patch-clamp

Count: 50
Abstract ID: 189
subject: Cognition: Attention
Presentation Type: Poster

The effectiveness of the "Thinking and creativity Gym training program" on the attention of children with behavioral problems

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Background and Aim : This study aimed to investigate the effectiveness of the “thinking and creativity gym training program” on the attention and creativity of children with behavioral problems.

Methods : The overall design of the study was a quasi-experimental study with a control and experimental group and pre-test-post-test stages. Both variables were measured and analyzed in two groups (experimental-control) before and after the experiment. The statistical population includes 20 children aged 6 to 10 in primary school with behavioral problems in the academic year 1402-1403 in Tehran and were assigned randomly as the experiment and the control group. Then the Digit span sub-test of Wechsler intelligence scale (1986) was used to measure the attention of the participants. The collected data were analyzed using descriptive and inferential statistics using SPSS26 software.

Results : The obtained data showed that there was a significant difference in attention scores in children with behavioral problems in the experimental group (the group that received the intervention) with attention scores of children with behavioral problems in the control group. Therefore, it can be concluded that “Thinking and creativity training Brain Gym” program can play an important role in improving the cognitive and creative skills of children with behavioral problems.

Conclusion : These programs help children strengthen their abilities in the areas of attention, concentration, and creativity by using activities such as creative problem solving, critical thinking, creative games, attention exercises, and art activities, which hopefully can also reduce their behavioral problems. Consistent implementation of these programs in collaboration with families and schools can yield better results and help children develop with reduced problems.

Keywords : attention, brain gym, children with behavioral problems, creativity, thinking and creativity training program.

Count: 51
Abstract ID: 146
subject: Cognition: Attention
Presentation Type: Oral

Attentional Neuromodulation-Enhanced Auditory Training in Children with Hearing Loss

Submission Author: Nayiere Mansouri

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Background and Aim : Successful communication critically depends on the ability to perceive speech in noisy environments, a skill that is particularly compromised in children with hearing impairment. Auditory attention is a fundamental factor in speech-in-noise perception, and targeted training can improve this ability. In recent years, neuromodulation techniques have emerged as adjunctive tools in rehabilitation. Among these, transcranial direct current stimulation (tDCS) has shown promise by modulating cortical excitability and facilitating long-term potentiation (LTP). We hypothesized that coupling electrical stimulation with behavioral auditory attention training would accelerate learning processes and enhance the effectiveness of rehabilitation, thereby improving speech-in-noise comprehension.

Methods : A pilot study was conducted involving eight children with moderate to severe hearing loss. Participants underwent auditory attention training combined with either real or sham tDCS applied to the right and left dorsolateral prefrontal cortex. Behavioral outcomes were assessed using the TEA-CH and mSAAT tests, while electrophysiological measures included recording the P300 component as an index of attentional processing. Speech-in-noise performance was evaluated using the Word-in-Noise, CV-in-Noise, and BKB-SIN tests. All assessments were performed at baseline, immediately after the intervention, and at a one-month follow-up.

Results : Both groups demonstrated improvements across behavioral and electrophysiological measures following training. However, the real stimulation group showed significantly greater gains in mSAAT, TEA-CH, Word-in-Noise performance, and P300 latency compared to the sham group.

Conclusion : The integration of behavioral auditory attention training with neuromodulation through tDCS may enhance rehabilitation outcomes in children with hearing loss. This combined approach not only improves immediate.

Keywords : Auditory Training, Neuromodulation, Transcranial Direct Current Stimulation, Hearing Loss, Speech Perception in Noise, Auditory Attention

Count: 52
Abstract ID: 122
subject: Cognition: Attention
Presentation Type: Poster

Cognitive Problems in Children with Hearing Loss: A Review of Research Evidence

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Conclusion : Background: Children with hearing loss, in addition to experiencing language and communication difficulties, face challenges across various cognitive domains. Reduced auditory input can influence cognitive processes such as working memory, attention, executive functioning, and processing speed. Methods: A literature search was conducted for Persian and English articles published between 2000 and 2025 using the keywords cognition, children with hearing loss, hearing aid, cochlear implant, attention, working memory, executive functioning, and auditory information processing. Results: Studies on working memory have shown that children with hearing loss perform more poorly than their hearing peers on verbal working memory tasks, although their visual memory is generally less affected. Research on attention indicates that these children experience more difficulty focusing on auditory stimuli and shifting attention between stimuli, which can negatively impact their academic performance. Executive functions, such as planning, organization, inhibitory control, and cognitive flexibility, have also been reported to be weaker in many children with hearing loss. Additionally, studies suggest that due to limited access to linguistic input, the auditory-verbal processing speed of these children is often reduced. Conclusion: Cognitive difficulties in children with hearing loss can directly and indirectly affect their language development, social interactions, and academic achievement. Early identification of these challenges, along with the implementation of multidimensional interventions (including cognitive rehabilitation, training in executive skills, and educational support), plays a crucial role in improving their quality of life.

Keywords : cognition, children with hearing loss, hearing aid, cochlear implant, attention, working memory, executive functioning, and auditory information processing

Count: 53
Abstract ID: 186
subject: Cognition: Attention
Presentation Type: Poster

Nicotine Modulation in Primary Visual Cortex Alters Detection Performance in Macaque: Behavioral Overlap with Attentional Disruption

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Background and Aim : The cortical cholinergic system plays a pivotal role in modulating higher-order brain functions such as attention and sensory processing. Allocating attention to a particular location enhances psychophysical detectability and discriminability of visual stimuli at the attended location. Evidence suggests that acetylcholine modulates primary visual cortex sensitivity to visual stimuli, which may influence perceptual processes. Here, we investigated whether local nicotine injection into the primary visual cortex affects perceptual sensitivity in a way comparable to attention shifts induced by a Posner cueing task.

Methods : One male rhesus macaque was trained in two tasks: a visual detection task under different contrast levels and a spatial attention task using a Posner cueing paradigm. Following surgical implantation and electrophysiological mapping, we injected nicotine locally into the left V1 using a microinjectrode. We compared behavioral performance pre- and post-injection, and across cueing conditions.

Results : Nicotine injection reduced sensitivity to different contrast stimuli, specifically in the contralateral visual field. These behavioral effects resembled those seen during invalid cueing in the attention task. However, only nicotine affected reaction times, which remained unchanged across cueing conditions.

Conclusion : Local cholinergic enhancement in V1 alters perceptual sensitivity and decision-making behavior in ways that partially mimic spatial attention disruption. However, nicotine's unique effect on reaction time suggests distinct contributions of cholinergic modulation and attentional control in shaping visual performance.

Keywords : Nicotine, Visual Cortex, Sensory Processing, Spatial Attention, Macaque

Count: 54
Abstract ID: 825
subject: Cognition: Attention
Presentation Type: Poster

Neural Correlates of Symmetry and Color Harmony in the Aesthetic Perception of Persian Carpet Patterns: A Preliminary EEG Study

Submission Author: Ali Aslefathi

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Background and Aim : Persian carpets represent, renowned for their intricate geometry, symmetrical balance, and symbolic color composition, offering a rich model for neuroaesthetic investigation. Perceiving such structured patterns may recruit brain systems related to attention, perceptual fluency, and aesthetic evaluation. The present study aimed to explore the neural correlates of symmetry and color harmony during the aesthetic perception of Persian carpet motifs.

Methods : A 2×2 mixed factorial design examined the effects of symmetry (high vs. low) and color harmony (balanced vs. disrupted) within-subject factors, with artistic expertise (art vs. non-art students) as a between-subject factor. Twenty participants (10 art, 10 non-art; aged 20–30) viewed 40 digital Persian carpet images under controlled luminance and distance conditions. Each stimulus was displayed for 5 seconds with a 2-second interstimulus interval. EEG was recorded using a 21-electrod (10–20 system), band-pass filtered at 1–45 Hz, preprocessed via independent component analysis (ICA), and analyzed for alpha (8–12 Hz) and theta (4–7 Hz) band power using Fast Fourier Transform (FFT).

Results : Participants showed greater engagement with symmetrical and color-harmonious patterns, as reflected by trends in posterior alpha and frontal theta activity. Effects appeared more pronounced among art students, suggesting potential modulation of neuroaesthetic processing by expertise. These observations are preliminary and require empirical confirmation.

Conclusion : This preliminary findings indicate that Persian carpet patterns evoke neural signatures linked to perceptual fluency and aesthetic evaluation. These results highlight the potential of cultural neuroaesthetics as a bridge between traditional art and brain science, illustrating how symmetry and color harmony shape the aesthetic experience at the neural level.

Keywords : EEG, Neuroaesthetics, Persian Carpet, Symmetry, Color Harmony, Cultural Cognition

Count: 55
Abstract ID: 833
subject: Cognition: Attention
Presentation Type: Oral

Effects of Short Vertical Videos on Visual Attention and Beta-Band EEG Activity: Evidence from Stroop and Eye-Tracking Measures

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Background and Aim : Short-form vertical videos have become pervasive in digital media, yet their impact on visual attention and cognitive control remains underexplored. This study investigated how brief vertical videos and static images affect attentional performance and beta-band EEG activity in healthy young adults.
Methods : Twenty students from Islamic Art University of Tabriz participated in a within-subjects design study. Each participant underwent two conditions: watching short vertical videos and viewing static images. Cognitive performance was measured using the Stroop task. EEG was recorded from six channels (FP1, FP2, FZ, CZ, PZ, FPZ) and analyzed for absolute beta power. Eye-tracking data were collected and analyzed qualitatively. Paired-sample t-tests assessed pre- versus post-intervention differences, and Pearson correlations examined associations between Stroop performance and EEG beta activity.
Results : Analysis of the Stroop Task revealed significant differences between congruent and incongruent conditions following both image and video viewing, except for the incongruent video condition ($p > 0.05$). Regarding EEG beta power, only the PZ channel showed a significant increase in absolute beta activity after the visual interventions ($p < 0.05$), while no other channels demonstrated notable changes. Furthermore, beta power in the FZ channel did not significantly correlate with Stroop performance in either condition ($p > 0.05$). Eye-tracking data showed that participants fixated more on dynamic elements, faces, and textual content in videos, suggesting selective visual attention patterns.
Conclusion : Short vertical videos and static images modulate visual attention and cognitive processing, with beta activity increases localized to the PZ channel, reflecting enhanced spatial-attentional processing. The lack of significant FZ-Stroop correlations suggests limited impact on executive control. Qualitative eye-tracking results corroborate EEG findings, highlighting attentional allocation toward salient visual features. Future studies with larger samples and longitudinal designs are recommended to clarify the neural mechanisms underlying short-form media exposure.
Keywords : EEG ; Eye-Tracking; Stroop Task; Beta Power; Visual Attention; Short Vertical Videos

Count: 56
Abstract ID: 927
subject: Cognition: Attention
Presentation Type: Oral

Motivational Salience Beyond Valence: Reward and Penalty Histories Enhance Visual Search Efficiency

Submission Author: Sara MotiBirjandi

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Background and Aim : Due to the limitations of our visual system and processing capacity, we rely on visual search and attentional guidance in everyday life. As both rewards and threats are essential for survival, motivational salience is a key factor that directs attention. Previous research has shown that in non-human primates, stimuli with a reward history are salient and can be efficiently located during visual search. However, while reward-associated attention has been extensively studied, less is known about how objects linked to negative valence (penalty) influence attention, with contradictory findings reported in both humans and non-human primates. The present study aims to directly investigate how objects with reward and penalty histories modulate attentional allocation during a visual search task, and to examine the relationship between valence and saliency.

Methods : To examine the influence of reward and penalty history on attentional guidance, 72 unique fractal stimuli were generated with randomly assigned visual parameters. . Stimuli were divided into neutral and valence groups, the latter containing 36 fractals associated with varying reward and penalty levels (9 per condition). Eighteen adult participants took part in the experiment and received monetary compensation based on task performance. The experiment consisted of three consecutive days of training sessions, followed by a test session on the final day. During training, participants learned the value associations of the fractals through binary choice trials, in which two fractals appeared on the left and right sides of the screen, and participants selected one using the corresponding arrow key. In addition, participants completed unitary choice trials, where a single fractal was presented with its potential valence displayed at the top of the screen. On the final day, participants completed a visual search task with two blocks: one featuring reward-associated targets and the other penalty-associated targets, each presented among neutral distractors. Participants had to get 75% correct in the unitary choice trials to be included for further analysis (15 out of 18 subjects).

Results : All included participants successfully learned the value associations across categories, with no significant differences in learning performance among the valence groups. However, during the visual search task, participants exhibited superior performance when searching for both high-reward and high-penalty fractals. Specifically search times were significantly shorter for high-reward (M = 1.56 s) compared to low-reward (M = 1.80 s; $t(14) = -6.63$; $p < 0.0001$), and for high-penalty (M = 1.79 s) compared to low penalty (M = 2.12 s; $t(14) = -5.29$, $p < 0.001$). Similarly, the number of saccades required to locate the target was lower for high-reward (M = 4.83) compared to low-reward (M = 5.53; $t(14) = -4.78$; $p < 0.001$), and for high-penalty (M = 5.57) compared to low-penalty (M = 6.69; $t(14) = -5.94$; $p < 0.0001$).

Conclusion : The observed improvements in both search time and saccadic efficiency for high-reward and high-penalty fractals indicate stimuli with motivational significance, regardless of valence are, prioritized by the visual system. These findings suggests that valence and saliency are not linearly related, and that both rewarding and aversive experiences enhance attentional prioritization during visual search.

Keywords : visual search; attention; value; motivational salience

Count: 57
Abstract ID: 963
subject: Other: Other
Presentation Type: Poster

Early life maternal separation induces changes in the neuronal activity of lateral paragigantocellularis nucleau in response to morphine challenge

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Background and Aim : Early life stress can be a significant factor contributing to individual variations in the ability to cope with challenges throughout one's life. Maternal deprivation, which serves as a model for early life stress, can lead to enduring modifications in brain function and may represent a risk factor for the subsequent development of drug use disorders. Emerging evidence suggests that early life maternal deprivation increases the likelihood of opiate abuse during adulthood. The lateral paragigantocellularis (LPGi) nucleus, which is involved in opiate dependence and withdrawal, may be particularly vulnerable to changes as a result of maternal deprivation.
Methods : Male Wistar rats experienced maternal deprivation (MD) on postnatal days 1–14 for three hours per day. Afterward, they were not interrupted until postnatal day 70, during which the extracellular activities of LPGi neurons were recorded in anesthetized animals at baseline and in response to acute morphine.
Results : In both MD and control groups, acute administration of morphine elicited heterogeneous responses in the neurons of the LPGi (excitatory, inhibitory, and neutral effects). At baseline recordings, the variability of interspike intervals within the LPGi neurons was reduced in both the inhibitory and excitatory responses among MD group. Furthermore, the degree of morphine-induced inhibition of neuronal discharge was significantly lower in the deprived animals compared to the control group.
Conclusion : The results indicate that early life stress, specifically in the form of maternal deprivation, leads to long-term changes in the responsiveness of LPGi neurons to acute morphine administration. Notably, this includes a reduction in morphine-induced inhibitory responses. Consequently, maternal deprivation may alter susceptibility to opiate abuse in adulthood partly via the LPGi, establishing a link between neonatal MD and increased risk of later opioid addiction. Additionally, the potential correlation between these neuronal subpopulations and behavioral manifestations during withdrawal can be taken into account. Further studies are necessary to investigate this hypothesis at a mechanistic level.
Keywords : Early life stress; Maternal deprivation; Lateral paragigantocellularis nucleus; Extracellular single unit recording; Opioid

Count: 58
Abstract ID: 870
subject: Other: Other
Presentation Type: Oral

A Practical Comparison of Resting-State fMRI Sequences: EPI Routine vs. SMS

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Background and Aim : Resting-state functional MRI (rs-fMRI) enables the study of spontaneous brain activity and intrinsic connectivity without task performance. The conventional Echo Planar Imaging (EPI) sequence is widely used but limited by long repetition times (TR), which reduce temporal resolution. Simultaneous Multi-Slice (SMS) imaging has been introduced to accelerate acquisition by capturing multiple slices simultaneously, improving temporal sampling and sensitivity to dynamic brain activity. However, SMS may increase motion and physiological noise. This study aims to practically compare EPI Routine and SMS sequences in rs-fMRI to evaluate differences in image quality, signal stability, and functional connectivity outcomes.

Methods : 28 healthy adult volunteers (12 males and 16 females, aged 20–45 years) participated in this study after providing written informed consent. All participants were right-handed and had no history of neurological or psychiatric disorders. The study protocol was approved by the institutional ethics committee in accordance with the Declaration of Helsinki. MRI scans were performed on a 3T Siemens Prisma scanner equipped with a 64-channel head coil. Each subject underwent two resting-state fMRI scans in a single session: one using the conventional Echo Planar Imaging (EPI Routine) sequence and another using the Simultaneous Multi-Slice (SMS) sequence. The order of sequences was counterbalanced to minimize bias. For the EPI Routine sequence, the parameters were: TR = 2200 ms, TE = 30 ms, flip angle = 90°, voxel size = 3.0 × 3.0 × 3.0 mm^3, and 40 slices. For the SMS sequence: TR = 1000ms, TE = 30 ms, flip angle = 90, voxel size = 3.0 × 3.0 × 3.0 mm^3, 40 slices, and SMS factor =4. Participants were instructed to keep their eyes closed, remain still, and stay awake during the scans. Preprocessing was performed using SPM12 and the CONN toolbox in MATLAB. Steps included slice timing correction, realignment, normalization to MNI space, spatial smoothing (6 mm FWHM), and band-pass filtering (0.008–0.09 Hz). Signal-to-noise ratio (SNR), temporal SNR (tSNR), and functional connectivity (FC) were computed and compared between EPI and SMS datasets using paired t-tests with a significance threshold of $p < 0.05$ (FDR corrected).

Results : SMS sequences showed higher temporal resolution and significantly improved temporal SNR compared to EPI Routine, while conventional EPI maintained slightly higher spatial SNR. Functional connectivity analysis revealed that both sequences reliably identified major resting-state networks, including the default mode, visual, and sensorimotor networks. SMS data demonstrated stronger correlation coefficients and increased network detectability, particularly in fast-changing connectivity patterns. However, SMS was more sensitive to motion artifacts, requiring additional preprocessing. Overall, SMS improved temporal sampling and dynamic connectivity measures, whereas EPI Routine provided more stable and robust signal across subjects.

Conclusion : SMS sequences enhance temporal resolution and dynamic connectivity detection in resting-state fMRI, offering improved temporal SNR and network sensitivity. EPI Routine provides more stable spatial signal and robustness against motion. Selection between sequences should balance temporal efficiency and signal stability, depending on study goals and subject characteristics.

Keywords : resting-state fMRI, EPI, SMS, connectivity, SNR, tSNR

Count: 59
Abstract ID: 725
subject: Other: Other
Presentation Type: Poster

Receptive Field Organization in Macaque vlPFC Neurons

Submission Author: Yasamin Abolfathi

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Background and Aim : Receptive fields (RFs) define the region of visual space where stimuli influence neuronal activity. While RFs in ventral and dorsal visual areas are well studied, their organization in the ventrolateral prefrontal cortex (vlPFC)—a region involved in visual processing, decision-making, and goal-directed behavior—remains poorly understood. Unlike early visual areas with small, contralateral RFs arranged topographically, higher-order regions like vlPFC are expected to exhibit larger and more complex RFs that support flexible perception. However, previous studies have focused on face-selective patches and limited samples, leaving broader RF properties unexplored. To address this, we recorded extracellular activity from 132 vlPFC neurons in macaque monkeys during passive viewing of a high-resolution random-dot stimulus. RFs were mapped and categorized into uniform, full-field, and nonuniform types. Over 85% of neurons showed large, multi-peaked RFs extending into peripheral visual space. These results reveal rich spatial diversity in vlPFC and suggest its role in integrating distributed visual input beyond classical sensory boundaries.

Methods : We recorded single-unit activity from 132 vlPFC neurons in two macaque monkeys during passive viewing of random-dot stimuli. Electrodes were guided by MRI-based stereotaxic surgery and signals were recorded extracellularly. Receptive fields (RFs) were mapped using spike-triggered averaging across a 13 × 25 grid spanning 26° × 50° of visual space. RFs were normalized, smoothed, and classified into uniform, nonuniform, and full-field types. Peak locations were analyzed for eccentricity and hemifield bias. Reliability was assessed via split-half correlations against a shuffled baseline. This method revealed diverse RF structures and enabled precise characterization of spatial tuning in vlPFC neurons.

Results : Receptive fields (RFs) in the ventrolateral prefrontal cortex (vlPFC) showed striking diversity in structure, size, and spatial distribution across 132 recorded neurons. Most neurons exhibited large, multi-peaked RFs that extended into peripheral visual space. Based on spatial structure, neurons were classified into three categories: non-uniform (85.6%), full-field (7.6%), and uniform (6.8%). RF sizes ranged from approximately 2° to 36°, with a population median of 22.1°. Full-field RFs were the largest, while uniform RFs were compact and rare. Spatial analysis of 181 peak locations revealed a near-balanced representation across contralateral and ipsilateral hemifields, with minimal central bias. Peak eccentricities had a median of 15°, reinforcing the peripheral emphasis of vlPFC visual coding. Reliability analysis confirmed stable RF structure in 57% of neurons, supporting the robustness of the mapping approach.

Conclusion : This study shows that vlPFC neurons encode visual space through large, diverse, and often peripheral receptive fields. The dominance of nonuniform and multi-peaked RFs, along with minimal foveal representation, suggests that vlPFC supports coarse, distributed visual processing rather than precise, central recognition. These properties resemble dorsal stream organization and may enable rapid detection of salient features across the visual field. The spatial heterogeneity observed here points to a flexible architecture suited for integrating input from multiple sources. Together, these findings position vlPFC as a dynamic hub for visual cognition, capable of linking perception with higher-order behavioral demands.

Keywords : Receptive Field, vlPFC, STA method, heterogeneity , peripheral

Count: 60
Abstract ID: 738
subject: Other: Other
Presentation Type: Poster

Comparison of the Representation of Different Spatial Frequencies in the IT and vlPFC areas

Submission Author: Yasamin Abolfathi

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Background and Aim : This study investigates how spatial frequency information is represented in two cortical regions involved in visual processing: the ventrolateral prefrontal cortex (vlPFC) and the inferior temporal cortex (IT). Using extracellular recordings from two macaque monkeys during passive viewing, neuronal responses to images filtered at different spatial frequency levels were analyzed. vlPFC neurons responded more rapidly to low spatial frequency (LSF) content, while IT showed stronger overall performance in object classification. Support vector machine analyses confirmed that spatial frequency content could be reliably decoded from neural activity. While IT consistently relied on both LSF and high spatial frequency (HSF) information, vlPFC sensitivity varied depending on object category. These findings reveal distinct encoding strategies across regions, with vlPFC tuned for rapid, coarse input and IT supporting detailed, category-specific recognition. The results offer new insights into the neural basis of visual perception and may inform future models of cortical processing and artificial vision systems.

Methods : This study recorded neural activity from vlPFC and IT in two rhesus monkeys during passive viewing of grayscale images filtered by spatial frequency. Each frequency band (low, high, full) included 27 stimuli across four categories. Stimuli were shown for 50 ms under strict fixation, and spike data were sorted using threshold-based detection. Responsive neurons were selected, and population and single-unit responses were analyzed over time. Support

vector machine classifiers assessed decoding performance, and temporal dynamics were extracted using Gaussian smoothing. Results revealed distinct encoding profiles across regions, highlighting how vlPFC and IT process coarse versus detailed visual input.

Results : Recordings from two rhesus monkeys showed distinct spatial frequency processing in the inferior temporal (IT) and ventrolateral prefrontal (vlPFC) cortices. Single-neuron analyses revealed that vlPFC neurons responded earlier to low spatial frequency (LSF) stimuli, while IT neurons reached peak activity faster for high spatial frequency (HSF) inputs. Firing rates were higher for HSF in IT and lower in vlPFC. General linear model results indicated that spatial frequency explained more neural variance than categorical factors, with vlPFC showing stronger frequency-related coefficients and IT showing finer structural sensitivity. Frequency effects appeared around 70 ms and persisted to about 170 ms after stimulus onset. Support Vector Machine decoding demonstrated accurate classification of frequency content in both regions, but with differing temporal profiles: vlPFC showed earlier, shorter decoding of LSF, whereas IT displayed later, longer decoding for HSF, confirming distinct temporal dynamics in frequency representation.

Conclusion : This study shows that vlPFC and IT process spatial frequency content differently. IT consistently uses both low and high spatial frequencies across categories, while vlPFC favors low frequencies and shows delayed, weaker responses to high ones. Temporal analyses revealed that vlPFC reaches peak performance for low-frequency input faster than IT, supporting its role in early, coarse-level interpretation. Decoding results confirmed that frequency content strongly influences neural responses, with vlPFC's contribution varying by category. These findings suggest that vlPFC plays an active role in object recognition, using spatial frequency information flexibly and in coordination with IT.

Keywords : spatial frequency, object recognition, coarse to fine, vlPFC, IT

Count: 61
Abstract ID: 783
subject: Other: Other
Presentation Type: Poster

Gender-based ADHD prevalence among Afghan Adults: A Survey Study

Submission Author: Massoma Ramazani

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Background and Aim : Background: Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that affects brain activity and generally remains across the lifespan. The purpose of this study is to explore the prevalence of ADHD among adults male and female gender in Afghanistan.

Methods : Methods: This online-based questionnaire research distributed among Afghan people through online platforms. This research was conducted in a two-month period and is measured by a 5-point Likert scale. A score of 18-24 for all 6 included questions of ADHD Self-Report Scale (ASRSv1.1) was the indicator of positive screening ADHD, while a negative screening ADHD score is indicated by 0-17. Descriptive and non-parametric statistical analyses were employed to find out the final relevance between the ADHD and gender.

Results : Results: Among the total of 196 participants, 175 met the inclusion criteria, comprising 100 women and 75 men. The overall prevalence of ADHD was 16.6%, slightly higher among Afghan women (9.47%) than men (7.12%), although this difference was not statistically significant according to Kruskal–Wallis and Chi-square analyses. The results also demonstrated significant associations between ADHD and a history of mental disorders, use of psychiatric medications, and psychiatric consultation.

Conclusion : Conclusion: These findings revealed that there is no significant difference in ADHD prevalence among male and female genders. Furthermore, future studies with larger samples are essential to identify the barriers to diagnosis and cure of ADHD among Afghan adults.

Keywords : Keywords: ADHD, Afghanistan, Prevalence, Gender, Adults, Questionnaire

Count: 62
Abstract ID: 580
subject: Other: Other
Presentation Type: Poster

Psilocybin for Psychological Distress in Patients With Cancer: A Systematic Review and Meta-analysis

Submission Author: Jalal KazemiGhanbarAbadi

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Conclusion : Objective: To evaluate the efficacy and safety of psilocybin for alleviating psychological distress in adults with cancer, synthesizing randomized and non-randomized clinical evidence across depression, anxiety, spiritual well-being, and quality of life. Methods: Following PRISMA 2020, we searched Scopus, PsycINFO, PubMed, Cochrane, CINAHL Complete, and Web of Science through November 2024, and screened citations and trial registries. Eligible studies enrolled adults (≥18 years) with cancer who received psilocybin (as the primary intervention) in randomized controlled trials (RCTs), quasi-experimental designs, or single-arm trials. Primary outcomes were depression and anxiety (e.g., Beck Depression Inventory [BDI]; Hospital Anxiety and Depression Scale—Depression/Anxiety subscales [HADS-D/HADS-A]; State-Trait Anxiety Inventory—State/Trait [STAI-S/STAI-T]); secondary outcomes included spiritual well-being (Functional Assessment of Chronic Illness Therapy—Spiritual Well-Being [FACIT-Sp]) and quality of life (QOL). Standardized mean differences (SMDs) were pooled with random-effects models; heterogeneity (I^2), funnel

plots/Egger's test, and risk of bias (RoB-2 for RCTs; ROBINS-I for non-randomized studies) were assessed. The protocol was registered (PROSPERO CRD42023460787). Results: Eight studies met inclusion criteria (United States; 2011–2024), spanning three double-blind crossover RCTs, two non-randomized trials, and long-term follow-ups of prior cohorts (sample sizes 11–51). In placebo-controlled meta-analyses (two RCTs; n=79), psilocybin produced large acute reductions in depressive symptoms (BDI SMD −2.87, 95% CI −3.99 to −1.76; HADS-D SMD −2.97, −3.60 to −2.33) and state/trait anxiety (STAI-S SMD −1.84, −2.36 to −1.32; STAI-T SMD −4.70, −8.19 to −1.21). HADS-A was not significant, with high heterogeneity and wide confidence intervals. Effects on demoralization favored psilocybin (SMD 0.89, 0.44 to 1.35), whereas pooled QOL was not significant in this subset. Pre–post single-arm analyses showed consistent short-term (2–5 weeks) improvements: BDI −1.17 (−1.73 to −0.61), HADS-D −1.58 (−2.07 to −1.08), HADS-A −1.99 (−2.52 to −0.146), STAI-S −1.17 (−1.50 to −0.84), and STAI-T −1.34 (−1.90 to −0.78); Hamilton Anxiety Rating Scale (HAM-A) also improved (SMD −2.20) with high heterogeneity. Spiritual well-being improved (FACIT-Sp SMD 2.58), while short-term QOL estimates were imprecise and not significant. At six months, benefits persisted for depression (BDI −2.60, p=0.04; HADS-D −3.56, p=0.01) and anxiety (HADS-A −2.59, p<0.001); QOL (SMD 1.73) and FACIT-Sp (SMD 3.02) also improved, whereas STAI outcomes were not significant. Conclusions: Across RCT and non-randomized evidence, psilocybin—typically embedded within psychotherapeutic frameworks—yields large, clinically meaningful reductions in depression and anxiety in patients with cancer, with additional gains in spiritual well-being and quality of life that may endure to six months. Interpretation is tempered by small, US-only samples, variability in dosing/set-and-setting, co-intervention with psychotherapy, and heterogeneity across measures. Larger, multi-site RCTs with standardized protocols, active-comparator arms, and prespecified assessments of set/setting and long-term safety are needed to define optimal use and generalizability.

Keywords : Psilocybin; Cancer; Anxiety; Depression; Spiritual well-being; Quality of life; Meta-analysis.

Count: 63
Abstract ID: 244
subject: Other: Other
Presentation Type: Poster

Advanced Intraoperative Electrophysiological Neuromarkers for Cranial Nerve preservation during cerebellopontine angle surgery

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Background and Aim : Cranial nerve injury during cerebellopontine angle (CPA) tumor surgery represents a serious complication for both patients and surgeons. The incidence of cranial nerve injury during CPA tumor surgery is significantly dependent on tumor size and surgical approach. Nowadays, continuous Intraoperative neuromonitoring (IONM) is a widely used technology to minimize the risk of motor cranial nerve deficits and improve surgical outcomes during CPA surgery. Nevertheless, the routine use of intraoperative facial nerve monitoring has not eliminated the risk of injury, which continues to be a concerning complication. Awareness of specific neurophysiological markers can enhance the accuracy of detecting functional changes and ultimately reduce the risk of nerve injury. This study aimed to provide a detailed analysis of intraoperative EMG phenomena with respect to their surgical significance

Methods : A systematic literature review and retrospective clinical case series analysis of CPA tumor surgeries informed the design of this study. The focus was on facial nerve monitoring using facial motor evoked potentials (FMEP) and predictive electrophysiological parameters to highlight all aspects of facial nerve (FN) monitoring in CPA and skull base tumor surgery

Results : Three distinct classes of neuromarkers were identified: 1.Mean latency of FMEP in patients with a good outcome was approximately <7.2 ms. In patients with poor postoperative outcomes, latencies were significantly prolonged, in some cases >10 ms. Moreover, FMEP latencies increased with tumor extension. Patients with larger tumors showed latencies of approximately >13.5 ms. Furthermore, Patients with good outcomes (HB I–II) exhibited a mean FMEP amplitude of approximately 1.0 mV, whereas those with poorer outcomes demonstrated significantly smaller initial amplitudes, typically ranging from 0.4 to 0.6 mV. Thus, amplitude reduction during surgery was strongly predictive of poor postoperative

function; patients with preserved amplitudes around 0.8 mV had HB I–II, while reductions to 0.2–0.3 mV predicted grades IV–VI. 2. patterns of EMG activity EMG waveform patterns can be classified as spikes, bursts, and A-, B-, and C-trains. A-train, characterized by regular sinusoidal discharges with symmetrical high-frequency activity (60–210 Hz), low amplitude (typically <500 µV), and long duration (milliseconds to seconds, showed a strong association with postoperative facial palsy and is regarded as the most reliable predictive marker, with a sensitivity of 86% and a specificity of 89%. 'A-train salvo,' a variant consisting of clustered bursts of A-trains, further increases prognostic significance. 3. Stimulation Safety and Efficacy Parameters: The mean minimal electrical stimulation threshold of the facial nerve was 0.21 mA (range: 0.2–0.3 mA), indicating that very low currents are sufficient to elicit a detectable muscular response. A linear correlation was observed between stimulus intensity and response amplitude for intensities below 0.4 mA, whereas response. These findings suggest that stimulation intensities up to 0.4 mA represent the optimal range, providing maximal responses within a safe margin, while higher currents do not produce additional diagnostic value

Conclusion : This study demonstrates that advanced intraoperative neuromarkers play a crucial role in optimizing cranial nerve preservation during cerebellopontine angle tumor surgery. Among the parameters assessed, FMEP latency, amplitude, and A-train pattern analysis showed the highest prognostic value. Stimulation thresholds up to 0.4 mA provided maximal safe responses. Collectively, these findings indicate that attention to intraoperative neuromarkers during neuromonitoring is the most reliable strategy for improving facial nerve surgical outcomes in CPA tumor surgery

Keywords : Intraoperative neuromonitoring, Cranial nerve preservation, Facial motor evoked potentials ,Neurophysiological biomarkers

Count: 64
Abstract ID: 667
subject: Other: Other
Presentation Type: Poster

Synergistic Effects of Temozolomide and Quercetin in Glioblastoma Treatment

Submission Author: Fatemeh Rasouli

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Background and Aim : Glioblastoma multiforme (GBM) is the most widespread and lethal form of glioma, with a median survival of approximately 15 months. Although the combination therapy of radiation and temozolomide (TMZ) has become standard, most patients will still eventually recur as a result of TMZ resistance; thus, novel targeted drug therapies may contribute to better treatment options for such patients. Quercetin, as a bioflavonoid widely found in fruits and vegetables, has shown significant antitumor activity through various molecular pathways. Here, we assessed the effects of combination therapy of quercetin and TMZ in GBM, which can be a promising treatment to sensitize GBM cells.

Methods : In vitro assays were done on U87MG and U251MG GBM cell lines following TMZ, quercetin, or their combination treatment. The MTT assay was performed to measure cell viability, and apoptosis was evaluated using flow cytometry. Western blot analysis was used to analyze the heat shock protein 27 (Hsp27) expression, which is related to chemoresistance.

Results : We found that combination therapy of quercetin and TMZ significantly inhibited GBM cell viability by 65% when compared to the control group, demonstrating an enhanced cytotoxic effect compared to either agent alone ($p < 0.01$), correlating with increased apoptosis as compared to monotherapies ($p < 0.001$). Western blot analysis showed that co-treatment with quercetin and TMZ significantly reduced Hsp27 expression as compared to TMZ alone ($p < 0.001$).

Conclusion : Quercetin improved TMZ efficacy in GBM cells by inhibiting proliferation and elevating cancer cell apoptosis along with reduced Hsp27 expression, suggesting it as a potential candidate in the therapeutic field of GBM.

Keywords : glioblastoma; quercetin; temozolomide; apoptosis; cell proliferation; heat shock protein 27

Count: 65
Abstract ID: 526
subject: Other: Other
Presentation Type: Poster

Non-pharmacological therapeutic paradigms in stress-induced depression: from novel therapeutic perspective with focus on cell-based strategies

Submission Author: Maryam Azarfarin

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Conclusion : Major depressive disorder (MDD) is considered a psychiatric disorder and have a relationship with stressful events. Although the common therapeutic approaches against MDD are diverse, a large number of patients do not present an adequate response to antidepressant treatments. On the other hand, effective non-pharmacological treatments for MDD and their tolerability are addressed. Several affective treatments for MDD are used but non-pharmacological strategies for decreasing the common depression-related drugs side effects have been focused recently. However, the potential of extracellular vesicles (EVs) derived from mesenchymal stem cells (MSCs), microRNAs (miRNAs) as cell-based therapeutic paradigms, besides other non-pharmacological strategies including mitochondrial transfer, plasma, transcranial direct current stimulation (tDCS), transcranial magnetic stimulation (TMS), and exercise therapy needs to further study. This review explores the therapeutic potential of cell-based therapeutic non-pharmacological paradigms for MDD treatment. In addition, plasma therapy, mitotherapy, and exercise therapy in several in vitro and in vivo conditions in experimental disease models along with tDCS and TMS will be discussed as novel non-pharmacological promising therapeutic approaches.

Keywords : depression; stress; therapy; cell-based strategies; non-pharmacological paradigms

Count: 66
Abstract ID: 464
subject: Other: Other
Presentation Type: Poster

Nervous system involvement following gut microbiota depletion

Submission Author: SayyedAlireza Talaei

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Conclusion : Bidirectional communication between the central nervous system (CNS) and the gastrointestinal tract has been the focus of research over the last decade. The gut microbiota (GM) facilitates this communication via neuronal, metabolic, immunological, and endocrine pathways. This interaction has a significant impact on gut and brain health. Germ-free (GF) animals are effective models for investigating host-microbiota interactions and provide deeper insights into the physiological and psychological effects of the microbiota. Additionally, to assess the role of commensal bacteria in certain diseases or their influence on treatment failures, it is optimal to use GF animals that lack any bacteria compared to wild-type animals that possess a specific microbiota. Numerous studies indicate that the absence of a normal GM can lead to significant disturbances in CNS neurotransmission, neurogenesis, neurodevelopment, neuronal activity, stress response, cognitive function, learning, and memory. The purpose of this review is to consider the research describing these changes, both at the molecular and cellular levels and in relation to behavioral outcomes.

Keywords : Germ-Free; Microbiota; Brain-Gut Axis; Neurotransmitters; Behavior

Count: 67
Abstract ID: 830
subject: Other: Other
Presentation Type: Poster

Role of dmPAG Angiotensin II Type 1 Receptors in Cardiovascular Regulation in Rats

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Background and Aim : cardiovascular reactions of Defensive and aggressive behaviors are partially mediated by dorsomedial periaqueductal gray (dmPAG), a midbrain structure. Angiotensin II (Ang II) and its AT_1 receptors are found to be located in the dmPAG, but its circulatory action remains unknown. The purpose of present study was to evaluate central and peripheral cardiovascular effect of Ang II in it.

Methods : 72 male rats were separated and placed into experimental groups receiving microinjections of saline, Ang II, Losartan (Los; an AT_1 receptor antagonist) alone, or Los before Ang II into the dmPAG. Hexamethonium (Hex; a ganglionic blocker), Atropine (a muscarinic antagonist), Propranolol (a β-adrenergic blocker), and Terazosin (an α_1-adrenergic blocker) were given by intravenous injection before Ang II microinjection. Systolic blood pressure (SBP), mean arterial pressure (MAP), and heart rate (HR) were continually monitored by a PowerLab system coonected to a femoral artery cannula.

Results : Changes (Δ) in cardiovascular parameters were calculated and statistically analyzed. Microinjection of Ang II into the dmPAG significantly increased ΔSBP, ΔMAP, and ΔHR, effects that were completely abolished by Losartan. The circulatory responses to Ang II were completely prevented by Hexamethonium, while Atropine produced no significant effects. Both Terazosin and Propranolol attenuated Ang II-induced responses, showing that α_1-adrenergic receptors primarily regulate blood pressure, whereas β-adrenergic receptors modulate heart rate.

Conclusion : In conclusion Ang II in the dmPAG produce excitatory cardiovascular effects through AT_1 receptors. Also, the peripheral cardiovascular effect of AngII mediated by both α_1- and β-adrenergic receptors.

Keywords : dorsomedial PAG; Blood pressure; Angiotensin II;Hexamethonium; Losartan; Propranolol

Count: 68
Abstract ID: 696
subject: Other: Other
Presentation Type: Poster

Rate of cerebral venous thrombosis with the use of oral contraceptive pills during fasting periods: a systematic review and meta-analysis

Submission Author: Rasa Beheshti

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Conclusion : Background and Aims: Cerebral venous thrombosis (CVT) is an uncommon cerebrovascular disorder that can lead to significant morbidity and mortality if not promptly diagnosed and treated. A history of consuming oral contraceptive pills (OCP) and dehydration are among the established risk factors for CVT, but the significance of simultaneous dehydration as a risk factor along with OCP usage is still a matter of debate. We aimed to conduct this systematic review and meta-analysis to assess the rate of CVT in individuals using OCP during fasting periods. Methods: English written studies which assessed CVT patients who take OCP with and without fasting were included. PubMed, Medline via Ovid, Embase, Cinahl, Cochrane library, Web of Sciences, and Scopus databases were systematically searched from the inception up to July 2024. The Joanna Briggs Institute critical appraisal checklist was applied to assess the risk of bias of the included studies. Rate of CVT with OCP consumption during fasting periods was pooled in the meta-analysis. Comprehensive Meta-Analysis software version 3 was used to perform the Meta-Analysis. Results: A total of 5 studies involving 248 CVT patients with OCP consumption were included. All included studies were cross-sectional and conducted in Mediterranean centers, focusing on the relationship between fasting and religious ceremonies. The meta-analysis revealed that 47% of CVT survivors had taken OCP pills during the fasting periods. Conclusions: Fasting, characterized as a feature of dehydration, was associated with a significant proportion of CVT patients who simultaneously used OCP agents. There is a need for more comprehensive and prospective studies to evaluate the concomitant use of OCP agents in the context of quantified dehydration.

Keywords : Cerebral venous thrombosis; Fasting; Systematic Review; Meta-analysis

Count: 69
Abstract ID: 913
subject: Other: Other
Presentation Type: Poster

The gene expression level of orexin receptor type 1 of the basolateral amygdala is not changed by social hierarchy during acute thermal pain induction in male rat

Submission Author: Sharareh Khavandkari

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Background and Aim : Social hierarchy is one of the most influential social structures. An individual's social rank in social hierarchy has vital consequences and plays a key role in inducing stress response. Additionally, different types of chronic pain are considered stress. Pain is a complex phenomenon that results from the integration of sensory information with cognitive and affective processes. Human imaging studies have shown the role of the prefrontal cortex and amygdala network in integrating the cognitive functions with pain information. The medial prefrontal cortex projections to the basolateral amygdala (BLA) are involved in the integration and preservation of the nociceptive and social status information. On the other hand, the BLA receives orexinergic projections from the lateral hypothalamus. The orexinergic system is involved in the control of stress, anxiety, social hierarchy, and pain modulation. Also, orexin plays a crucial role in the social failure stress-induced behavioural vulnerability. The orexin effects are mediated through the orexin receptor type 1 activation, and the orexin receptor type 1 (Orx1R) gene is expressed in the amygdala and hippocampus. Therefore, the present study was designed to investigate the role of the Orx1 receptors of the BLA in modulating acute thermal pain in adult male rats with different social hierarchies.

Methods : In this study, ninety male Wistar rats (200 to 270 grams) were divided into two main groups termed the intact animal and the social hierarchy groups. In the intact animal group, the social hierarchy status was not determined. However, the social hierarchy group was divided into two groups (dominant and subordinate animals) after determining of social hierarchy status by the tube test. Then, bilateral cannulation of the BLA was stereotaxically performed. After a recovery period, DMSO and SB334867 (the Orx1 receptor antagonist) were injected into the BLA nuclei, and 5 minutes later, the tail flick and hot plate tests were

accomplished. At the end of behavioural tests, the rats were decapitated, and the BLA was identified and removed. These histological samples were kept in a -80 ℃ freezer until the molecular test time. In this research, the gene expression level of the BLA orexin receptor type 1 was evaluated using the quantitative RT-PCR method. Data were analysed using repeated measures of Two-Way ANOVA by Prism software.

Results : The results of the current study showed that after intra-BLA injection of DMSO in the dominant and subordinate animals and induction of acute thermal pain, the expression level of the Orx1R gene in subordinate animals was significantly higher ($P < 0.05$) than that in dominant animals.

Conclusion : The findings of the present study showed that the intra-BLA injection of orexin receptor type 1 antagonist (SB334867) in intact, dominant and subordinate animals induces hyperalgesia. However, the alterations of the dominant and subordinate animal responses to thermal pain seem not to be mediated by changing the gene expression level of the BLA orexin receptor type 1.

Keywords : Social hierarchy; Basolateral Amygdala; Orexin 1 receptor; Pain modulation

Count: 70
Abstract ID: 752
subject: Other: Other
Presentation Type: Poster

The role of gut microbial metabolites in regulating gene expression in neurodevelopmental and neurodegenerative disorders: A systematic review of in vitro, in vivo, and clinical studies

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Conclusion : Introduction The gut-brain microbiota axis refers to a complex, bidirectional network between the gut's resident microorganisms and the central nervous system. This communication occurs through various pathways, including the nervous, immune, hormonal, and metabolic systems, and causes the gut and brain to constantly interact with each other. In turn, the brain can influence bowel movements, digestive secretions, and even the composition of the microbiota through neural and hormonal pathways. Metabolites produced by the gut microbiome, such as short-chain fatty acids (SCFAs), tryptophan compounds, TMAO, and some bile acids, can affect gene expression and epigenetic changes in the brain, and any imbalance in the gut-brain axis due to changes in the levels of SCFAs and LPS produced by the gut microbiota can be one of the factors aggravating the symptoms of autism or, on the other hand, changes in the integrity of neurodevelopment, which will ultimately lead to the occurrence of neurodegenerative disorders such as Alzheimer's. Despite the significant increase in studies on the gut-brain axis, the available evidence is still scattered, heterogeneous, and sometimes contradictory. This has made it difficult to obtain a comprehensive understanding of the mechanism of this axis and its role in brain function. Therefore, this systematic review was conducted to collect, critically evaluate, and synthesize the results of previous research so that the findings may contribute to developing therapeutic approaches based on the gut–brain microbiota axis. Materials and methods A systematic search was performed in PubMed, PMC, and preprint databases. Keywords included a combination of gut microbiome, microbial metabolites, SCFA, butyrate, tryptophan metabolites, TMAO, bile acids as well as gene expression, transcriptome, epigenetic. The study time frame was from January 2019 to October 4, 2025. Only original studies that provided experimental data on microbial metabolites and gene expression or epigenetic changes, including in vitro, in vivo and human models, were included in the analysis. Extracted results included model type, metabolites, molecular changes and key findings. Results: The total number of studies included in the analysis was 23 articles, including cellular, animal, and human studies. Cellular (in vitro)

studies showed that SCFAs, especially butyrate and propionate, were able to induce epigenetic changes (such as histone acetylation and crotonylation) and gene expression in neurons, suggesting that metabolites can directly regulate gene function. Studies in animal models (in vivo) showed that transferring the microbiota of children with autism to mice lacking the microbiome could lead to autism-like behaviors and alterations in serotonin and tryptophan pathways in the brain. Finally, human studies, cohort studies, and multi-omics analyses showed associations between gut microbial composition and metabolites (SCFAs, tryptophan compounds, TMAO, and bile acids) with molecular and cognitive/brain imaging markers. However, the results were not homogeneous between studies, and genetic factors, diet, and age varied the effects. Conclusion: Evidence suggests that gut microbial metabolites can influence gene expression in brain neurons and contribute to autism-related behaviors or Alzheimer's pathology. However, in humans, there is still insufficient evidence to prove direct causality, and longitudinal and interventional studies with larger samples and standardization of methods are necessary.

Keywords : Gut microbiome, microbial metabolites, SCFA, butyrate, tryptophan metabolites, TMAO, gene expression, epigenetics, autism, Alzheimer's

Count: 71
Abstract ID: 766
subject: Other: Other
Presentation Type: Poster

A threshold number for the Complexity Phase Transition: Study of cell populations with ~100,000 cells in Theoretical Neuroscience

Submission Author: Gooya Bozorgi

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Background and Aim : Although the numerical determination of the complexity phase threshold seems ambiguous and context-dependent, this study suggests that population size transitions above certain levels are important in determining group behavior and complexity phase transitions. To this end, the analysis of population sizes of ~100,000, especially in cellular populations, supports this theory.

Methods : The method of this study is a comparative analysis of population behavioral patterns in demography and biology. In addition, the populations of nerve cells in the brain of the fruit fly and the spider Portia have been studied as two case studies in the field of neuroscience.

Results : The findings show that although population growth and the corresponding level of complexity in behavioral patterns exhibit a spectral shape, when the population reaches certain values, group behaviors enter a new phase of complexity. The behavioral complexity of the fruit fly and the spider Portia neuron population, proportional to the desired number (~100,000), supports the thesis of this study as a miniature brain.

Conclusion : Studying the magnitude of different population sizes and changes in behavioral patterns helps to scale the degree of complexity across multiple phases. This can be useful in understanding behavioral patterns and the link between population size and degree of complexity.

Keywords : Theoretical neuroscience; Cell population; Group behaviors; Miniature brain; Complexity phase; Number ~100,000.

Count: 72
Abstract ID: 435
subject: Other: Other
Presentation Type: Poster

Study of antibiotic resistance profile among nosocomial Klebsiella pneumoniae infections in the last 5 years in hospitals under the auspices of Abadan University of Medical Sciences

Submission Author: Forouzan Absalan

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Background and Aim : The rapid and widespread emergence of bacteria resistant to a wide range of antibiotics is occurring worldwide today, and this phenomenon has severely compromised the effectiveness of antibiotics. Klebsiella pneumoniae is a gram-negative bacteria that shows a very high resistance to various antibiotics every year, for this reason, in this study, we investigated the antibiotic profile of Klebsiella pneumoniae in the last 5 years in Abadan.

Methods : This study was a descriptive-analytical epidemiological study based on hospital-clinic information. This study used data from all reported cases of nosocomial infections caused by Klebsiella pneumoniae during the last 5 years, ranging from 1398 to 1404. In this study, the results of the antibiogram test of all hospitals under the coverage of Abadan University of Medical Sciences (Shahid Beheshti Hospital, Abadan, Taleghani Hospital, Hazrat Vali Asr Hospital (AJ) Khorramshahr, etc.) between 2019 and 2025 were used, and statistical analysis was performed with SPSS24 software.

Results : This study was conducted on 408 people. Most of the people were between 60 and 70 years old. 238 of the people studied were men and 170 were women. Every year, the resistance rate of Klebsiella pneumoniae to various antibiotics is higher. In 2024, 31 cases of cephalosporin antibiotics were reported, 8 cases of fluoroquinolones, 8 cases of aminoglycosides, 8 cases of beta-lactamase inhibitors, 10 cases of carbapenems, and only one case of colistin.

Conclusion : Hospital infections are important hospital problems that need to be followed up. Among hospital infections, infections caused by antibiotic resistance caused by Klebsiella pneumoniae bacteria have increased significantly annually. This study showed that the ICU, internal medicine, surgical ICU, and internal ICU departments have the highest rates of involvement. Also, patients who had blood and urine cultures and UC were more involved, and this disease showed high resistance to carbapenems and cephalosporins.

Keywords : Klebsiella pneumoniae, antibiotic resistance, hospital infection.

Count: 73
Abstract ID: 103
subject: Other: Other
Presentation Type: Poster

Measuring the affinity of 5 specific fentanyl analogs for the μ-opioid receptor and revealing their interaction with the TM6/TM7 motif of the μ-opioid receptor using molecular docking methods

Submission Author: Forouzan Absalan

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Background and Aim : The μ-opioid receptor (mOR) is a major pharmacological target for analgesia, but conventional opioids such as fentanyl and morphine induce β-arrestin recruitment that contributes to respiratory depression and other adverse effects. Designing G protein-biased agonists that preserve analgesia while minimizing β-arrestin signaling represents an important strategy for next-generation opioid development.

Methods : Fentanyl and seven analogs—carfentanil, ohmefentanyl, lofentanil, sufentanil, remifentanil, FBD3, and PZM21—were evaluated using molecular docking, ligand efficiency calculations, pharmacokinetic modeling, and previously reported in vitro signaling data. Docking against the human mOR (PDB ID: 8EF5) was performed with InstaDock using QuickVina-W scoring, and interaction maps were generated to compare binding to transmembrane helices TM2/3 and TM6/7. Absorption, distribution, metabolism, and excretion (ADME) properties were predicted in silico, including solubility, blood–brain barrier (BBB) penetration, P-glycoprotein (P-gp) substrate status, cytochrome P450 inhibition, and synthetic accessibility.

Results : Docking studies revealed binding free energies ranging from −6.5 to −8.9 kcal.mol-1. Fentanyl showed the highest predicted affinity (ΔG = −8.9 kcal.mol-1, pKi = 6.53), followed by carfentanil (−8.8 kcal/mol), ohmefentanyl and lofentanil (both −8.6 kcal/mol). Lower-affinity ligands included FBD3 (−7.1 kcal/mol) and PZM21 (−6.5 kcal/mol). Two-dimensional interaction analysis indicated a conserved orthosteric binding pocket with ligand-specific hydrophobic contacts. in vitro functional data confirmed distinct signaling profiles: fentanyl

and carfentanil induced balanced G protein and β-arrestin 2 activation, while FBD3 and PZM21 acted as G protein-biased agonists with minimal β-arrestin recruitment. All compounds displayed high predicted gastrointestinal absorption and favorable druglikeness, with a uniform bioavailability score (0.55). Most were moderately soluble; remifentanil and PZM21 were more soluble. BBB penetration was predicted for all except remifentanil and PZM21. Only PZM21 was identified as a P-gp substrate, potentially limiting central nervous system accumulation. CYP inhibition varied: fentanyl, carfentanil, lofentanil, and sufentanil inhibited CYP3A4 and CYP2D6, whereas remifentanil and PZM21 lacked major CYP liabilities. Synthetic accessibility scores (2.2–3.7) indicated feasible chemical synthesis, and no Pan-Assay Interference (PAINS) or Brenk alerts were detected except a single alert for remifentanil. Reported Ki values for mOR (0.024–1.66 nM) aligned with docking predictions, supporting computational validity.

Conclusion : This integrated structural, functional, and pharmacokinetic analysis identifies FBD3 and PZM21 as promising scaffolds for developing safer opioid analgesics. Both compounds exhibit strong G protein signaling with minimal β-arrestin recruitment, favorable predicted ADME profiles, and reduced potential for drug–drug interactions compared with high-affinity balanced agonists such as fentanyl and carfentanil. By combining molecular docking, pharmacological assays, and in silico ADME profiling, this study provides a mechanistic framework for rational optimization of μ-opioid receptor ligands to achieve an improved efficacy–safety balance.

Keywords : Mu-opioid receptor agonist, G protein-biased agonists, Molecular docking, Fentanyl analogs, ADME profiling.

Count: 74
Abstract ID: 624
subject: Other: Other
Presentation Type: Poster

Stem Cell-Based Strategies for Neural Regeneration in Alzheimer's and Parkinson's Disease

Submission Author: Somaye Hesami

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Conclusion : Introduction: Progressive neurodegenerative diseases, Alzheimer's disease (AD), and Parkinson's disease (PD), present with profound cognitive or motor deficits, synaptic abnormalities, and cell degeneration. Currently available therapies are mostly symptomatic and do not arrest or reverse neurodegenerative processes. In the recent past, stem cell therapies have emerged as possible treatments for modulating the disease trajectory and promoting neural restoration. **Methods:** A structured search of the literature was carried out using PubMed, Scopus, and Web of Science. Search terms included "stem cell therapy," "neural regeneration," "Alzheimer's disease," "Parkinson's disease," "mesenchymal stem cells," "induced pluripotent stem cells," and "cell transplantation." We examined articles published from 2012 to 2024, reviewing both experimental studies and clinical trials to assess therapeutic efficacy, underlying biological mechanisms, and challenges in clinical translation. Results: Stem cell transplantation, as shown by preclinical studies and early clinical trials, may improve synaptic plasticity, neuronal survival, and cognitive or motor function. MSCs have exhibited anti-oxidative and anti-inflammatory effects, and in PD models, function was restored by iPSC-derived neurons. While long-term safety and efficacy continue to be investigated, these data show the therapeutic power of stem cell approaches. Conclusion: Through the integration of

immunomodulation, neuroprotection, and neuronal replacement, stem cell therapies hold the thrilling promise for neural regeneration in PD and AD. There are, however, points of concern to be addressed, including tumorigenesis, immunologic rejection, ethical implications, as well as standardization of protocols. Future developments of extracellular vesicle-based delivery platforms, gene editors, and biomaterials could also improve therapeutic efficacy as well as provide access to personalized regenerative medicine.

Keywords : Stem cells, Neural regeneration, Alzheimer's disease, Parkinson's disease, Neurodegeneration

Count: 75
Abstract ID: 765
subject: Other: Other
Presentation Type: Oral

The role of the "Reverse direction" plan in improving lifestyle and controlling temptation in addicts undergoing treatment

Submission Author: Narges Hedayati

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Background and Aim : A deeper look at the phenomenon of addiction reveals that this phenomenon is not limited to drug use, but encompasses the entire life of the addict. In a way, substance use can actually be considered a product of an addictive lifestyle, Therefore, recovery, more importantly than stopping use, depends on changing the addict's lifestyle,therefore, the idea of " Reverse Direction" was implemented as an innovative idea in the form a field research.

Methods : : For this research 30 people were randomly selected by the available sampling method as a research sample among the male addicts who referred to Addiction treatment center in Shiraz and were randomly placed into two groups of 15 people, experimental and control groups.The control group received only drug therapy, but the experimental group, in addition drug therapy, was asked to write a complete report of their daily life, If possible, mention the time. Then the "Reverse Direction " plan was given to the patient in the form of a daily schedule that covered from morning to the end of the day. In this program, the patient is supposed to do everything that he was not doing before in his current life: wake up at a specific time, exercise and walk as much as possible, eat three meals at a specific time and with his family whenever possible, socialize more and better with his family, cooperate in doing household and outside chores, and be more focused and committed in performing job duties. He should also have a specific time for sleeping at night, and in order to have quality sleep, put away all audio and video devices when he goes to bed, and the bedroom should be a place solely for sleeping and resting ……..etc. In order to investigate the effect of the program on improving the "lifestyle" and controlling "the temptation to use drugs" in patients who are quitting, the "Laali et al., 2012" lifestyle questionnaire and the "Fedardi et al., 2008" questionnaire for measuring the temptation to use drugs after quitting were used as research tools.

Results : The research findings showed that despite initial resistance and hesitation from the addicts in the experimental group and their families to implement the "Reverse Direction" plan, after joining this program, the experimental group experienced fewer temptations and a better and healthier lifestyle compared to the control group

Conclusion : The result of implementing the "Reverse Direction" plan was satisfactory feedback from the experimental group and their families. In this group, people who followed the daily plan carefully and really changed their lifestyle compared to those who did not fully follow the plan, had more successful treatment, physical and mental health, self-confidence, better relationships with their families, and overall, according to them, they experienced "better mood." Therefore, considering the successful results of the program, it is suggested that the "Reverse Direction" plan be implemented in other addiction treatment centers and clinics

Keywords : Reverse direction , lifestyle , temptation , Addict in treatment

Count: 76
Abstract ID: 1031
subject: Other: Other
Presentation Type: Poster

Examining the role of personality traits in Alzheimer's disease

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Background and Aim : Alzheimer's is the most common cause of forgetfulness and dementia among the elderly. This disease is a neurodegenerative disorder characterized by attention deficit, memory impairment, and learning disorders. Therefore, considering the severe complications of Alzheimer's, which can even lead to the death of the patient, it is of particular importance to pay attention to the factors that contribute to its development. However, it is important to note that so far, the treatment of this disease has been mainly studied from a neurobiological perspective, and less attention has been paid to the psychological and personality aspects of Alzheimer's. However, paying attention to the role of personality traits as a stable aspect of a human's identity in the study of all disorders is of particular importance. Therefore, considering the less treatment of this aspect, the present study has examined the role of personality traits as one of the important factors that can be a basis for the occurrence of this disease.

Methods : Due to the lack of research resources and research similar to the subject of the present study, this project was conducted at a micro-level and experimentally with a small sample and using a causal comparative method. The statistical population of this study was elderly people living in Shiraz, from whom 10 people were selected as the research sample through purposive sampling and were placed in two groups with and without Alzheimer's (5 people in each group). The tool for comparing the personality traits of the two groups was the NEO 5-factor test, and after collecting data, the independent groups T-test and analysis of covariance were used to compare the questionnaires and analyze the data.

Results : The findings of the study showed differences between the two groups in comparing their personality traits. This means that in relation to the "psychopathic" factor, the scores of the elderly with Alzheimer's were higher, but in the context of the "conscientiousness" and "openness to new experiences" factors, the group without Alzheimer's had higher scores. The difference between the two groups was significant compared to the aforementioned factors. In the context of the other two factors, namely "extraversion" and "agreeableness", the elderly without Alzheimer's had also obtained higher scores compared to the other group, but the difference in scores in these two factors was not significant.

Conclusion : Analysis based on the NEO model suggests that personality traits are not simply behavioral characteristics, but rather represent enduring coping styles, physiological responses to stress, and lifestyle choices that directly influence brain pathophysiology. “Psychopathology” acts as a significant risk factor through inflammatory biological pathways and unhealthy behaviors, while “Conscientiousness” and “Openness to Experience” have strong protective roles by promoting cognitive reserve and active self-regulation. “Extraversion” and “Adjustability” also reduce secondary risks from isolation and chronic stress by creating stable, socially supportive environments.

Keywords : Alzheimer's, aging, personality traits, NEO 5-factor test

Count: 77
Abstract ID: 873
subject: Other: Other
Presentation Type: Poster

In vitro blood-brain barrier models for drug screening and permeation studies

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Conclusion : In vitro blood–brain barrier (BBB) models are essential tools for studying CNS drug delivery, permeability, and neurotoxicity under controlled laboratory conditions. These models aim to replicate the structural, functional, and selective properties of the BBB, which is primarily composed of brain microvascular endothelial cells closely associated with astrocytes and pericytes. Static Transwell models provide simple, cost-effective platforms for measuring TEER and apparent permeability (Papp), enabling high-throughput drug screening. Co-culture models enhance tight junction integrity and transporter activity by mimicking cellular interactions, while dynamic microfluidic BBB-on-a-chip systems incorporate shear stress and flow, offering a more physiologically relevant environment and real-time monitoring of drug transport. Three-dimensional spheroid and organoid models replicate complex tissue architecture and intercellular signaling, supporting studies on nanoparticle delivery and disease modeling. Recent advances using human iPSC-derived BBB cells, organ-on-chip platforms, and computational prediction tools have improved translational relevance, making in vitro BBB models indispensable for screening drug permeability, evaluating neurotoxicity, and developing CNS-targeted therapeutics.

Keywords : Blood–Brain Barrier; Drug Permeability; BBB-on-a-Chip; iPSC-Derived Endothelial Cells; Neurotherapeutics

Count: 78
Abstract ID: 116
subject: Other: Other
Presentation Type: Poster

The Microbiota–Gut–Brain Axis (MGBA) in Neuropsychiatric Disorders: Insights from Animal Models and Emerging Clinical Applications

Submission Author: Ghazal Golalizadeh

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Conclusion : Neuropsychiatric disorders—including depression, anxiety, Parkinson's disease, and Alzheimer's disease—affect millions of people worldwide and remain major challenges for both medicine and society. While their origins are complex, growing evidence highlights the microbiota–gut–brain axis (MGBA)as a critical player in regulating brain function and behavior. The MGBA refers to the bidirectional communication between gut microbes, the immune and endocrine systems, and the central nervous system. Disturbances in this network have been consistently linked to mood changes, cognitive decline, and neurodegeneration. Animal studies have been instrumental in revealing these connections. For example, chronic stress in rodents alters gut microbial diversity, leading to depression- and anxiety-like behaviors, which can be partially reversed with probiotics or antibiotics. In models of Parkinson's disease induced by toxins such as Rotenone or 6-OHDA, shifts in microbial composition parallel increased inflammation and abnormal protein aggregation in the brain. Similarly, in Alzheimer's models, modulating the microbiota—through probiotics or even fecal microbiota transplantation—has been shown to enhance memory performance and reduce amyloid pathology. Mechanistically, the MGBA acts through several interconnected routes: vagus nerve signaling, modulation of the hypothalamic–pituitary–adrenal (HPA) stress axis, immune responses, and microbial metabolites like short-chain fatty acids. These pathways collectively influence neuroplasticity, neurogenesis, and the availability of growth factors such as BDNF. Importantly, these insights are no longer limited to animal research. Early clinical studies suggest that dietary interventions and probiotics may improve mood and cognition in patients, while more advanced approaches—such as stem cell–based therapies—are being investigated for their ability to modulate both immunity and gut microbial balance. In summary, the MGBA offers a promising framework for understanding brain–gut communication and opens new avenues for treatment. Bridging animal research with human studies could lead to innovative strategies for managing neuropsychiatric disorders.

Keywords : Microbiota–gut–brain axis, MGBA, depression, Parkinson's disease, Alzheimer's disease, probiotics

Count: 79
Abstract ID: 137
subject: Other: Other
Presentation Type: Poster

Ventral tegmental area inhibition enhances cortical delta and theta rhythms during non-REM sleep without altering sleep architecture

Submission Author: Zahra Abdi

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Background and Aim : While sleep architecture is physiologically crucial for maintaining neural, metabolic, and systemic homeostasis, contributions of different brain regions to its regulation still remain unclear. Ventral tegmental area (VTA) is one of the important components of arousal system, but its role in shaping sleep architecture is not well characterized. In this study, we pharmacologically inhibited this area to assess its contribution to sleep structure and brain cortical population activity by examining the sleep EEG.

Methods : Male Wistar rats weighing 250–270 g underwent stereotaxic surgery for the bilateral implantation of guide cannulas into the VTA. In addition, an electroencephalography electrode was implanted above the somatosensory cortex on the skull and an electromyography electrode was implanted in the nuchal muscle. After the post-surgical recovery period, animals in the lidocaine group received bilateral intra-VTA injections of lidocaine hydrochloride (2%, 0.5 µL over 2 min), while control animals received saline (0.9%, 0.5 µL). The animals were then allowed to sleep for 2 hours, and their sleep structure was evaluated.

Results : No significant difference was found between the lidocaine and control groups in the number of sleep episodes, total sleep duration, sleep duration, or REM/NREM ratio. EEG analysis during NREM sleep revealed that VTA inhibition increased delta ($P < 0.05$) and theta ($P < 0.01$) power in the lidocaine group compared to controls. In contrast, no significant changes were observed in any of the EEG bands during REM sleep.

Conclusion : These findings suggest that the VTA plays an important role in regulating the pattern of cortical neuronal activity in NREM sleep.

Keywords : Sleep architecture, ventral tegmental area, brain rhythm, electroencephalography

Count: 80
Abstract ID: 29
subject: Other: Other
Presentation Type: Oral

Correlating EEG Topography and MRI Features for Early Detection of Alzheimer's Disease: A Case Study

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Background and Aim : Early diagnosis of Alzheimer's disease (AD) remains a major clinical challenge, with significant implications for treatment and care. Alzheimer's disease is a leading cause of dementia, imposing significant clinical and socioeconomic burdens worldwide. Early diagnosis is critical for effective intervention and disease management. Modern diagnostic approaches leverage advanced neuroimaging, biomarkers, and artificial intelligence to detect pathological changes prior to clinical symptom onset. Continued research in this area is essential to improve early detection and therapeutic outcomes.

Methods : In this case study, we investigate the potential of combining quantative electroencephalographic (qEEG) with structural magnetic resonance imaging (MRI) to enhance early diagnostic accuracy. A 68-year-old patient with mild cognitive impairment and clinical suspicion of prodromal AD underwent high-resolution qEEG recording and brain MRI.

Results : EEG analysis revealed specific alterations in alpha and theta band activity, predominantly in parietal and temporal regions. MRI findings showed hippocampal atrophy and cortical thinning, consistent with early neurodegenerative changes. Importantly, a topographic correlation was observed between EEG slowing and the regions of structural damage identified on MRI.

Conclusion : These findings suggest that qEEG may reflect functional deterioration in regions already affected structurally, highlighting its potential as a non-invasive, cost-effective tool for early AD screening. This case supports the integration of EEG and MRI in clinical protocols for improved early-stage Alzheimer's detection. Further studies are warranted to validate these observations across larger samples.

Keywords : Alzheimer's disease; qEEG; MRI; early diagnosis; brain mapping; multimodal imaging

Count: 81
Abstract ID: 747
subject: Other: Other
Presentation Type: Poster

Generation of Motor Neuron-Replacement Cells in ALS Using Engineered Bacteria and Signal Analysis via Neuralink Chip

Submission Author: Hossein Zare

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Background and Aim : Amyotrophic Lateral Sclerosis (ALS) is a progressive neurodegenerative disease characterized by the selective loss of upper and lower motor neurons, leading to paralysis and respiratory failure. Current treatments, including riluzole and edaravone, primarily slow the disease progression but do not restore lost neurons. The pathological hallmarks of ALS—TDP-43 aggregation and elevated neurofilament light chain (NfL) levels—serve as potential diagnostic and therapeutic targets (Ling et al., 2013; Benatar et al., 2021). Recent advancements in synthetic biology and brain–computer interface (BCI) technologies such as Neuralink open new possibilities for real-time disease monitoring and regenerative therapy. This study proposes an integrative bioelectronic model in which engineered bacteria act as biosensors and neurotransmitter mediators that communicate with the Neuralink chip to analyze disease biomarkers and stimulate stem cell differentiation into motor neurons.

Methods : The proposed system integrates three components: Engineered Bacteria as Biosensors: Nonpathogenic bacterial strains such as E. coli Nissle 1917 or Lactobacillus casei are genetically modified using ligand-sensitive promoters and synthetic gene circuits to detect ALS-specific biomarkers, including misfolded TDP-43 proteins and increased NfL concentrations (Nguyen et al., 2020). Upon detection, these bacteria release natural or synthetic neurotransmitters (e.g., glutamate, GABA, dopamine analogs). Neuralink Interface as a Bioelectronic Analyzer: The Neuralink chip detects these neurotransmitters using nanoscale biosensors such as organic electrochemical transistors (OECTs) or graphene-based field-effect transistors (graphene-FETs) (Rivnay et al., 2018). The chemical information is converted into electrical signals and processed via embedded algorithms to assess neuronal damage or activity status. Stem Cell Response Module: Based on Neuralink's analysis, electrical, optical, or chemical stimuli are delivered to stem cells—either iPSC-derived motor neuron progenitors (iPSC-MNPs) or neural stem cells (NSCs)—to promote differentiation into mature motor neurons (Lu et al., 2012; Sances et al., 2016). This forms a closed-loop bioelectronic network, where each stage continuously feeds back into the next: detection → transmission → processing → response → recalibration.

Results : In this conceptual framework, engineered bacteria successfully function as in vivo biosensors, detecting ALS biomarkers and translating them into neurotransmitter-based chemical signals. The Neuralink system, using its ultra-sensitive detection platform, can interpret these biochemical signals with millisecond-scale resolution. Simulations and prior experimental analogs from microbial-electronic systems indicate that neurotransmitter release rates can be correlated with disease activity, enabling Neuralink to dynamically compute the necessary regenerative command. Subsequently, stem cell cultures respond to Neuralink-driven stimulation by differentiating into functional motor neurons with normal electrophysiological activity (as supported by Sances et al., 2016). The feedback loop then re-engages as bacteria detect biochemical changes resulting from neural repair, maintaining homeostatic control. Such a system could minimize therapeutic dosing errors, prevent uncontrolled proliferation, and adapt in real-time to patient-specific neural responses (Zhang et al., 2023).

Conclusion : This integrated bacteria–Neuralink–stem cell model offers a novel approach to treating ALS through dynamic, self-regulating neuroregeneration. By combining microbial biosensing, nanoelectronic analysis, and stem cell neurodifferentiation, the system establishes a biological–electronic interface capable of autonomous monitoring and repair of neural damage. Although theoretical, this framework represents a step toward future precision neuroregenerative medicine, potentially extending to other neurodegenerative diseases such as Parkinson’s and spinal muscular atrophy. Further experimental validation will be essential to evaluate microbial neurotransmitter signaling efficiency, Neuralink’s detection sensitivity, and long-term integration with neural tissues.

Keywords : Amyotrophic Lateral Sclerosis (ALS); Engineered bacteria; Synthetic biology; Neuralink; Brain–computer interface (BCI); Neuroregeneration; Stem cell differentiation; Bioelectronic closed-loop system

Count: 82
Abstract ID: 972
subject: Other: Other
Presentation Type: Oral

Assessing the effects of chronic morphine administration during adolescence on electrophysiological properties of locus coeruleus neurons

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Background and Aim : Adolescence represents a critical period of neurodevelopment characterized by extensive remodeling of neural circuits and increased vulnerability to drug-induced neuroadaptations. Among the brain regions affected by opioids, the locus coeruleus (LC) plays a key role in regulating arousal, pain perception, stress responses, and opioid dependence. Previous studies have shown that morphine exposure during adolescence can induce long-lasting alterations in nociceptive processing, opioid tolerance, and neuronal function, which may even extend to subsequent generations. The present study investigated how repeated morphine administration during adolescence influences the electrophysiological properties of LC neurons in adulthood.
Methods : During adolescence, male Wistar rats were administered subcutaneous morphine (twice daily) according to an escalating dose regimen, whereas control rats received saline. In adulthood, whole-cell patch clamp recordings in current clamp mode were conducted on locus coeruleus neurons from brain slices.
Results : Morphine exposed rats exhibited a significant increase in rheobase current and utilization time compared to controls, indicating alterations in spike initiation threshold and enhanced stability of excitability. Additionally, a trend toward increased irregularity in action potential firing rate was observed in the morphine treated group relative to saline-treated controls.
Conclusion : These findings indicate that chronic morphine exposure during adolescence leads to persistent modifications in the electrophysiological properties of LC neurons. These neuroadaptations may underlie altered regulation of arousal, pain, and stress, offering mechanistic insight into the enduring effects of adolescent opioid exposure. Elucidating these changes may advance our understanding of opioid-induced neuroplasticity and inform the development of targeted interventions for opioid dependence and its long-term consequences.
Keywords : Opioid; adolescence; locus coeruleus; patch clamp recording

Count: 83
Abstract ID: 494
subject: Other: Other
Presentation Type: Poster

Neurodevelopmental Trajectories of Siblings in Genetic Neurological Disorders: A Framework for Identity, Resilience, and Neural Adaptation

Submission Author: Mohammadamir Beigihabibabadi

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Conclusion : Siblings of individuals with genetic neurological disorders constitute an understudied and theoretically informative population whose developmental trajectories can illuminate mechanisms of identity formation, resilience, and neuroplasticity. Here, we integrate findings from developmental neuroscience, clinical genetics, behavioral genetics, psychoneuroendocrinology and family systems research to propose a unifying framework—the Sibling Neurogenetic Adaptation Model (SNAM)—which specifies how shared genotypes, familial caregiving roles, and dynamic environmental exposures interact across developmental windows to shape neural circuitry underlying social cognition and self-concept. We first synthesize behavioral and clinical literatures showing that siblings of affected probands exhibit heterogeneous psychosocial outcomes that cluster into profiles characterized by (i) increased prosocial orientation and empathic accuracy, (ii) heightened stress responsivity and internalizing symptoms, or (iii) normative functioning with subclinical cognitive or

socioemotional differences. Next, we review neurobiological substrates plausibly implicated in these profiles, highlighting convergent evidence implicating the medial prefrontal cortex, temporoparietal junction, anterior insula, and distributed default-mode and salience networks in social-cognitive processing and identity-related self-referential cognition. We evaluate psychoneuroendocrine data—altered diurnal cortisol rhythms, hair cortisol accumulation, and autonomic reactivity—and consider how sustained caregiving demands or family stressors may produce allostatic load that interacts with neurodevelopmental timing. Importantly, we integrate genetic and genomic perspectives: siblings frequently share rare variants, copy-number changes, or polygenic liabilities with probands, yet phenotypic penetrance is variable, suggesting potent moderating effects of environment, epigenetic regulation, and stochastic developmental processes. We propose explicit, testable mechanistic pathways by which (i) shared genetic architecture predisposes to subtle neurocognitive endophenotypes, (ii) caregiving role adoption and family dynamics produce environmentally mediated neural adaptations, and (iii) gene×environment and mediation processes yield the observed psychosocial heterogeneity. To operationalize SNAM, we outline an empirical roadmap requiring multisite longitudinal cohorts with deep phenotyping: harmonized structural and functional neuroimaging (resting-state connectivity, social-cognitive tasks, diffusion MRI), whole-genome sequencing and polygenic scoring, epigenetic profiling, dense ambulatory assessment (ecological momentary assessment, actigraphy), and repeated psychoneuroendocrine sampling. We specify analytic strategies—multilevel growth models, structural equation modelling, causal mediation analysis, and integrative multiomic machine-learning pipelines—and recommend statistical power targets and sensitivity analyses to disentangle familial confounding. Finally, we discuss translational implications for family-centered genetic counseling, targeted prevention for at-risk siblings, and policy that recognizes siblings both as potential resilience resources and as individuals with discrete service needs. By positioning siblings at the intersection of genes, brain, and family ecology, SNAM reframes neurogenetic research to capture graded, transdiagnostic developmental processes with broad implications for neuroscience, clinical practice, and public health. This synthesis also highlights ethical considerations surrounding disclosure, reproductive counseling, and differential allocation of family resources, and it proposes testable hypotheses for next-generation cohort studies aimed at elucidating causal pathways and implementation science.

Keywords : Neurodevelopment; Genetic Neurological Disorders; Neuroplasticity; Gene–Environment Interaction; Identity and Resilience

Count: 84
Abstract ID: 531
subject: Other: Other
Presentation Type: Oral

Investigation of the Capillary Structure of Hippocampal Tissue from Healthy Rats Following Endurance Training

Submission Author: Fariba Gudarzi

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Background and Aim : Background and Objective: The structure and function of the hippocampal capillary are closely related to the regulation of neurogenesis and synaptic plasticity. Maintaining the health and proper organization of the hippocampal capillary network is essential for optimal hippocampal tissue function and cognitive performance. Given the effective effects of exercise training on the body, this study was conducted to investigate the effect of endurance training (Ex) on histomorphometric estimates of the capillaries of the hippocampal tissue of healthy rats.

Methods : Materials and Methods: In the present experimental study, 14 male Wistar rats were divided into 2 groups of 7: control and endurance training (Ex). After one week of familiarization with the laboratory conditions and treadmill running, endurance training was performed 5 days a week for six weeks. 48 hours after the completion of the research protocol, the rats were sacrificed, then the hippocampal tissue was removed and placed in 10% formalin for hematoxylin-eosin staining. After tissue staining, histomorphometric estimates of the number of sections, length, volume, and area of capillaries in rat hippocampal tissue were examined and compared.

Results : Findings: The results showed that the number of sections, length, volume, and area of capillaries in hippocampal tissue were not significantly affected by endurance training.

Conclusion : Conclusion: These findings may indicate that under certain conditions or at certain time intervals, endurance training does not have positive effects on capillary structure or that a longer period of time may be required to observe changes.

Keywords : Hippocampus; exercise training; vascular structure.

Count: 85
Abstract ID: 288
subject: Other: Other
Presentation Type: Poster

Comparison of pain modulatory effect of the LPGi estragon receptor on inflammatory pain between pro-estrus and estrus phases and OVX rats

Submission Author: Sanam Ansari

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Background and Aim : This study investigates how variations in circulating estrogen levels modulate inflammatory pain through the lateral paragigantocellularis (LPGi) nucleus. Comparing intact female rats during pro-estrus (high estrogen) and estrus (low estrogen) phases with ovariectomized (OVX) estrogen-depleted rats, it examines the role of LPGi estrogen receptors in formalin-induced nociceptive behaviors. The research aims to determine whether hormonal status influences pain perception via central mechanisms, addressing a critical gap in understanding sex-specific pain modulation. The findings could elucidate hormonal pathways in pain processing, relevant to developing targeted analgesics for hormonal fluctuation states .

Methods : The study utilized female rats grouped into pro-estrus, estrus, and OVX categories. Microinjections of 17β-estradiol (0.8 μmol) and the estrogen receptor antagonist ICI 182,780 (15 nmol) were administered into the right LPGi nucleus. Formalin-induced inflammatory pain was assessed by measuring flexing and licking durations of the hind paw for 60 minutes. Statistical analyses compared responses across groups and treatments to evaluate drug effects and hormonal influences. All procedures adhered to ethical guidelines (NIH Publication No. 80-23, revised 1996) .

Results : Intra-LPGi 17β-estradiol significantly reduced flexing and licking durations in pro-estrus and estrus rats ($P < 0.001$), demonstrating analgesia. In OVX rats, it increased flexing ($P < 0.05$) but decreased licking ($P < 0.05$). The antagonist ICI 182,780 reversed these effects: it abolished analgesia in intact rats ($P < 0.001$ for flexing; $P < 0.05$–0.001 for licking) and restored nociception in OVX rats ($P < 0.001$). Results indicate estrogen's analgesic role in intact rats is hormone-level-independent but requires basal estrogen receptor activation .

Conclusion : The findings confirm that LPGi estrogen receptors mediate inflammatory pain modulation, with analgesia observed in both pro-estrus and estrus phases, suggesting hormonal state independence in intact rats. Conversely, OVX outcomes highlight the necessity of basal estrogen for coherent analgesic effects. The antagonist reversal underscores receptor-specific mechanisms. These results emphasize the LPGi's role in hormonal pain processing and suggest that estrogen depletion disrupts balanced modulation. Clinically, this highlights the need for considering hormonal status in pain management, particularly in low-estrogen conditions, to develop effective therapies targeting central estrogen pathways .

Keywords : 17β-Estradiol · ICI 182,780 · Pain · Analgesia · Rat

Count: 86
Abstract ID: 346
subject: Emotion, Motivation and Behavior: Neural Basis of Human Behavior
Presentation Type: Oral

Investigating the Amplitude of the P1 Component in Multisensory Information Processing during Decision-Making in the Central-Frontal Regions of Brain

Submission Author: Kosar Abbaspour

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Background and Aim : One of the fundamental questions in sensory information processing research is whether experience and expertise can influence sensory-perceptual processing. The aim of the present study is to investigating the amplitude of the p1 component in multisensory information processing during decision-making in the frontal and central regions of brain in elite badminton players.

Methods : The present study was a semi-experimental study with an applied objective and was conducted on 13 elite badminton players based on simple random sampling. Brain signals of badminton players were measured using a 30-channel active electroencephalography device with three blocks of 72 trials of visual, auditory, and visual-auditory stimuli based on four spatial decision-making conditions. Data analysis was performed using MATLAB, ERPLAB, EXCEL and SPSS 26 software at a significance level of 0.05.

Results : For the early component P1 in the 40 to 120 millisecond range, significant differences were observed in the peak amplitude of P1 in the Cz and Fz channels across three sensory

conditions. The repeated measures ANOVA revealed significant differences in the amplitude of the P1 component at the Cz channel ($F(2,24) = 3.53$, $p = 0.045$, partial $\eta^2 = 0.23$) and the Fz channel ($F(2,24) = 3.46$, $p = 0.046$, partial $\eta^2 = 0.23$). Based on grand-average values, the peak amplitudes at the Cz channel for the visual, auditory, and visuo-auditory conditions were 0.37 µV, 0.50 µV, and 0.54 µV, respectively, within the 40–120 ms time window. The peak amplitude at the Cz channel was higher in the sensory integration condition (visuo-auditory) compared to the auditory and visual conditions. At the Fz channel, peak amplitudes were 0.83 µV for the visual condition, 0.77 µV for the auditory condition, and 0.32 µV for the visuo-auditory condition. Pairwise comparisons at the Cz channel indicated significant differences in peak amplitude between the sensory integration (visuo-auditory) condition and both the visual and visuo-auditory conditions ($p = 0.013$). At the Fz channel, the peak amplitude was highest in the visual condition, with a significant difference observed between the auditory and visuo-auditory conditions ($p = 0.01$).

Conclusion : Based on the model of cross-modal integration, elite individuals have utilized cognitive brain regions and attention allocation areas according to their level of expertise for processing multi-sensory and even unisensory information (visual). Therefore, long-term reinforcement (sport experience) may potentially increase brain information processing speed during decision-making in central-frontal area. Cognitive demands and task-related experience contribute to the brain's adaptive efficiency. For instance, professional athletes, compared to novices, exhibited not only reduced activity in the occipito-parietal visual cortex but also enhanced efficiency in central-frontal regions. As an individual's experience increases, reliance on predictive feedback becomes critical, enhancing the processing speed of cognitive regions. According to the top-down processing perspective, perception is guided by higher-level cognitive processes, particularly those involving frontal regions. In the context of sensory processing, this implies that prior experiences (e.g., specialized sports training) and expectations significantly influence the detection and interpretation of sensory information.

Keywords : Multisensory, Information Processing, Decision-Making, Brain, Sport

Count: 87
Abstract ID: 682
subject: Emotion, Motivation and Behavior: Neural Basis of Human Behavior
Presentation Type: Poster

The Cerebellar Personality: How Age and Gender Modulate the Link Between Openness to Experience and the Cerebellum

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Background and Aim : Challenging its traditional depiction as a motor control center, the cerebellum is increasingly recognized for its role in higher-order cognitive and affective functions. This study extends this view to the personality trait of Openness, a core component of the 'Plasticity' metatrait, a higher-order dimension reflecting a unified drive for cognitive and behavioral exploration. This exploratory function is fundamentally reliant on the cerebellum's hypothesized role as a predictive system for navigating novelty. However, the direct structural link between cerebellar volume and openness to experience in different ages and genders remains ambiguous. This study investigates this link, hypothesizing that any association is critically moderated by age and gender.

Methods : Data were obtained from the Human Connectome Project (HCP-1200). A final sample of 609 healthy participants was stratified by gender (281 males, 328 females) and into two distinct adult age groups (22-25 and 30-35 years). Volumetric data for four cerebellar subdivisions (left/right cortex, left/right white matter) were extracted from high-resolution T1-weighted (T1w) structural MRI scans. Non-parametric Spearman's rank correlation was employed to assess the relationship between these volumes and scores from the NEO Five-Factor Inventory (NEO-FFI). Outlier data were systematically managed using the IQR method to ensure robustness.

Results : Our focused analysis revealed a highly specific finding. A significant positive correlation emerged between Openness and the volume of both the left cerebellar cortex (ρ=0.24, p=0.003) and the right cerebellar cortex (ρ=0.22, p=0.008), but exclusively in the younger male cohort (22-25 years). No significant associations with openness were observed in older males or in females of either age group, establishing this as a highly moderated effect.

Conclusion : These findings provide compelling evidence for a specific cerebellar substrate of openness to experience, but only within the neurodevelopmental context of young manhood. This suggests the cerebellum's structure is dynamically linked to the exploratory drive characteristic of this life stage. By employing a stratified analysis, our study helps resolve previous inconsistencies and underscores the necessity of accounting for demographic factors in neurobehavioral research. This supports the cerebellum's integral role within the complex, dynamic neural architecture that underpins human personality.

Keywords : Cerebellum; Personality; Openness to experience; Plasticity; Gender Differences; Age Differences

Count: 88
Abstract ID: 695
subject: Emotion, Motivation and Behavior: Neural Basis of Human Behavior
Presentation Type: Poster

How Gender Differences Influence the Role of Temporal Lobe Morphology in Personality Associations Across Young Adulthood

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Background and Aim : The temporal lobe plays a central role in memory, emotion regulation, and social cognition—functions tightly linked to personality traits. While both brain structure and personality exhibit gender-specific developmental patterns, their interactive effects on personality organization remain poorly characterized. This study examines how gender differences influence the relationship between temporal lobe morphology and big five personality traits during middle adulthood.
Methods : Structural MRI and personality data were obtained from the Human Connectome Project (HCP) Young Adult dataset (age 22-35). The present study focused on a middle-aged young adult cohort aged 31-35 from the larger HCP sample. Participants were stratified by gender (females: N=268; males: N=150). Thickness and surface area measures were extracted from 36 bilateral temporal lobe regions defined by the Desikan-Killiany atlas using Free Surfer and HCP preprocessing pipelines. Bayesian variable selection identified the most predictive regions for each trait, followed by non-parametric Spearman correlation analysis with false discovery rate correction (significant level at $q < 0.05$).
Results : Analysis revealed distinct, gender-specific associations between temporal lobe morphology and personality traits in middle adulthood. In females, a positive association was observed between agreeableness and cortical thickness in the bilateral Banks of the Superior Temporal Sulcus (BanksSTS) and the left entorhinal cortex. Additionally, a positive association was observed between openness and the surface area of the right parahippocampal cortex. In males, a different pattern was observed, with a negative association between conscientiousness and surface area in several regions, including the bilateral middle temporal, left superior temporal, and right entorhinal cortices.
Conclusion : Our findings reveal gender-specific associations between temporal lobe morphology and personality. This underscores the necessity of considering gender as a key variable in temporal lobe-personality research.
Keywords : Temporal Lobe Morphology; MRI; Big Five Personality Traits; Gender Differences; Brain-Behavior Relationships; Human Connectome Project

Count: 89
Abstract ID: 102
subject: Emotion, Motivation and Behavior: Neural Basis of Human Behavior
Presentation Type: Poster

Examining the relationship between severity of externalizing disorders and EEG-based resting-state functional connectivity

Submission Author: Sara Souri

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Background and Aim : Externalizing disorders are characterized by a set of specific behavioral problems often rooted in weak cognitive control and emotion regulation. Individuals with these disorders exhibit difficulties in inhibiting impulsive behaviors, controlling emotions, and responding appropriately to social cues, which can lead to destructive and aggressive actions. Neurobiological-cognitive mechanisms underlying these deficits, especially disruptions in the functioning of brain networks and regions responsible for executive functions and emotional processing, have become an area of interest for researchers. In the present study, we aim to examine potential alterations in functional connectivity within two major networks, the default mode network (DMN) and the cognitive control network (CCN), through resting-state EEG signals, and to investigate their association with the severity of externalizing symptoms.

Methods : The study population consisted of male and female adolescents aged 13 to 15 years attending middle schools in Tehran. Participants were divided into two groups: control (17 individuals, mean = 14.2) and externalizing (17 individuals, mean = 13.8). Individuals in the externalizing group were selected based on their scores on the externalizing subscale of the Child Behavior Checklist. Using electroencephalography (EEG) during resting-state conditions, we examined functional connectivity differences between the two groups using the lagged phase synchronization (LPS) index within the DMN and the CCN with LORETA software. Significant between-group connectivity differences were then correlated with externalizing symptom severity within the externalizing group.

Results : Reduced functional connectivity was observed in both DMN and CCN networks among the externalizing group. This reduced connectivity included alpha-band functional

connectivity between the parahippocampal gyrus and the right prefrontal cortex in the DMN and between the right and left anterior insula (frontal operculum) and the right and left dorsolateral prefrontal cortex, as well as between the left dorsolateral prefrontal cortex and the right inferior frontal junction in the beta band within the CCN. A significant negative correlation was found between functional connectivity of the left dorsolateral prefrontal cortex and the right inferior frontal junction and aggression scores.

Conclusion : our findings show that altered resting-state connectivity in the DMN, involved in self-referential and emotional processing, and the CCN, central to executive control, is associated with externalizing symptom severity, suggesting network-level dysfunction as a potential biomarker for psychopathology.

Keywords : Externalizing disorder; Functional connectivity; Default mode network; Cognitive control network; Aggression

Count: 90
Abstract ID: 534
subject: Emotion, Motivation and Behavior: Neural Basis of Human Behavior
Presentation Type: Poster

The effectiveness of virtual reality-based mindfulness training on reducing cognitive avoidance in students with specific learning disabilities

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Background and Aim : Specific learning disorder in children is a neurodevelopmental disorder that affects the ability of the brain to receive and process information through the interaction of environmental and hereditary factors. Despite having normal intelligence and due to the lack of biological defects in academic fields (reading, writing and arithmetic), these children have weaker academic performance. When these students experience unpleasant behavior in social relationships, they mentally process that situation and use cognitive avoidance strategies to change their mental contents about the situation and try to use avoidance measures to get rid of worrying thoughts about social situations. Frequent avoidance of the environment reduces social activities and positive behaviors, limits responses, and on the other hand strengthens the person's immobility, which can negatively affect the person's adaptation to external events. The aim of this study was to investigate the effect of virtual reality-based mindfulness therapy on improving cognitive avoidance in students with specific learning disabilities.

Methods : The research method was semi-experimental with a pre-test, post-test and a two-month follow-up. The statistical population was female students with learning disabilities who referred to learning disability centers in the academic year 1401-1402 in Kermanshah city. cognitive avoidance questionnaire was administered to the students, and among those who met the criteria for entering the research, 24 students were selected by purposive sampling and randomly placed in two groups of 12 people, experimental and control. The experimental group underwent 10 sessions of mindfulness training based on virtual reality, but the group did not receive the training certificate. One week after the last training session, a post-test was conducted and two months later, a follow-up test was conducted

Results : Data were analyzed using repeated measures analysis of variance. The results showed that mindfulness training reduces students' cognitive avoidance ($P<0.001$) and these effects were stable in the follow-up phase.

Conclusion : Since mindfulness training based on virtual reality reduces cognitive avoidance of these students, this efficient method can be used to reduce the incompatibility of these students

Keywords : Specific learning disorder, mindfulness, virtual reality, cognitive avoidance.

Count: 91
Abstract ID: 47
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Oral

Prenatal exposure to polystyrene nanoplastics: neuroinflammation and impaired brain development in offspring

Submission Author: Mojtaba Ehsanifar

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Background and Aim : The increase in plastic production worldwide makes the environmental threat posed by microplastics, which originate from large amounts of plastic waste, increasingly significant. These small plastic particles are prevalent among various organisms in different ecosystems, raising concerns about their potential effects. In this study, we investigated how polystyrene nanoplastics affect the development of the central nervous system.

Methods : We studied mice exposed to nanoplastics during their development to study these effects.

Results : Our findings show that when nanoplastics are administered to mothers during pregnancy and lactation, they disrupt the normal function of NSCs, alter the composition of nerve cells, and alter the brain structure of their offspring. Furthermore, we discovered that exposure to significant concentrations of nanoplastics during development leads to abnormal brain development that leads to gender-specific neurophysiological and cognitive impairments.

Conclusion : This suggests that exposure to high levels of nanoplastics could increase the risk of neurodevelopmental disorders.

Keywords : Nanoplastics, Prenatal exposure, Brain development, Neuroinflammation.

Count: 92
Abstract ID: 88
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

Neurotoxicity and neurobehavioral alteration following exposure to micro and nanoplastics

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Background and Aim : In the age of plastic, living organisms are constantly exposed to micro- and nanoplastic particles. Plastics accumulated in living organisms have negative effects on the body, although studies evaluating their effects on brain development are scarce.

Methods : In this study, the effects of nanoplastics on nervous system development were investigated using mice exposed to nanoplastics at different stages of brain development.

Results : Gene expression related to brain development was reduced after exposure to nanoplastics in the brains of fetal and adult mice exposed to nanoplastics. Furthermore, offspring of mothers treated with nanoplastics showed signs of anxiety and depression-like behaviours and abnormal social behaviours.

Conclusion : We observed that the accumulation of plastic particles in the brain disrupts brain development and behavior in mice. This study provides new information about plastic toxicity and its harmful effects on neural development and behavior in mammals.

Keywords : Neurotoxicity, Behavioral alteration, micro and nanoplastics, oxidative stress, neuroinflammation

Count: 93
Abstract ID: 605
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

Integrative Auditory Entrainment and Mindfulness/CBT Reduce Anxiety via QEEG Network Modulation

Submission Author: Mohammad Asadi

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Background and Aim : Exposure to chronic stress contributes considerably to the development of cognitive impairments in psychiatric disorders such as depression, generalized anxiety disorder (GAD), obsessive-compulsive disorder (OCD), posttraumatic stress disorder (PTSD), and addictive behavior. While mindfulness- and CBT-based programs are effective in reducing stress and improving psychological outcomes, their effects on physiological regulation (e.g., autonomic balance, cortical oscillations) may be enhanced when combined with bottom-up approaches such as auditory frequency entrainment. auditory neural entrainment (theta/alpha) may complement top-down strategies by modulating cortical rhythms and enhancing prefrontal control. We tested an integrative program combining auditory neural entrainment, mindfulness, and CBT-informed self-care and evaluated outcomes using quantitative EEG (QEEG) and validated anxiety scales.

Methods : -Methods *Participants and Design: Sixty adults experiencing elevated stress levels participated in a quasi-experimental study lasting four-weeks (experimental group: n = 25; control group: n = 35). Participants were recruited from the community and screened for moderate-to-high perceived stress. The study employed a mixed design, with pre–post assessments of both neurophysiological and psychological outcomes. *Intervention: The experimental group received a four-week, multicomponent program integrating: Nightly auditory entrainment using theta–alpha frequency stimulation, Daily mindfulness practice (guided sessions, 10–15 minutes), Weekly CBT-informed self-care modules focusing on cognitive reframing and behavioral regulation. The control group received no intervention and continued their usual daily routines. *Measures Primary neurophysiological outcomes included absolute power in theta (4–7 Hz), alpha (8–12 Hz), and beta (13–30 Hz) frequency bands at frontal regions of interest (F3, F4). Additional network indices related to alertness, default

mode network (DMN) modulation, and mood regulation were derived from QEEG analyses. Primary psychological outcomes included changes in anxiety severity, measured by the Hamilton Anxiety Rating Scale (HAM-A) and the Beck Anxiety Inventory (BAI). Secondary and exploratory outcomes included qualitative feedback collected through semi-structured interviews at post-intervention, analyzed using thematic (inductive) methods. *Data Analysis: Within-group pre–post changes were examined using paired-sample t-tests. Between-group differences were analyzed using ANCOVA, controlling for baseline anxiety, age, and sex. Effect sizes were calculated using Cohen's d for psychological outcomes and Hedges' g for QEEG measures. For neurophysiological data, multiple comparisons across frequency bands and regions were corrected using the Holm–Bonferroni procedure. Secondary and exploratory outcomes, including participant feedback and semi-structured interviews, were analyzed descriptively and qualitatively (thematic/inductive analysis) without inferential statistics.

Results : In the experimental group, HAM-A decreased by 7.25 points (95% CI −10.79 to −3.71; paired $g \approx 0.82$), and BAI decreased by 4.58 points (95% CI −8.02 to −1.15; $g \approx 0.53$). QEEG showed frontal alpha increases and theta/beta reductions: e.g., F3 alpha rose from ~16→~32 μV^2 (large; reported $d \approx 1.25$), while F3 theta fell from ~18.5→~15.3 μV^2. Network indices improved (Alertness, DMN modulation, Mood Regulation; $\Delta \approx +1.2$ to +1.7), whereas the control arm exhibited minimal change across these QEEG summaries. Exploratory Cattell dimensions indicated reductions in tension (Q4) and apprehension (O) in the experimental cohort, with heterogeneity across factors.

Conclusion : A brief intervention combining auditory entrainment with mindfulness/CBT reduced anxiety and produced measurable frontal QEEG changes in adults with elevated stress. These results support non-pharmacological stress-regulation strategies and motivate future randomized trials to optimize intervention parameters and individual outcomes.

Keywords : Auditory entrainment; Mindfulness; CBT; Chronic stress; Anxiety reduction, QEEG, Integrative neurobehavioral intervention

Count: 94
Abstract ID: 686
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Oral

AdipoRon as a Dual Anti-Inflammatory and Neuroprotective Modulator in Sleep Loss–Driven Depressive Phenotypes

Submission Author: Beriwan Malaie

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Background and Aim : Chronic sleep restriction (SR) leads to significant neurobiological alterations that play a role in the development of depression-like behaviors, primarily through inflammatory activation and diminished neural resilience. This research examined the antidepressant mechanisms of AdipoRon, a pharmacological activator of adiponectin receptors, in alleviating depressive phenotypes induced by SR.

Methods : . Forty male Swiss albino mice were randomly divided into four groups: control (wide platform), SR, SR + melatonin, and SR + AdipoRon. SR was initiated utilizing a modified multiple-platform approach over a period of 21 consecutive days, following which intranasal drug treatments were implemented. Behaviors indicative of depression were assessed through the Forced Swimming Test (FST) to evaluate behavioral despair and the Sucrose Splash Test (SST) to measure apathy and deficits in self-care.

Results : Exposure to SR notably extended the duration of immobility in the FST and decreased grooming time in the SST, suggesting an increase in behavioral despair and a decline in motivation. The intranasal administration of AdipoRon (10 μg/mouse/day) successfully mitigated these behavioral impairments, leading to a reduction in immobility and a restoration of grooming activity, demonstrating more effective results than melatonin. Molecular analyses demonstrated significant upregulation of phosphorylated nuclear factor kappa B (NF-κB) and increased interleukin-1β (IL-1β) expression in the prefrontal cortex (PFC) of mice exposed to stress, establishing a connection between chronic neuroinflammation and depressive behavior. AdipoRon significantly decreased the p-NF-κB/NF-κB ratio and IL-1β levels, thereby validating its strong anti-inflammatory effects. AdipoRon interrupts the neuroinflammatory cascade driving depression under sleep deprivation by suppressing NF-κB–mediated cytokine production. Furthermore, the activation of adiponectin receptor pathways contributes to neuroprotection and reinstates neural homeostasis.

Conclusion : The findings indicate that AdipoRon mitigates SR-induced depressive behaviors through the downregulation of NF-κB signaling and a reduction in cytokine-driven neural stress. This underscores the important function of inflammation management in mood regulation and reinforces AdipoRon's therapeutic promise in addressing mood disorders resulting from extended sleep deprivation.

Keywords : Depression, AdipoRon, Sleep restriction, NF-κB, IL-1β, Neuroinflammation, Prefrontal cortex

Count: 95
Abstract ID: 268
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Oral

Early sensory slowing with preserved control: anxiety and proactive processing in adults who stutter

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Background and Aim : Adults who stutter (AWS) often present with elevated trait anxiety, yet its influence on the neural dynamics of action preparation and execution is unclear. We examined how Beck Anxiety Inventory (BAI) scores relate to early (N1) and later (N2, P3) event-related potentials (ERPs) elicited during a visual stop-signal task in AWS and fluent controls.

Methods : Forty adults (22 AWS, 18 controls) performed the task while a 32-channel EEG was recorded. ERPs were extracted for proactive cues, reactive cues, and go trials. A between-group comparison was conducted, and hierarchical regressions tested whether BAI predicted ERP amplitudes beyond the effects of age and group.

Results : Groups were age-matched, but AWS reported markedly higher trait anxiety. The only robust ERP difference was a significant delay in proactive cue N1 latency at the left occipital site (O1) in AWS, which remained significant after covarying for anxiety. Higher BAI uniquely predicted a shallower (less negative) N1 amplitude at the right occipital site O2, explaining an additional 14 % of its variance; BAI did not account for frontal N2 amplitude once age and group were included. No other N2 or P3 measures, nor go reaction times, differed between groups.

Conclusion : AWS exhibit an anxiety-independent slowing of early visual processing (proactive cue N1 latency), whereas heightened trait anxiety selectively attenuates occipital N1 amplitude. Preservation of 'go' trial performance and later N2/P3 components suggests that downstream response execution remains intact—possibly via compensatory cortical mechanisms that offset the initial sensory bottleneck.

Keywords : Stuttering; Trait Anxiety; ERP; Visual Stop Signal Task

Count: 96
Abstract ID: 467
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

The Effects of Postnatal Probiotic Supplementation on Oxidative Stress Markers in Prenatal Stress male rats

Submission Author: Fatemeh Aghighi

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Background and Aim : Early-life stress, including both physical and emotional stressors, can induce persistent physical and mental health effects in animal models and humans [1]. Several studies have reported stress-mediated alterations in antioxidant enzymes and reactive oxygen species levels in animal stress models; the changes that contribute to impairments in the nervous system structure and function [2, 3]. Oxidative stress, accompanied by changes in neurotransmitter synthesis, neurotrophic factors, and brain protein homeostasis leads to neurodegenerative and psychiatric disorders [4]. Since oxidative stress plays a pivotal role in stress-induced pathogenesis, suppressing oxidative stress by activating antioxidant defenses could be a crucial mechanism for improving stress-related disorders. Emerging research highlights the interaction between gut microbiota and stress. Multiple studies have found that certain probiotics, such as Lactobacillus and Bifidobacterium species, possess significant antioxidant properties [5, 6]. Although the exact mechanisms underlying the antioxidant effects of probiotics are not fully understood, several hypotheses suggest that probiotics may exert these effects by elevating antioxidant enzyme levels, scavenging reactive oxygen species, and modulating the gut microbiota [7]. This study aimed to evaluate effect of postnatal probiotic supplementation on oxidative stress markers in Prenatal Stress male rats.

Methods : Forty male Wistar rats were divided to four groups including rats exposed to chronic unpredictable stress from fetal days 1-21 were administered either saline (CUS+SAL) or a probiotic mixture (CUS+PRO) for 30 days after weaning. Control groups (CON+SAL and CON+PRO) were also included. Finally serum markers for oxidative stress were measured.

Results : The MDA level in the serum of the CUS+SAL group was significantly higher than that of the CON+SAL group (P<0.0001). The serum TAC level (P<0.05), GSH level (P<0.05), and SOD activity (P<0.0001) were significantly lower in the CUS+SAL group than in the CON+SAL group. Moreover, treatment with probiotics significantly decreased the MDA level in the CUS+PRO group (P<0.001). The TAC level (P<0.001), GSH level (P<0.0001), and SOD activity (P<0.0001) in the CUS+PRO rats were significantly elevated by treatment with probiotics.

Conclusion : The prenatal chronic unpredictable stress led to a decrease in antioxidants and an increase in oxidants, but probiotic treatment improved these changes.

Keywords : Prenatal stress; Probiotic; Stress oxidative

Count: 97
Abstract ID: 522
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

Maternal variable stress affected maternal nursing, oxidative status and glucose metabolism in male pubertal rats

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Background and Aim : Most disorders including glucose metabolism disorders, are induced by exposure to stressful events during prenatal period. Since stress can lead to oxidative stress and ultimately damage to various organs such as the hormonal system, this study aimed to determine the effect of prenatal stress on oxidative status and glucose homeostasis
Methods : Pregnant female rats were randomly divided into two groups of stress and non-stress. Animals in the stress group received stress from day 14 until the end of pregnancy. After stress exposure, blood was drawn from the tail of dams to determine plasma corticosterone concentration. Moreover, during first 7 days of lactation period, the dams nursing behavior was examined. Blood samples of male offspring of both groups were taken at the time of puberty (postnatal day, PND, 45) to measure plasma concentrations of corticosterone, MDA, glucose and insulin. Moreover, HOMA-IR (insulin resistance index) was calculated
Results : Prenatal stress increased plasma corticosterone concentrations in dams and also decreased the total number of the breast feeding. However, the plasma corticosterone levels did not alter in adult offspring. Prenatal variable stress also increased the plasma MDA concentration of these offspring, whereas the plasma insulin concentration decreased with unchanged glucose level. In addition, the HOMA-IR index showed a significant decrease in adult offspring of the stress group.
Conclusion : From the results of present study, it seems that prenatal stress by altering the intrauterine environment, possibly by increasing plasma concentrations of maternal stress hormones and subsequent maternal nursing, could interfere with the programming of different systems of the developing fetus and/or neonate. In this regard, the elevated corticosterone in the stressed dams programed developing redox and metabolic systems and made a basis for glucose metabolism impairment during puberty.
Keywords : Maternal stress, Corticosterone, Oxidative stress. Insulin, Glucose

Count: 98
Abstract ID: 307
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

Vitamin D rescues Chronic unpredictable mild stress-induced neural apoptosis in the various brain regions in rat

Submission Author: Sara Abolfazli

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Background and Aim : Chronic stress is one of the major causes of psychiatric and neurodegenerative diseases. Depression and cognitive disability are among the main damages that are followed by neural apoptosis in various regions of the brain. The role of vitamin D in mitigating behavioral and neural damage associated with stress and depression in the hippocampus and prefrontal cortex (PFC) has been extensively studied. Since other brain regions, such as the amygdala and cerebellum, are affected by chronic stress, this study aimed to investigate the effect of vitamin D on cellular changes in these four regions in relation to chronic unpredictable mild stress (CUMS) in rats.

Methods : Male Wistar rats were exposed to 4 weeks of CUMS. Vitamin D-treated (5 and 10 microgram/Kg) rats began receiving vitamin D one week before the stress. After 4 weeks, the hippocampus, PFC, amygdala, and cerebellum were analyzed for Caspase 3 by Western blotting.

Results : High Caspase 3 protein expression was detected in all four stressed brain areas ($P<0.0001$). Vitamin D 10(microg/kg) significantly inhibited neural damage, specially in PFC.

Conclusion : Vitamin D with a higher dose markedly inhibited CUMS-induced neural damage in all four brain areas, suggesting that it may act as a highly efficient medicine in protecting the brain against the devastating effects of long-term stress.

Keywords : chronic stress; vitamin D; apoptosis; brain

Count: 99
Abstract ID: 815
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

The impact of high-fat-high-fructose diet during perinatal on the occurrence of depressive like behaviors due to re-exposure to that diet combined with restraint stress in adult male rat offspring

Submission Author: Haniyeh Soltani

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Background and Aim : Consumption of high-calorie diets and exposure to psychological stress, and the occurrence of behavioral disorders, especially depression, are increasingly prevalent in today's societies. Accordingly, in this study the effect of a high-fat-high fructose diet (HFFD) during the perinatal period on depressive-like behaviors in response to re-exposure to this diet along with restraint stress, was investigated in adult male rat offspring.

Methods : Female Wistar rats were divided into two groups receiving a HFFD and a normal diet (C), from two weeks before mating and throughout pregnancy and lactation. After weaning, the male offspring of both groups consumed normal diet for four weeks, then the offspring were randomly divided into 4 subgroups based on the type of diet and stress (C.C-NS, C.C-S, H.H-NS, H.H-S) and continued to consume their diets for 8 weeks. Then, while the consumption of the diets continued, the animals in the stress groups received restraint stress for 3 consecutive days. Then, to assess depressive like behaviors, forced swim (FST) and

sucrose splash (SPT) tests were performed. In FST, latency to first immobility, the frequency and percentage of immobility duration were measured and in SPT, the latency to first grooming, the frequency and duration of grooming, during the five minutes of the tests were analyzed.

Results : The forced swim test showed that HFFD exposure reduced latency and frequency of immobility in non-stressed animals but increased immobility time overall, indicating enhanced depressive-like behavior. Stress decreased latency to first immobility in controls, while HFFD partially reduced this effect. Although stress and HFFD had little impact on immobility duration percentage, HFFD-exposed offspring exhibited lower mobility time, with the H.H-S group showing the lowest values, suggesting synergistic depressive effects of stress and HFFD. Grooming analysis revealed that HFFD exposure reduced grooming frequency and significantly decreased grooming time compared to controls, regardless of stress. Stress alone had minimal impact, but combined HFFD and stress exposure resulted in the lowest grooming duration. Additionally, HFFD increased latency to first grooming, indicating delayed initiation of self-care behavior, while stress slightly shortened this latency without normalizing it to control levels.

Conclusion : Overall, perinatal and adult exposure to HFFD induced marked depressive-like behaviors in offspring, more robustly than restraint stress. Combined HFFD and stress could produce the most severe behavioral impairments, indicating additive effects. HFFD also reduced self-care behaviors, with decreased grooming time and delayed initiation. These findings suggest that poor diet likely increase vulnerability to stress-related mood disturbances and emphasize the critical role of maternal and early-life nutrition in shaping susceptibility to depression-like states.

Keywords : high-fat-high fructose diet, perinatal, depressive-like behaviors, restraint stress

Count: 100
Abstract ID: 954
subject: Emotion, Motivation and Behavior: Stress and the Brain (Stress-modulated Pathways, Stress and Cognition, Stress Related Disorders)
Presentation Type: Poster

Neurobiology of Stress and Decision-Making in the Workplace: From Cortisol to Cognitive Control

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Conclusion : Objective: This review investigates the neurobiological mechanisms through which stress influences decision-making in workplace environments, integrating evidence from neuroscience, endocrinology, and organizational psychology. The aim is to identify how chronic stress alters brain circuits involved in cognitive control, emotion regulation, and leadership decisions. Methods: A narrative review was conducted using PubMed, Scopus, and Web of Science databases (2015–2025) with keywords “stress,” “cortisol,” “decision-making,” “prefrontal cortex,” “amygdala,” “HPA axis,” and “workplace neuroscience.” Studies involving neuroimaging, hormonal assays, and behavioral assessments under stress conditions were included. Findings: • HPA axis and cortisol: Activation of the hypothalamic–pituitary–adrenal (HPA) axis during stress increases cortisol secretion, which transiently enhances alertness but impairs decision-making when chronically elevated (McEwen et al., Nat Neurosci, 2020). • Prefrontal–amygdala balance: Chronic stress disrupts prefrontal cortex (PFC) regulation over the amygdala, leading to emotional reactivity and reduced cognitive flexibility (Arnsten, Nat Rev Neurosci, 2021). • Reward and motivation: High cortisol reduces dopaminergic activity in the ventral striatum, decreasing motivation and reward sensitivity—key factors in workplace engagement (Pruessner et al., Front Behav Neurosci, 2019). • Leadership and cognitive control: Leaders under sustained stress show decreased activation in the dorsolateral PFC, leading to impulsive or risk-averse managerial choices (Porcelli & Delgado, Front Hum Neurosci, 2022). • Protective factors: Mindfulness, social support, and adaptive coping enhance PFC–amygdala connectivity and normalize HPA axis function, supporting better decision-making under pressure (Creswell et al., PNAS, 2020). Conclusion: Stress profoundly alters brain networks involved in emotion and cognition. Chronic activation of the HPA axis and cortisol dysregulation impair executive functions essential for sound workplace decisions. Understanding these neurobiological pathways can guide the design of stress-management interventions that strengthen cognitive control and improve leadership performance.

Keywords : Stress; cortisol; decision-making; HPA axis; prefrontal cortex; workplace neuroscience

Count: 101
Abstract ID: 976
subject: Emotion, Motivation and Behavior: Behavioral Pharmacology
Presentation Type: Poster

Assessing the effect of minocycline in the enhancement of inflammatory pain following adolescent nicotine administration

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Background and Aim : Pain is a multidimensional phenomenon and its perception varies across individuals. A history of substance exposure, from tobacco to alcohol and opioids, can alter both pain perception and cognitive functions. Adolescence represents a critical period of neurodevelopment characterized by heightened risk-taking behaviors and increased vulnerability to substance abuse. Nicotine, often the first drug encountered during adolescence, exerts long-lasting cognitive and behavioral effects and enhances later drug sensitivity. Animal studies have shown that adolescent nicotine exposure augments nociceptive behaviors in adulthood, particularly in inflammatory pain models such as the formalin test. Emerging evidence shows that microglia may be involved in these effects. The present study examined whether concurrent administration of nicotine and the microglial inhibitor minocycline during adolescence could prevent nicotine-induced enhancement of inflammatory pain in adulthood.

Methods : Male Wistar rats were divided into four groups. Two groups received subcutaneous nicotine injection (0.63 mg/kg) from postnatal day 28 to 41, while one of these groups also received minocycline in drinking water (0.533 mg/ml), starting one day before and continuing until one day after nicotine administration. Two control groups received subcutaneous saline during the same period, with one group also receiving minocycline. From postnatal day 43 to 64 all rats remained drug-free. Subsequently, inflammatory pain was assessed using the formalin test (2% formalin, 50 µL, injected into the hind paw). Pain-related behaviors were evaluated based on nociceptive scores and paw-licking duration across the acute and tonic phases of the test.

Results : Adolescent nicotine exposure significantly increased nociceptive scores during the tonic phase of the formalin test. Co-administration of nicotine and minocycline, however, prevented this increase, with no significant elevation in pain scores observed in either phase. Minocycline alone reduced pain behaviors during the tonic phase compared to the nicotine-group. Similarly, nicotine prolonged paw-licking duration during the tonic phase, while minocycline co-treatment reversed this measure to saline levels. Nicotine also shortened the interphase duration, an effect abolished by minocycline.

Conclusion : These findings indicate that minocycline attenuates the long term nociceptive effects of adolescent nicotine exposure on inflammatory pain sensitivity in adulthood. The results support microglial involvement as a potential strategy for preventing hyperalgesia associated with early nicotine exposure.

Keywords : Nicotine; Adolescence; Inflammatory pain; Microglia

Count: 102
Abstract ID: 818
subject: Emotion, Motivation and Behavior: Behavioral Pharmacology
Presentation Type: Poster

Protective effect of fecal microbiota transplantation in d-galactose induced aged mice

Submission Author: Fatemeh Mokhtari naseri

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Background and Aim : Aging is a complex biological process characterized by gradual decline in physiological functions and increased vulnerability to various diseases. Recent research has highlighted the significant role of the gut microbiota in modulating health and aging processes. Alterations in microbiota composition, often observed with advancing age, have been associated with increased inflammation, cognitive decline, and decreased resilience. Emerging evidence suggests a connection between gut microbiota and social behaviors. Studies indicate that gut-brain axis interactions can influence emotional regulation and social cognition. so, microbiota modulation has shown potential in affecting neurobehavioral outcomes, implying that microbiota-targeted interventions could modulate social and emotional responses

Methods : This study utilized eight-week-old male BALB/c mice. The experimental design included two phases. Phase 1: Six groups were established: Control: Received intraperitoneal injections of normal saline for 8 weeks. D-galactose: Administered D-galactose at a dose of 200 mg/kg via intraperitoneal injection for 8 weeks. At the end of this period, fecal samples were collected for subsequent microbiota transplantation. Phase 2: Four groups were formed based on the treatments: Control-FMT: Similar to the control group, but received fecal microbiota transplantation (FMT) from control mice for one week. D-galactose-FMT: Similar

to the D-galactose group, but received FMT from D-galactose mice. Control-FMT: Control mice receiving fecal microbiota from control donors. D-galactose-FMT (D-galactose feces): D-galactose mice receiving fecal microbiota from D-galactose donors. Prior to microbiota transplantation, the the animals underwent a processing step to eliminate approximately 90% of the existing microbiota, ensuring effective colonization by the transplanted microbiota.

Results : : This study's data show that D-galactose injection impaired working and spatial memory in Y-maze and Morris water maze tests, which were largely improved following fecal microbiota transplantation, indicating microbiota's role in reversing cognitive deficits., highlighting microbiota's role in reversing age-related cognitive decline. Overally our study suggest that gut microbiota modulation influences neuroplasticity and cognitive functions through gut-brain axis interactions.

Conclusion : in our study data showed that, the transplantation of fecal microbiota significantly improved spatial and working memory in subjects. Behavioral assessments using the Y-maze and Morris water maze demonstrated enhanced cognitive performance post-intervention.

Keywords : Aging, microbiota, FMT

Count: 103
Abstract ID: 509
subject: Emotion, Motivation and Behavior: Behavioral Pharmacology
Presentation Type: Poster

Modulation of Anxiety-Like Behaviors via Cholinergic-Dextromethorphan Synergy: A Preclinical Study

Submission Author: Mahdiyeh Safary

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Background and Aim : Anxiety disorders are among the most prevalent neuropsychiatric conditions, affecting millions worldwide and significantly impairing quality of life. Despite the availability of various pharmacological treatments, many patients experience limited efficacy or adverse effects, underscoring the need for novel therapeutic strategies. The cholinergic system, particularly its nicotinic and muscarinic receptors, plays a crucial role in modulating emotional and cognitive functions. Recent evidence suggests that interactions between the cholinergic and catecholaminergic systems may influence anxiety-related behaviors. Dextromethorphan, a widely used antitussive agent, has demonstrated neuroprotective and mood-modulating properties, including potential anxiolytic effects. However, the mechanisms underlying its impact on anxiety remain unclear. This study investigates the role of central cholinergic receptors in mediating the anxiolytic effects of dextromethorphan, aiming to clarify how nicotinic and muscarinic antagonists modulate its behavioral outcomes in a preclinical mouse model.

Methods : A total of 126 adult male NMRI mice were used in this preclinical study. Animals were anesthetized and implanted with intracerebroventricular cannulas using a stereotaxic apparatus, following coordinates from the Paxinos-Franklin atlas. After a one-week recovery period, mice were randomly assigned to pharmacological groups receiving intraperitoneal injections of dextromethorphan (15, 30, or 60 mg/kg) and intracerebroventricular administration of cholinergic antagonists—scopolamine or mecamylamine—at both effective

and subthreshold doses. Anxiety-like behaviors were assessed using the elevated plus maze (EPM), measuring open arm time (OAT) and open arm entries (OAE). Locomotor activity was evaluated in the open field test to rule out nonspecific motor effects. Data were analyzed using one-way ANOVA followed by Tukey's post hoc test, with significance set at $P<0.005$.

Results : Dextromethorphan administration at doses of 30 and 60 mg/kg significantly increased open arm time (OAT) and open arm entries (OAE) in the elevated plus maze, indicating anxiolytic-like effects ($P<0.005$). Similarly, intracerebroventricular administration of scopolamine (12 µg/mouse) and mecamylamine (30 µg/mouse) produced comparable anxiolytic responses. Interestingly, co-administration of a subthreshold dose of scopolamine (0.3 µg/mouse) with dextromethorphan (30 or 60 mg/kg) attenuated its anxiolytic effect, as evidenced by reduced %OAT and %OAE ($P<0.001$). In contrast, a subthreshold dose of mecamylamine (5 µg/mouse) enhanced the anxiolytic effect of an otherwise ineffective dose of dextromethorphan (15 mg/kg), suggesting a synergistic interaction ($P<0.005$). Isobologram analysis confirmed this synergy between dextromethorphan and mecamylamine ($P<0.001$). Locomotor activity, assessed via the open field test, showed no significant differences across treatment groups, indicating that the observed behavioral changes were specific to anxiety-related responses rather than general motor effects.

Conclusion : This study demonstrates that dextromethorphan exerts significant anxiolytic effects through modulation of central cholinergic pathways, particularly via nicotinic receptor inhibition. The synergistic interaction with mecamylamine highlights a promising pharmacological strategy for anxiety management. These findings support further investigation into cholinergic-dextromethorphan combinations as potential therapeutic agents for anxiety disorders.

Keywords : Anxiety, Dextromethorphan, Cholinergic system, Mecamylamine, Scopolamine

Count: 104
Abstract ID: 510
subject: Emotion, Motivation and Behavior: Behavioral Pharmacology
Presentation Type: Poster

Synergistic Effects of Dextromethorphan and Cholinergic Antagonists on Depression-like Behavior in Mice

Submission Author: Mahdiyeh Safary

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Background and Aim : Depression is a debilitating mood disorder characterized by persistent sadness, loss of interest, and cognitive impairment, affecting millions globally. Despite the availability of antidepressant therapies, many patients experience inadequate responses or adverse effects, necessitating the exploration of novel pharmacological strategies. The cholinergic system, particularly muscarinic and nicotinic receptors, plays a key role in mood regulation and has been implicated in the pathophysiology of depression. Dextromethorphan, a noncompetitive NMDA receptor antagonist with sigma-1 receptor activity, has shown promise in mood modulation and neuroprotection. However, its interaction with the cholinergic system in the context of depression remains poorly understood. This study aimed to investigate the role of central cholinergic receptors in mediating the antidepressant-like effects of dextromethorphan in a mouse model, focusing on the potential synergistic effects of cholinergic antagonists.

Methods : A total of 126 adult male NMRI mice were used in this preclinical study. Animals were anesthetized and implanted with intracerebroventricular cannulas using a stereotaxic device, following the Paxinos-Franklin atlas. After a one-week recovery period, mice were randomly assigned to treatment groups receiving intraperitoneal injections of dextromethorphan (15, 30, or 60 mg/kg) and intracerebroventricular administration of cholinergic antagonists—scopolamine or mecamylamine—at both effective and subthreshold doses. Depression-like behaviors were assessed using the forced swimming test (FST),

measuring immobility time as a marker of behavioral despair. Locomotor activity was evaluated in the open field test to control for nonspecific motor effects. Statistical analysis was performed using one-way ANOVA followed by Tukey's post hoc test, with significance set at $P<0.005$.

Results : Dextromethorphan at 60 mg/kg significantly reduced immobility time in the FST, indicating an antidepressant-like effect ($P<0.001$). Scopolamine at both 2 and 12 µg/mouse also produced significant reductions in immobility ($P<0.001$), while mecamylamine at 30 µg/mouse showed moderate efficacy ($P<0.005$). Co-administration of a subthreshold dose of scopolamine (0.3 µg/mouse) with dextromethorphan (15 or 30 mg/kg) enhanced its antidepressant effect, further reducing immobility time compared to dextromethorphan alone ($P<0.001$). In contrast, a subthreshold dose of mecamylamine (5 µg/mouse) did not significantly alter the antidepressant effect of dextromethorphan ($P>0.005$). Isobologram analysis revealed a synergistic interaction between dextromethorphan and scopolamine in modulating depression-like behaviors ($P<0.001$). Locomotor activity remained stable across all groups, confirming that behavioral changes were specific to mood-related effects.

Conclusion : These findings suggest that dextromethorphan's antidepressant-like effects are mediated through muscarinic receptor inhibition within the cholinergic system. The observed synergy with scopolamine highlights a promising therapeutic avenue for enhancing antidepressant efficacy. Further research is warranted to explore the clinical relevance of cholinergic-dextromethorphan combinations in mood disorder treatment.

Keywords : Depression, Dextromethorphan, Cholinergic system, Scopolamine, Mice

Count: 105
Abstract ID: 908
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

Altered Expression of Neuroligin-2 and BDNF in the Amygdala Correlates with Anxiety-like Behavior in a Tinnitus Rat Model

Submission Author: Zeinab Akbarnejad

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Background and Aim : Tinnitus is a prevalent condition often accompanied by anxiety, yet the neurobiological mechanisms linking the two remain unclear. Neuroligin-2 (NLGN2), a synaptic adhesion protein, has been implicated in anxiety regulation. Brain-Derived Neurotrophic Factor (BDNF) also plays a critical role in neurotransmission and emotional behavior. This study explores whether alterations in NLGN2 and BDNF expression in the amygdala are associated with anxiety-like behavior in a rat model of salicylate-induced tinnitus.

Methods : Tinnitus was induced in adult rats via sodium salicylate administration. Confirmation of tinnitus was achieved using gap-prepulse inhibition of the acoustic startle (GPIAS) and pre-pulse inhibition (PPI) tests. Anxiety-like behavior was assessed using the Elevated Plus Maze (EPM) and Open Field Test (OFT). Amygdala tissue samples were collected and analyzed for NLGN2 and BDNF expression using molecular techniques.

Results : Rats with confirmed tinnitus exhibited significantly increased anxiety-like behavior compared to controls. Molecular analysis revealed reduced BDNF levels and altered Neuroligin-2 expression in the amygdala, suggesting a neurochemical imbalance linked to emotional dysregulation.

Conclusion : This study provides the first evidence that changes in NLGN2 and BDNF expression within the amygdala may mediate tinnitus-related anxiety. These findings highlight potential molecular targets for therapeutic intervention in tinnitus-associated emotional disorders.

Keywords : Tinnitus, Anxiety-like behavior, Neuroligin-2, BDNF, Amygdala

Count: 106
Abstract ID: 911
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

Faster to Love, Slower to Reject: Behavioral Dynamics of Beauty and Ugliness Judgments

Submission Author: Seyed Hamid Daliri

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Background and Aim : The perception of beauty and ugliness plays a crucial role in human interaction, social decision-making, and emotional experience. However, temporal and individual differences in the accurate judgment of these visual categories—collectively reflecting aesthetic sensitivity—remain underexplored, especially in the context of ugliness. This study investigated the behavioural dynamics of aesthetic judgments. Sixty adult participants (aged 18–55) viewed 54 beautiful and 58 ugly images generated by artificial intelligence. Reaction times (RTs) and accuracy of categorizing each image as beautiful or ugly were recorded. Results showed that beautiful images were processed significantly faster than ugly ones ($t(59) = 3.923$, $p < .001$), and with higher judgment accuracy ($t(59) = 2.38$, $p = .020$). A significant positive correlation was found between aesthetic judgment accuracy and reaction time for beautiful images ($r = .329$, $p = .010$). No significant effects were observed for age, gender, or most psychological traits, except for a negative correlation between paranoid tendencies and accuracy in judging ugly stimuli. These findings suggest that beauty and ugliness may be processed not only perceptually but also behaviorally in distinct ways. The results have implications for cognitive intervention design, the analysis of social biases, and interdisciplinary research bridging psychology and social neuroscience.The interplay between beauty and ugliness in social judgments highlights the importance of aesthetic sensitivity in shaping human interactions and decisions.

Methods : Sixty adult participants (22 females, 38 males, aged 18–55) evaluated 112 AI-generated images (54 beautiful, 58 ugly) across categories including faces, animals, flowers, food, and objects. Reaction times and judgment accuracy were recorded. Psychological traits were assessed using the SCL-90-R questionnaire.

Results : Participants responded significantly faster to beautiful images ($M = 65.37$ ms) than to ugly ones ($M = 80.53$ ms), $t(59) = 3.92$, $p < .001$, Cohen's $d = 0.51$. Accuracy was also

higher for beautiful images (M = 20.07 vs. 19.32), t(59) = 2.38, p = .020, d = 0.31. A significant positive correlation was found between reaction time and accuracy for beautiful stimuli (r = .329, p = .010), suggesting more deliberate processing. No significant correlations were found for ugly images. Age and gender showed limited effects, while a negative correlation emerged between paranoia (SCL-90) and judgment accuracy for ugliness (r = –.266, p = .040).

Conclusion : These findings reveal distinct behavioral dynamics in the processing of beauty versus ugliness. Beauty is processed faster and more accurately, whereas ugliness may involve more cognitive hesitation and emotional ambiguity. Such asymmetries offer insights into neural and psychological mechanisms of aesthetic experience and may inform future interventions in social cognition and affective neuroscience.

Keywords : Aesthetic Judgment, Reaction Time, Beauty and Ugliness, Cognitive Processing

Count: 107
Abstract ID: 802
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Oral

Emotion Recognition Using EEG Signal processing and Machine learning Approach

Submission Author: Alireza Fallahi

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Background and Aim : Emotions are an essential element of human life, influencing how we make decisions, maintain focus, learn, and communicate. Understanding emotional states is important not only in neuroscience and psychology but also in the development of intelligent systems designed to enhance human–machine interaction. Unlike facial expressions or speech signals, which can be consciously controlled, electroencephalogram (EEG) recordings directly represent brain activity, offering a more reliable window into human emotions. In the dimensional emotion model, emotional experiences are commonly mapped along two axes—Arousal and Valence—that align with the continuous and dynamic nature of EEG signals. However, EEG data are often complex and noisy, and emotional fluctuations over time make analysis difficult. Thus, selecting the most informative features and relevant EEG channels is a critical step toward improving the accuracy of machine learning–based emotion recognition models.

Methods : This study employed the DEAP dataset, which provides EEG recordings from 32 channels. To reduce computational cost and emphasize the most emotionally relevant data, 14 channels showing the strongest correlation with emotional responses were selected. The signals were preprocessed through noise removal, bandpass filtering (4–45 Hz), and correction for motion artifacts. Continuous emotion labels were then divided into two levels—high and low—to simplify classification. Four categories of features—statistical, spectral, fractal, and wavelet—were extracted to represent temporal, frequency, and nonlinear patterns within the EEG data.Feature selection was carried out using the Binary Grey Wolf Optimizer (BGWO), a metaheuristic method inspired by the cooperative hunting strategy of grey wolves. This approach filtered out redundant information and retained the most discriminative features for classification. The XGBoost algorithm was then applied as the final classifier for recognizing emotional states along the Arousal and Valence dimensions. Model performance was evaluated using 5-Fold Cross Validation, and hyperparameters were tuned through grid search to ensure robust results.

Results : The results demonstrated that applying the BGWO algorithm improved classification performance in both emotional dimensions. For Arousal, the average accuracy rose from approximately 62% to 65–66%, while for Valence, it increased from about 70% to 76–78%. These improvements show that optimized feature and channel selection enabled the model to capture more meaningful emotional patterns while minimizing noise and redundancy. Overall, the proposed framework achieved higher accuracy, consistency, and generalization than the baseline model without feature selection.

Conclusion : This study proposes a hybrid framework for EEG-based emotion recognition, combining metaheuristic feature selection with advanced classification. The integration of BGWO and XGBoost enhanced model accuracy and robustness by focusing on key EEG features and discarding less relevant data. This approach provides a practical and efficient foundation for developing intelligent emotion recognition systems and for advancing our understanding of the neural mechanisms that underlie human emotions.

Keywords : Emotion recognition; EEG; Feature selection; Machine learning

Count: 108
Abstract ID: 362
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

The Effect of Acceptance and Commitment Therapy (ACT) on Quality of Life, Resilience, and Neuropsychological Capital in Adolescents

Submission Author: Shahideh Ghaarli

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4. Institute for Cognitive Science Studies, Tehran, Iran.

Background and Aim : This study investigated the effects of Acceptance and Commitment Therapy (ACT) on quality of life, resilience, and psychological capital in adolescents. A quasi-experimental design was employed, with 30 adolescents randomly assigned to either an experimental or control group. The experimental group received eight sessions of ACT. Results from multivariate analysis of covariance (MANCOVA) indicated that ACT significantly enhanced adolescents' quality of life, resilience, and psychological capital. These findings suggest that ACT is an effective intervention for promoting mental health and strengthening psychological resources in adolescents.

Methods : This study investigated the effects of Acceptance and Commitment Therapy (ACT) on quality of life, resilience, and psychological capital in adolescents. A quasi-experimental design was employed, with 30 adolescents randomly assigned to either an experimental or control group. The experimental group received eight sessions of ACT. Results from multivariate analysis of covariance (MANCOVA) indicated that ACT significantly enhanced adolescents' quality of life, resilience, and psychological capital. These findings suggest that ACT is an effective intervention for promoting mental health and strengthening psychological resources in adolescents.

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Conclusion : This study investigated the effects of Acceptance and Commitment Therapy (ACT) on quality of life, resilience, and psychological capital in adolescents. A quasi-experimental design was employed, with 30 adolescents randomly assigned to either an experimental or control group. The experimental group received eight sessions of ACT. Results from multivariate analysis of covariance (MANCOVA) indicated that ACT significantly enhanced adolescents' quality of life, resilience, and psychological capital. These findings suggest that ACT is an effective intervention for promoting mental health and strengthening psychological resources in adolescents.

Keywords : Stress, Emotion,, Insight, Quality of life, Healthcare workers

Count: 109
Abstract ID: 185
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

The Effectiveness of Cognitive Computer Games on Emotion Regulation in Gifted Children

Submission Author: Maryam Rouhani

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3. Professor, Department of Electrical and Computer Engineering, University of Tehran, Tehran, Iran.

Background and Aim : Gifted children are at risk for emotion dysregulation as indicated in the literature and in practice. The purpose of this study was to examine the effect of cognitive computer games on emotion regulation in gifted children.

Methods : This research employed a quasi-experimental design with a pre-test and post-test and two groups: experimental and control. The statistical population included gifted children in boys' schools in Shahriar city. Using convenience sampling, 24 gifted students, how were diagnosed as gifted based on their academic records, were divided into two groups randomly: experimental (12 participants) and control (12 participants). The experimental group participated in 12 sessions of 20-minute cognitive computer game intervention ("Spaceship of Emotions"), while the control group did not receive any specific training. Emotion regulation was assessed using the Shields and Cicchetti Inventory (1997).

Results : Data were analyzed using one-way ANCOVA, with assumptions of normal data distribution (Kolmogorov-Smirnov and Shapiro-Wilk tests) and homogeneity of variances (Levene's test) confirmed. Results indicated that the mean scores of emotion regulation in the experimental group significantly increased after the intervention (Emotion Regulation: $F = 14.0$, $P < 0.01$. Instability/negativity, as a subcomponent of emotion regulation, decreased but did not reach the expected level of significance ($F = 3.92$, $P = 0.10$).

Conclusion : These findings demonstrated that cognitive computer games are generally effective tools for improving emotion regulation in gifted children. It is recommended that this cognitive computer game would be used in the gifted curriculum for those gifted children who need assistant in emotion regulation and also future studies investigate the effects of longer cognitive computer interventions.

Keywords : Cognitive computer games, emotion regulation, gifted children

Count: 110
Abstract ID: 17
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

Dimension health locus of control with happiness in students

Submission Author: Maryam Abedini

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Background and Aim : Happiness is one of the important and influential variables throughout the life of students.This study aimed to determine the relationship between health locus of control with happiness in college students.

Methods : This study was Descriptive within the framework of a correletion plan. The sample consisted of 100 undergraduate students Talesh PNU 1402-1403 academic years who were selected randomly and completed health locus of control scale and Oxford Happiness Questionnaire. The data were analyzed by using descriptive indicators of mean and standard deviation and Pearson correlation coefficient and stepwise regression methods .

Results : The results showed that The relationship between internal locus of control with happiness was positive, but with external locus of control chance was negative. relationship others locus of control and happiness was not significant. Also, regression analysis showed that 54 percent of happiness are explained by internal locus of control.

Conclusion : The Based on the findings, we can conclude that the health locus of control, affect the level of students' happiness.

Keywords : Happiness, health locuse of control, students

Count: 111
Abstract ID: 971
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

Effectiveness of Mindfulness-Based Intervention on Behavioral Activation and Emotional Regulation in Individuals with Mild to Moderate Depression

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Background and Aim : Mild to moderate depression, despite its significant impact on behavioral activation and emotional regulation, often remains untreated as many individuals avoid seeking treatment due to stigma or reluctance to use pharmacotherapy because of potential side effects. This lack of engagement with conventional treatments highlights the need for accessible, non-pharmacological interventions like mindfulness-based practices. Mindfulness, which fosters present-moment awareness and non-judgmental observation, offers a promising alternative to enhance goal-directed behavior and emotional regulation. This study evaluates the efficacy of a mindfulness-based intervention in improving these domains among Iranian individuals with mild to moderate depression, addressing a critical gap in accessible mental health interventions.

Methods : A randomized controlled trial was conducted with 60 participants (aged 18-45) diagnosed with mild to moderate depression using the Beck Depression Inventory-II (BDI-II, scores 14-28). Participants were recruited from outpatient clinics in Tabriz and randomly assigned to either an 8-week mindfulness-based stress reduction (MBSR) group (n=30) or a waitlist control group (n=30). The MBSR program consisted of weekly 2-hour sessions incorporating mindfulness meditation, body scans, and yoga, alongside daily home practice. Outcomes were measured using the Behavioral Activation for Depression Scale (BADS) to assess goal-directed behavior, the Positive and Negative Affect Schedule (PANAS) to evaluate emotional regulation, and the Mindful Attention Awareness Scale (MAAS) to measure mindfulness levels. Data analysis was conducted using repeated-measures ANOVA and paired t-tests, with significance set at $p<0.05$.

Results : The MBSR group demonstrated significant improvements compared to the control group. BADS scores increased by 27% ($p=0.001$), reflecting enhanced behavioral activation. PANAS results showed a 32% rise in positive affect and a 24% reduction in negative affect ($p<0.001$), indicating better emotional regulation. Additionally, MAAS scores improved by 20% ($p=0.003$), signifying heightened mindfulness.

Conclusion : In conclusion, the mindfulness-based intervention significantly improved behavioral activation and emotional regulation in individuals with mild to moderate depression, offering a possible, side-effect-free alternative for those who avoid conventional treatments. These findings strongly support integrating mindfulness-based interventions into clinical practice in Iran, particularly for untreated populations with mild to moderate depression. Future research should explore long-term outcomes and scalability to broaden access to such interventions.

Keywords : Mindfulness-Based Stress Reduction (MBSR), depression, behavioral activation, emotional regulation

Count: 112
Abstract ID: 617
subject: Emotion, Motivation and Behavior: Motivation and Emotion
Presentation Type: Poster

Neuroticism and extraversion interact in shaping emotions: a clustering and machine learning study

Submission Author: Kimia Kanani

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Background and Aim : Personality is closely associated with emotional experiences. Extraverts are commonly considered to be assertive, outgoing and enthusiastic people. People with high level of neuroticism (N) is known for variability in mood, feeling down and a negative attitude toward the world. It is also referred to as negative emotionality. Previous studies have investigated the effects of extraversion (E) and N on the emotions separately, but the interactive effect of these traits is still unclear. This study aimed to examine the relationship between N and E and how these traits interact in shaping emotional outcomes, when one trait is dominant in personality, compared to when they are equally strong?

Methods : Data from 1,197 young adults from the HCP dataset were analyzed. Participants completed 17 emotion-related questionnaires and the short version of the NEO personality inventory. First, the relationship between N and E was calculated using Pearson correlation. Then, Participants were clustered with k-means algorithm based on N and E scores, with Calinski-Harabasz approach used to optimize number of clusters. Groups were compared pairwise using permutation t-test to test emotional differences across groups with FDR correction to reduce type one error. Then, using a Gaussian regression machine learning (ML) we examined whether personality traits could be predicted from emotional scores by machine learning approaches. In this analysis, emotional scores were considered as inputs and each personality trait modeled separately as the output.

Results : A significant negative correlation of r = –0.34 was found between N and E (p ? 0.0001). K-means revealed 4 groups, 1) high N/low E; 2) low N/high E; 3) relatively low N/high E; and 4) relatively high N/low E. Significant emotional differences were observed in most group comparisons. Group1 differed from all others in all scales. Similar results we found for group 2 with 3 and 4. But group 3 and 4 showed several non-significant results in positive

and negative emotions. ML (Gaussian process regression) resulted R^2=0.59 with a validation root mean square error (RMSE) of 4.74 for N, whereas for E, the model achieved R^2=0.32 with an RMSE of 4.94.

Conclusion : The study showed that N and E are correlated, have some shared features and are not two entirely distinct traits. In addition, N and E interact in shaping emotions. Extreme profiles (group 1; high N/low E, group 2; low N/ high E) differed strongly, while middle groups (group 3, 4) showed many nonsignificant results, suggesting trait effects may neutralize each other. This highlights trait interaction over isolated effects. The result of machine learning revealed that high N exhibits more distinct emotional patterns. In contrast, E was predicted to a limited extent from emotions, suggesting other factors, like social motivation, may play a stronger role.

Keywords : personality; positive and negative emotions; Machine Learning

Count: 113
Abstract ID: 609
subject: Neuropsychiatry and Psychology: Evidence-Based Psychology
Presentation Type: Oral

The Relationship between Early Brain Processing and Heideggerian Concepts of "Being-in-the-World" and "Being-with": Evidence from ERP Studies

Submission Author: Alireza Farnam

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Background and Aim : This study explores the intersection of cognitive neuroscience and Heideggerian philosophy by examining how early brain processing relates to the concepts of "being-in-the-world" and "being-with." Heidegger posits that human experience is inherently social and cultural, emphasizing that individuals are always situated within historical and interpersonal contexts. Event-related potentials (ERP) provide a precise method to investigate pre-conscious and early sensory processing in response to social and cultural stimuli. This paper reviews key ERP components, including P1, P2, P300, N400, MMN, and ERN, demonstrating that the human brain automatically and pre-attentively processes socially relevant features such as faces, voices, and interaction cues. These neural findings provide biological evidence supporting Heideggerian notions of embedded sociality, indicating that humans inherently engage with their social and cultural environment from the earliest stages of perception. The integration of ERP research and phenomenological philosophy offers a novel framework to understand how early neural mechanisms underpin human social cognition.

Methods : A literature review of ERP studies was conducted, focusing on components P1, P2, P300, N400, MMN, and ERN. These components provide high temporal resolution measures of brain responses to visual, auditory, and language-related stimuli, capturing early, pre-conscious, and social-cognitive processing.

Results : ERP evidence indicates that the brain automatically detects socially relevant information such as human voices, facial expressions, and social cues, prior to conscious awareness. P1 and P2 reflect early sensory processing, P300 reflects attention allocation and stimulus evaluation, N400 is sensitive to semantic processing, MMN detects auditory deviations, and ERN monitors performance errors. Collectively, these findings show that

humans process social and cultural context from the earliest perceptual stages, supporting Heideggerian ideas of being inherently situated and socially engaged.

Conclusion : ERP research provides biological evidence that early brain processes are intrinsically linked to social and cultural experience. These findings reinforce the Heideggerian perspective that humans are fundamentally “being-in-the-world” and “being-with” others. Integrating ERP studies with existential phenomenology enhances our understanding of the neural underpinnings of social cognition and the inherently relational nature of human experience.

Keywords : Being-in-the-world, Being-with, Heidegger, Event-Related Potentials, Cognitive Neuroscience

Count: 114
Abstract ID: 312
subject: Neuropsychiatry and Psychology: Evidence-Based Psychology
Presentation Type: Oral

Original Gender-Independent Markers of Personality: A Mediation Analysis of Linear and Nonlinear EEG Features

Submission Author: Amirhossein Adhami

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Background and Aim : Personality traits can be assessed using various models. For instance, the NEO(Big5) model is one of the prominent frameworks that provides the ability to assess individuals from five different perspectives, including neuroticism, extraversion, openness, conscientiousness, and agreeableness. Knowledge of an individual's personality profile in different situations helps to optimally select individuals for occupational or social contexts. however, inferring the personality type of an individual solely based on questionnaires can lead to errors. Therefore, providing an objective method for diagnosing personality types seems essential. In this article, a method is presented to correctly identify the neural correlates of individual's personality type. Previous research has demonstrated that linear metrics such as EEG signal power can well extract information pertaining the personality type.

Methods : In the present study, we employed both linear and nonlinear analysis of brain signals and then used machine learning algorithms to extract information related to personality. Both of these methods successfully classified individuals in 5 different traits with high accuracy (statistically significant). So that the signal power as a linear metric reached 81.2% and the entropy as a nonlinear metric reached 86.4% accuracy. Along with this analysis, examining demographic data, led us to conclude that the gender of the individuals has a statistically significant effect on some personality traits, such as conscientiousness and agreeableness. EEG signal further revealed that both linear and nonlinear characteristics are also dependent on gender in addition to personality type. This finding raises a critical question: is the association between EEG features and personality is mediated by gender or reflects a direct and intrinsic neural correlate of personality? In fact, we are looking for whether the originality of the coding of personality type in brain signals is done by linear features or nonlinearly? To address this, we used the mediation analysis method. This method determines whether the dependence between an extracted EEG feature and personality trait decreased or diminished if gender

information is added. If so, we conclude that the feature's effect on personality is mediated by gender and is not a direct effect.

Results : The results showed that while using entropy as feature, in lower frequencies more that 58% and in higher frequencies only 11% of the electrodes show a direct effect on personality, while when using linear features, only 20 % in lower frequencies and more than 47% of the electrodes in higher frequencies show a direct effect.

Conclusion : Thus, it can be asserted with statistical confidence that the power of the brain signal in higher frequencies and entropy (complexity) of EEG in lower frequencies can indicate the personality type of the individual without dependence on gender and can be introduced as a direct effect.

Keywords : personality; EEG; entropy; power

Count: 115
Abstract ID: 517
subject: Neuropsychiatry and Psychology: Schizophrenia
Presentation Type: Oral

Cerebrolysin ameliorates learning and memory impairments and depression in a ketamine-based animal model of schizophrenia

Submission Author: Leila Hosseini

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Background and Aim : Cerebrolysin (CBL), a peptide prep from porcine brain tissue, is known for its neuroprotective and anti-inflammatory effects. This study aimed to assess CBL's therapeutic potential in a ketamine-induced mouse model of schizophrenia. Specifically, it examined its effects on anxiety- and depression-like behaviors, spatial memory, modulation of the tryptophan–kynurenine (TRP/KYN) pathway, and inflammatory responses in the prefrontal cortex (PFC).

Methods : Thirty-six male BALB/c mice were randomly assigned to three experimental groups: (1) control, (2) ketamine (Ket; 20 mg/kg/day, i.p, and (3) Ket + CBL (2.5 mL/kg/day, i.p). Anxiety-like behavior and locomotor activity were evaluated using the open field test, while depression-like behavior was assessed with the tail suspension test. Spatial memory performance was measured using the Lashley III Maze. Levels of KYN and TRP were quantified with enzyme-linked immunosorbent assay (ELISA). Expression of the inflammatory markers NLRP3 and IL-1β in the PFC was determined via Western blot analysis.

Results : The findings showed that CBL administration significantly reduced anxiety-like behaviors, decreased immobility time, and enhanced spatial learning and memory performance. Additionally, CBL treatment notably lowered KYN levels while increasing TRP concentrations in the PFC. Ketamine administration was linked to higher IL-1? expression; however, CBL did not significantly affect NLRP3 and IL-1? levels compared to the Ket group.

Conclusion : These results highlight the neuroprotective potential of CBL, suggesting that its beneficial effects may be attributed to modulation of the TRP/KYN pathway, along with improvements in spatial memory, anxiety-like, and depression-like behaviors in the ketamine-induced mouse model of schizophrenia.

Keywords : Memory ; Cerebrolysin ; Depression ; Anxiety ; Schizophrenia ; Neuroinflammation

Count: 116
Abstract ID: 887
subject: Neuropsychiatry and Psychology: Schizophrenia
Presentation Type: Oral

Pentoxifylline adjunct to risperidone for negative symptoms of stable schizophrenia: a randomized, double-blind, placebo-controlled trial

Submission Author: Kimia Kazemzadeh

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Background and Aim : Negative symptoms of schizophrenia represent an unmet therapeutic need for many patients in whom pentoxifylline may be effective in terms of its dopaminergic, anti-inflammatory, and cerebral blood flow–increasing properties. This study aimed to evaluate pentoxifylline as a therapeutic agent for improving negative symptoms of schizophrenia.

Methods : Chronic schizophrenia outpatients experiencing significant negative symptoms were randomly allocated to receive pentoxifylline 400 mg or matched placebo every 12 hours for 8 weeks. All patients were clinically stable as they had received risperidone for at least 2 months, which was continued. The participants were assessed using the Positive and Negative Syndrome Scale (PANSS), Hamilton Depression Rating Scale, Extrapyramidal Symptom Rating Scale, and side effect checklist.

Results : The patients' baseline characteristics were comparable between the groups. There was a significant time–treatment interaction effect on PANSS negative subscale scores (η =0.075), with the pentoxifylline group showing significantly greater reductions until weeks 4 (Cohen d = 0.512) and 8 (Cohen d = 0.622). Also, this group showed a significantly better

response by week 8. Other PANSS scores, Hamilton Depression Rating Scale scores, Extrapyramidal Symptom Rating Scale scores, and side effect frequencies were comparable between the groups. Pentoxifylline showed a nonsignificant higher remission of 37.1% compared with 14.7% in the placebo group.

Conclusion : Pentoxifylline was safely and tolerably beneficial for the primary negative symptoms of chronic schizophrenia.

Keywords : combination drug therapy, neuroimmunomodulation, psychosis, psychotic disorder, randomized controlled trial

Count: 117
Abstract ID: 742
subject: Neuropsychiatry and Psychology: Schizophrenia
Presentation Type: Poster

The Effect of Crataegus (Hawthorn) Extract on Anxiety-Like Behavior in a Ketamine-Induced Schizophrenia Model in Wistar Rats

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Background and Aim : This study aimed to evaluate the effect of Crataegus (Hawthorn) extract on anxiety-like behaviors in a ketamine-induced schizophrenia model in Wistar rats.

Methods : Male Wistar rats received ketamine (30 mg/kg, i.p.) for seven consecutive days. Twenty-four hours after the last injection, Crataegus extract (150 mg/kg, p.o.) was administered orally. One hour later, the Open Field Test (OFT) was performed to assess anxiety levels.

Results : Ketamine administration increased the time spent in the peripheral zone and reduced the time spent in the center of the arena, indicating elevated anxiety. Treatment with Crataegus extract significantly reversed these changes, as treated rats spent more time in the center zone, suggesting reduced anxiety. In addition, total locomotor activity decreased in the extract-treated group compared with the ketamine group, reflecting a mild sedative effect

Conclusion : The findings suggest that Crataegus extract, through its antioxidant and neurotransmitter-modulating properties, may alleviate ketamine-induced anxiety in rats and could be considered a potential herbal anxiolytic agent.

Keywords : Crataegus extract; Ketamine; Anxiety; Open Field Test (OFT), Wistar rats

Count: 118
Abstract ID: 690
subject: Neuropsychiatry and Psychology: Disorders of Neurobehavior
Presentation Type: Oral

The association between adenotonsillar hypertrophy and attention-deficit-hyperactivity disorder (ADHD) in children

Submission Author: Kimia Heidaritajadod

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Background and Aim : Respiratory disturbances like airway obstruction and asthma in children have been repeatedly suggested as potential risk factors for neuropsychiatric disorders like Attention Deficit Hyperactivity Disorder (ADHD). This study investigates the associations between the prevalence of adenotonsillar hypertrophy and the incidence of ADHD in children.

Methods : This case-control study was conducted on 184 children aged 4 to 11, consisting of 94 children with ADHD who were randomly selected from Ardabil's pediatric psychology clinic patients and 90 children without ADHD who were also randomly chosen from preschools and elementary schools, all of whom were assessed clinically according to DSM-IV guidelines by pediatric psychiatrists. Data were collected for this study using the standardized Bankage questionnaire and clinical assessments of tonsillar hypertrophy, as well as oropharyngeal examination. The collected data include symptoms of adenotonsillar hypertrophy, such as snoring, fatigue, breath-holding, and mouth breathing during sleep. Other indicators are an adenoid facial shape, a history of asthma or allergies, exposure to cigarette smoke, premature

birth, and sleep disorders. The gathered data were analyzed using SPSS software, employing the chi-square test, t-test, and logistic regression analysis.

Results : The average ages of the male and female participants were 7.28 and 7.35 years, respectively. Children with ADHD demonstrated a higher prevalence of adenotonsillar hypertrophy, 51.1%, which was significantly higher than the 18.9% observed in the control group. Also, a higher prevalence of adenoid face shape was observed, with rates of 25.5% compared to 6.7% in the control group. Furthermore, symptoms such as daytime fatigue, habitual snoring, and mouth breathing were also more frequent among the children with ADHD, although these differences were not statistically significant, likely due to the small sample size.

Conclusion : : The findings of this study suggest a significant association between ADHD and childhood adenotonsillar hypertrophy, highlighting the importance of diagnosing and treating pediatric airway obstruction symptoms. Further research is needed to better understand the extent of this correlation and to explore pathophysiological mechanisms that link these conditions.

Keywords : ADHD, Adenotonsillar hypertrophy, airway obstruction, Pediatrics, lifestyle factors

Count: 119
Abstract ID: 715
subject: Neuropsychiatry and Psychology: Disorders of Neurobehavior
Presentation Type: Poster

Neuropsychological Comparison of Existential Depression and Major Depressive Disorder with Melancholic Features: Distinctions in Activation Patterns and Prefrontal Cortex Function

Submission Author: Abolfazl Sarlaki

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Background and Aim : Although the roles of large-scale brain networks such as the Default Mode Network (DMN), Salience Network (SN), and Executive Control Network (ECN) in the pathophysiology of depression have been identified in previous research, the neurobiological differences between existential depression (arising from profound meaninglessness) and major depressive disorder with melancholic features have not yet been clearly characterized. The present study aimed to compare brain activation patterns and Prefrontal Cortex (PFC) functioning among three clinically distinct groups: patients with existential depression, patients with melancholic depression, and healthy controls.

Methods : Participants consisted of three groups (each including three individuals): existential depression, melancholic depression, and healthy controls. Diagnoses were confirmed by a clinical psychologist, and the groups were matched for age and gender. Functional Magnetic Resonance Imaging (fMRI) was conducted while participants performed two tasks: a Meaning Judgment Task and a Meaningful Autobiographical Memory Retrieval Task. fMRI data were analyzed using a general linear model to determine activation patterns, and functional connectivity between key regions, including the Hippocampus and the Prefrontal Cortex, was examined. Additionally, activity in reward-related circuits such as the Striatum and Ventral Tegmental Area (VTA) was evaluated.

Results : fMRI analyses revealed significant differences among the groups. Patients with existential depression showed markedly greater activity in the DMN compared with both melancholic patients and healthy controls. Functional connectivity between the SN and ECN was significantly reduced in the existential group. Furthermore, disrupted connectivity between the Hippocampus and PFC was observed exclusively in patients with existential depression. In the same group, activity in reward circuits (including the Striatum and VTA) was substantially lower than in the other two groups. In contrast, melancholic patients—although exhibiting some abnormalities relative to controls—lacked most of the distinctive neural alterations seen in existential depression.

Conclusion : The observed pattern suggests that existential depression is associated with a distinct neurocognitive profile compared with melancholic depression. This unique profile is characterized by DMN hyperactivity, weakened integration between the SN and ECN, disrupted Hippocampus–PFC connectivity, and reduced reward-system function. DMN overactivation likely reflects ruminative self-referential thinking and preoccupation with questions of life meaning. Decreased SN–ECN coupling indicates difficulty shifting attention from internal thoughts to external stimuli. Hippocampus–PFC disconnection may underlie deficits in processing meaningful autobiographical memories, and diminished reward activity aligns with the severity of anhedonia. These neural differences highlight the dominance of introspective and meaning-related processes in existential depression and emphasize the importance of addressing existential dimensions in assessment and treatment. The findings may contribute to identifying novel neurocognitive targets for tailored therapeutic interventions in existential depression.

Keywords : Existential Depression, fMRI, Prefrontal Cortex

Count: 120
Abstract ID: 1038
subject: Neuropsychiatry and Psychology: Disorders of Neurobehavior
Presentation Type: Oral

Enhancing the effects of Diosgenin in the rat model of autism induced by Valproic acid via ERK signaling.

Submission Author: Razieh Hajisoltani

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Background and Aim : Autism spectrum disorder (ASD) is a neurodevelopmental disorder with two core behavioral symptoms restricted/repetitive behavior and social-communication deficit. The unknown etiology of ASD makes it difficult to identify potential treatments. Valproic acid (VPA) is an anticonvulsant drug with teratogenic effects during pregnancy in humans and rodents. Prenatal exposure to VPA induces autism-like behavior in both humans and rodents. This study aimed to investigate the protective effects of Diosgenin in prenatal Valproic acid-induced autism in rats.

Methods : : pregnant Wister female rats were given a single intraperitoneal injection of VPA (600 mg/kg, i.p.) on gestational day 12.5. The male offspring were given oral Dios (40 mg/kg, p.o.) or Carboxymethyl cellulose (5 mg/kg, p.o.) for 30 days starting from postnatal day 23. On postnatal day 52, behavioral tests were done. Additionally, biochemical assessments for oxidative stress markers were carried out on postnatal day 60. Further, histological evaluations were performed on the prefrontal tissue by Nissl staining and Immunohistofluorescence.

Results : The VPA-exposed rats showed increased anxiety-like behavior in the elevated plus maze (EPM). They also demonstrated repetitive and grooming behaviors in the marble burying test (MBT) and self-grooming test. Social interaction was reduced, and they had difficulty detecting the novel object in the novel object recognition (NOR) test. Also, VPA-treated rats have shown higher levels of oxidative stress malondialdehyde (MDA) and lower GPX, TAC, and superoxide dismutase (SOD) levels. Furthermore, the number of neurons decreased and the ERK signaling pathway upregulated in the prefrontal cortex (PFC). On the other hand, treatment with Dios restored the behavioral consequences, lowered oxidative stress, and death of neurons, and rescued the overly activated ERK1/2 signaling in the prefrontal cortex.

Conclusion : Chronic treatment with Dios restored the behavioral, biochemical, and histological abnormalities caused by prenatal VPA exposure.

Keywords : Autism spectrum disorder Diosgenin Oxidative stress

Count: 121
Abstract ID: 388
subject: Neuropsychiatry and Psychology: Disorders of Neurobehavior
Presentation Type: Poster

Obsessive–Compulsive Disorder Following Orthopedic Surgery in a Pomeranian: A Neurobehavioral Case Report

Submission Author: Anahita Vosoughi

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Background and Aim : Obsessive–compulsive disorder (OCD) is a well-recognized neurobehavioral disorder in dogs, characterized by repetitive, persistent, and seemingly purposeless behaviors such as excessive licking, tail chasing, or flank sucking. Although its pathophysiology is not fully understood, dysfunction in serotonergic neurotransmission within the central nervous system has been implicated. Accurate diagnosis requires exclusion of medical causes and careful behavioral assessment. Here, we report a case of canine OCD in a 4-year-old male Pomeranian.

Methods : Approximately one month after pelvic surgery, the dog developed excessive and compulsive licking of one forelimb, which gradually led to a self-induced wound. The initial treating surgeon administered corticosteroid therapy under suspicion of an alternative medical condition; however, the symptoms persisted and the patient was eventually referred to behavioral medicine. Upon referral, a comprehensive examination was performed. Internal medicine and dermatological causes (e.g., gastrointestinal, dermatological, and systemic diseases) were excluded. A detailed behavioral history was obtained from the owner, and standardized behavioral assessment questionnaires were applied. Based on these evaluations, a final diagnosis of canine OCD was established.

Results : The patient was started on a pharmacological regimen including clomipramine and diazepam, in addition to behavioral management strategies designed to reduce anxiety and aggression. Over a two-month follow-up period, a significant reduction in compulsive licking was observed, with an estimated 80% improvement in clinical signs and healing of the lesion.

Conclusion : The onset of OCD behaviors in this patient shortly after orthopedic surgery suggests that the surgical procedure may have acted as a psychological or physiological stressor, triggering anxiety-related compulsive behaviors. This aligns with evidence indicating that OCD can be categorized within anxiety-related disorders, with stress or traumatic events often precipitating clinical manifestation. This case highlights the importance of considering OCD as a neurobehavioral disorder with a likely serotonergic basis when evaluating repetitive behaviors in dogs. Thorough exclusion of medical and neurological differentials is essential before confirming the diagnosis. The combined use of behavioral assessment tools and

neuroactive pharmacological agents can provide substantial clinical improvement and enhance quality of life in affected patients.

Keywords : OCD, neurobehavioral disorder, clomipramine, diazepam, compulsive licking, behavioral assessment, anxiety-related disorder

Count: 122
Abstract ID: 508
subject: Neuropsychiatry and Psychology: Disorders of Neurobehavior
Presentation Type: Oral

Citicoline as an Adjunctive Therapy in Major Depressive Disorder (MDD)

Submission Author: Mahtab RoohiAzizi

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Background and Aim : Residual symptoms of major depressive disorder (MDD) contribute significantly to long-term morbidity and reduced quality of life. Current treatments often fail to fully resolve these symptoms, highlighting the need for novel adjunctive therapeutic strategies. Citicoline, a neuroprotective compound traditionally used in the treatment of cerebrovascular accidents, has shown promise in cognitive rehabilitation. Additionally, it has demonstrated efficacy in reducing cravings in individuals with substance use disorders and in supporting mood stabilization in bipolar disorder. Given its neurochemical profile and prior therapeutic successes, this study investigates the effectiveness of citicoline as an adjunctive treatment for major depressive disorder.

Methods : A double-blind, randomized trial was conducted involving 50 patients with major depressive disorder who were already receiving treatment with citalopram. The patients were randomly assigned to two groups and received either citicoline (100 mg twice daily) or a placebo as an adjunctive treatment for 6 weeks. Depressive symptoms were evaluated using the Hamilton Depression Rating Scale (HDRS) at baseline and at weeks 2, 4, and 6.

Results : A significantly greater improvement in HDRS scores was observed in the citicoline group compared to the placebo group at weeks 2, 4, and 6 relative to baseline (P = 0.030, 0.032, and 0.021, respectively). Repeated measures general linear modeling revealed a significant

time × treatment interaction effect on HDRS scores (F2.10, 101.22 = 3.12, P = 0.045). Additionally, the remission rate was significantly higher in the citicoline group than in the placebo group (P = 0.045

Conclusion : Citicoline proved to be an effective adjunct to citalopram in the treatment of major depressive disorder.

Keywords : Citicoline, Antidepressant, Hamilton Depression Rating Scale, Major Depressive Dsorder

Count: 123
Abstract ID: 38
subject: Neuropsychiatry and Psychology: Disorders of Executive Functions
Presentation Type: Poster

The Relationship Between Chronic Smartphone Use, Emotion Regulation, and Prefrontal Cortex Functioning: A Cognitive-Behavioral Neuroscience Approach

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Background and Aim : Given the widespread and growing use of smartphones among young adults, this study aimed to examine the relationship between chronic smartphone use and impairments in emotion regulation and executive functions—key capacities governed by the prefrontal cortex.

Methods : In a cross-sectional correlational design, 150 university students were assessed using the Smartphone Addiction Scale (SAS), the Difficulties in Emotion Regulation Scale (DERS), and the Behavior Rating Inventory of Executive Function (BRIEF). Data were analyzed using Pearson correlation and multiple regression techniques to identify predictive relationships.

Results : Findings revealed a significant positive correlation between smartphone addiction and difficulties in emotion regulation ($r = 0.41$, $p < 0.01$). Moreover, higher smartphone usage was significantly and negatively associated with executive functioning—specifically in domains such as response inhibition, working memory, and cognitive flexibility ($r = -0.46$, $p < 0.01$). These results suggest that chronic smartphone use may adversely affect essential emotional and cognitive brain functions.

Conclusion : The findings indicate that chronic smartphone use may contribute to disruptions in self-regulatory processes and executive functioning, likely due to impaired regulation of the prefrontal cortex. These results highlight the importance of promoting digital health and developing targeted interventions to mitigate the cognitive and emotional costs associated with excessive smartphone use.

Keywords : Smartphone addiction, emotion regulation, executive function, prefrontal cortex, cognitive control

Count: 124
Abstract ID: 174
subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Which tests do students with attention deficit disorder score lower on?

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Background and Aim : The present study aimed to identify a pattern of cognitive strengths and weaknesses in students with attention deficit hyperactivity disorder (ADHD) in the small-scale tests of the fifth edition of the Wexler Children's Intelligence Scale (WISC-V).

Methods : The research method was descriptive-analytical with a cross-sectional approach. The statistical community included students with a definite diagnosis of ADHD (based on the DSM-5-TR) in education and counselling centres in districts 1, 4 and 6 and two private clinics in Tehran. Of these, 170 subjects (85 subjects with attention deficit disorder and 85 subjects with attention deficit hyperactivity disorder) were selected in a targeted manner. The WISC-V research tool included five major scales of verbal comprehension, spatial vision, fluid reasoning, active memory, and processing speed.

Results : The results showed that the students with ADHD performed within the normal range in the verbal and visual-spatial comprehension tests. On the scale of fluid reasoning, a clear weakness was observed in the "computation" sub-test. On the active memory scale, the lowest averages were for the tests of "straight number density", "reverse number density", "serial number density", and "image density", all of which were more than two standard deviations below the standard mean. Also, on the processing speed scale, all the micro-tests were significantly below the standard average. These findings suggest that the main weaknesses of students with ADHD are in the components of active memory and processing speed, while their verbal and spatial-visual abilities are in the normal range.

Conclusion : Accordingly, attention to this pattern can be effective in designing targeted educational and therapeutic interventions to enhance cognitive function in this group of students.

Keywords : Attention deficit hyperactivity disorder, children's Wexler scale of intelligence, fifth edition

Count: 125
Abstract ID: 386
subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Antidepressant-like effects of phycocyanin in a rat model of chronic unpredictable mild stress

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Background and Aim : Chronic uncontrolled stress may result in anxiety, depression, and other stress related disorders. External stress factors such as stressful life events and internal stress factors such as chronic inflammation may induce inflammatory and oxidative stress pathways to precipitate depression in susceptible individuals. In animals, the chronic psychosocial stress may induce neuroinflammation and apoptosis. Current therapies for depression have limitations. Hence, development of drugs with better efficacy and safety is required. Recently, natural plant compounds have been shown to possess neuroprotective effects. One of these compounds is phycocyanin, which is abundant in Spirulina. This study aimed to assess the effects of phycocyanin on neurobehavioral and inflammatory parameters in chronic unpredictable mild stress model of depression in rats.

Methods : In this experimental study, 24 male Wistar rats were randomly assigned to 3 groups (n=8): control group, CUMS group and CUMS+ phycocyanin group. For CUMS induction, the animals were subjected to CUMS for six weeks by using variety of stressors. According to previously study, the phycocyanin (100 mg/kg) administered orally during the last 3 weeks. At the end of CUMS protocol, animals were subjected to Forced Swim Test (FST), Y-maze test, and Novel Object Recognition Test. After completing the behavioral assessments, the rats were decapitated, and their brains were carefully removed from the skull. Then, the hippocampus was quickly separated from the brain for biochemical assessments. In addition, Interleukin (IL)-1 and tumour necrosis factor alpha (TNF-α) by ELISA kits were evaluated.

Results : In the FST, immobility time was measured as the primary parameter to assess depression-like behavior. CUMS increased immobility time compared to control group ($p<0.05$). However, phycocyanin significantly decreased immobility time compared to CUMS group ($p<0.05$). CUMS also impaired memory function in Y-maze test, and Novel Object Recognition Test compared to control group ($p<0.05$). However, the data showed that phycocyanin significantly improved memory of rats that were subjected to the CUMS procedure ($p< 0.05$). Moreover, CUMS increased IL-1 and TNF-? levels in hippocampus of rats compared control group ($p< 0.05$). However, it decreased IL-1 and TNF-? levels ($p< 0.05$) in hippocampus.

Conclusion : The present study highlights the neuroprotective effects of phycocyanin, noting that it improves depressive-like behaviour, and learning and memory, and decreased IL-1 and TNF-α in the hippocampus. We can conclude that the anti-depressive effects of phycocyaninon is partly related to its multiple effects on the neuroinflammation in the hippocampus.

Keywords : Phycocyaninon, depression, memory

Count: 126
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subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Role of Glia in Regulating Circadian Rhythms and Improving Sleep Disorders in Parkinson's Disease

Submission Author: Fatemezahra Ghasemifard

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Background and Aim : Sleep disorders and circadian rhythm disruptions affect over **80%** of Parkinson's disease (PD) patients, significantly impairing quality of life in Iran's aging population. This study investigates glial cells' (astrocytes and microglia) role in regulating circadian rhythms via gliotransmitter signaling and neuroinflammation modulation, proposing novel therapeutic strategies for PD-related sleep disorders using optogenetics and pharmacology.

Methods : : A preclinical-clinical design was employed. In the preclinical phase, 60 male Wistar rats with 6-OHDA-induced PD (n=15 per group) received either optogenetic astrocyte stimulation in the suprachiasmatic nucleus (SCN) or sham treatment, with a subgroup receiving minocycline (45 mg/kg, 14 days) to reduce microglial inflammation. Clock gene expression (CLOCK, BMAL1, PER2) was measured via qPCR, inflammation via ELISA, and sleep behavior via actigraphy. Clinically, 30 PD patients (Hoehn and Yahr stages 2–3) and 20 controls from Imam Khomeini Hospital, Tehran, were assessed using polysomnography (PSG), Epworth Sleepiness Scale (ESS), functional near-infrared spectroscopy (fNIRS), and cerebrospinal fluid (CSF) analysis for glial markers (GFAP, Iba1). Data were analyzed using ANOVA, t-tests, and Pearson correlations ($p<0.05$).

Results : In PD rats, optogenetic stimulation increased SCN clock gene expression by 75–82% ($p<0.05$), total sleep time by 32%, and REM sleep by 41% ($p<0.05$) compared to sham. Minocycline reduced TNF-? by 52% ($p<0.01$), enhancing sleep efficiency by 35%. **The combined intervention led to an 85% improvement in overall outcomes ($p<0.01$).** In PD patients, sleep efficiency was 62.4 ± 8.3% vs. 85.7 ± 5.1% in controls ($p<0.001$), with REM sleep reduced (4.8% vs. 12.5%, $p<0.01$). Elevated CSF GFAP (580 ± 125 ng/L) and Iba1 (420 ± 90 ng/L) levels correlated negatively with sleep efficiency ($r=-0.58$, $p<0.01$). fNIRS showed 38% reduced hypothalamic oxyhemoglobin signals in patients ($p<0.05$).

Conclusion : Glial-targeted interventions, including optogenetic astrocyte activation and microglial inflammation suppression, enhance circadian regulation and sleep in PD. These findings support personalized neurorehabilitation strategies, aligning with the congress's Emerging Technologies, Gliology, and Neuroendocrinology sections. Given PD's burden in Iran, non-invasive translations (e.g., rTMS) and clinical trials are recommended, with neuroethical considerations ensuring safe access in developing contexts. This study aligns with the 14th Iranian Congress of Basic and Clinical Neuroscience 2025's themes of personalized medicine, emerging technologies, and neuroendocrinology, addressing the critical need for novel treatments for PD-related sleep disorders in Iran's aging population.

Keywords : Glia, Circadian Rhythms, Parkinson's Disease, Sleep Disorders, Optogenetics, Neurorehabilitation.

Count: 127
Abstract ID: 521
subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Managing breathing pattern by the effects of mental imagery: rehabilitation (health psychology) after dyspnea

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Conclusion : Dyspnea or air hunger is distressed breathing that is associated with mental awareness. The aim of this narrative study is an assessment of the effectiveness of mental imagery on respiratory function. In this study we must answer this question: can consciousness-mind improve in the air hunger situation? Awareness and dyspnea are both mental sensations, and dyspnea has a multidimensional and multidisciplinary nature. A narrative synthesis approach was used to review studies showing how personality traits are associated with breathing pattern management by the effects of mental imagery following dyspnea. The multidimensional nature of dyspnea in the sensory-conceptual domain can result in emotional distress. The perception of dyspnea involves complex afferent and efferent signals that must integrate into the cerebral cortex for effective processing. Air hunger signaling by a rise in the medullary respiratory stimulus arrives at the cerebral cortex and is sensed by stretch receptors of the pulmonary vague nervous system. Air hunger as a distress breathing can be improved by increasing in the tidal volume of the lungs. Low-volume lung-protective ventilation decreases tidal stretch receptor input and often increases respiratory drive (via elevated carbon dioxide tension). Thus, air hunger signaling begins to activate brain pathways that are known to play a significant role in the incidence of post-traumatic stress disorder (PTSD), anxiety, and depression. The level of dyspnea is not the same in both people, because the feeling of dyspnea changes at different times, and in different situations. Mental imagery is a common occurrence in most people's daily lives. Mental imagery is the motivational amplifier for promoting activities. Mental imagery can increase participation in planned behaviors, potentially in representative outstanding emotional practices. This review emphasizes on managing the emotional distress caused by dyspnea by imagery in the brain through three different themes: 1) Attention and bodily sensations in the dyspnea, 2) Episodic memory and cognitive load in dyspnea, and 3) Management of dyspnea with mental imagery to perform the process of health psychology.

Keywords : mental imagery, dyspnea, health psychology

Count: 128
Abstract ID: 371
subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Short- and Long-Term Impacts of Ketamine on Wistar Rat Sleep Patterns

Submission Author: Mehdi Amirhooshangi

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Background and Aim : Ketamine, a noncompetitive antagonist of N-methyl-D-aspartate (NMDA) receptors, has been widely investigated for its anesthetic, analgesic, and rapid antidepressant properties (1,2). Although commonly applied in clinical and experimental settings, its precise influence on sleep architecture remains unclear, especially regarding non-rapid eye movement (NREM) and rapid eye movement (REM) sleep cycles (3). Given that disturbances in sleep are strongly linked to psychiatric and neurological disorders (4), clarifying how ketamine modulates sleep parameters is essential. Previous studies have reported that ketamine alters cortical oscillations and promotes slow-wave activity (5,6), yet systematic studies comparing acute and chronic exposure remain limited. The present research aimed to evaluate both immediate and repeated effects of ketamine on sleep structure and cortical synchronization in Wistar rats using local field potential (LFP), electroencephalogram (EEG), and electromyogram (EMG) recordings.

Methods : Adult male Wistar rats were surgically implanted with hippocampal and cortical electrodes for continuous polysomnographic monitoring (7). Baseline recordings were collected under standardized light–dark cycles. After recovery, animals were randomly assigned to receive intraperitoneal injections of either saline or ketamine (30 mg/kg). Sleep patterns were analyzed at two stages: (i) acute effects during the 24 hours following a single injection and (ii) chronic effects after 7 consecutive days of daily administration. NREM and

REM sleep were scored using established sleep scoring criteria (8), while delta power (0.5–4 Hz) during NREM was quantified with fast Fourier transform (FFT) analysis (9). Statistical analyses were performed using parametric tests, with significance set at $p < 0.05$.

Results : Acute administration of ketamine significantly increased NREM sleep during the first 6 hours compared to controls (239.1 ± 12.8 vs. 169.5 ± 10.6 min, $p < 0.01$). REM sleep duration was markedly reduced (17.8 ± 3.2 vs. 36.9 ± 4.1 min, $p < 0.05$). Delta power during NREM rose by 47.6% from baseline ($p < 0.01$), indicating deeper sleep and enhanced cortical synchronization. After 7 days of repeated dosing, NREM sleep remained elevated (204.7 ± 11.3 min, $p < 0.05$) though attenuated compared to the acute response. REM sleep partially recovered (29.3 ± 3.6 min), while delta power persisted 35.1% above baseline ($p < 0.05$), suggesting sustained slow-wave activity despite repeated exposure.

Conclusion : These findings indicate that ketamine acutely enhances NREM sleep and suppresses REM sleep, with persistent increases in delta power following chronic administration. While partial tolerance develops, the maintenance of slow-wave activity underscores ketamine's long-lasting influence on cortical synchronization. Such properties may explain part of its therapeutic potential for mood and sleep-related disorders.

Keywords : Ketamine; Sleep patterns; Wistar rats; NREM sleep; REM sleep; Local field potential

Count: 129
Abstract ID: 14
subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Phonological Loop Versus Visual-Spatial Memory: Which Is More Vulnerable in Patients with Breast Cancer?

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Background and Aim : Cancer, as a chronic physical and non-communicable disease, constitutes a major public health challenge for healthcare systems worldwide, including Iran. Beyond the well-documented physical and emotional burdens, individuals diagnosed with cancer frequently experience significant cognitive sequelae. Among these, working memory deficits are particularly prevalent and are often linked to neurotoxic effects of chemotherapy regimens. Chemotherapy-related cognitive impairment (CRCI), colloquially termed "chemobrain," commonly manifests as memory difficulties, potentially affecting auditory-verbal processing, visual-spatial processing, or integrative functions. Given the critical role of working memory subsystems – specifically the phonological loop for auditory-verbal information and the visual-spatial sketchpad for visual information – in daily functioning, this study aimed to conduct a targeted assessment of these distinct components in breast cancer patients undergoing chemotherapy, comparing their performance to a healthy control group.

Methods : This study employed a causal-comparative design. Participants were initially assessed using a diagnostic interview and a demographic information questionnaire. The statistical population comprised all women with breast cancer undergoing chemotherapy and all non-cancer individuals residing in Rafsanjan, Iran, in 2023 (1402 SH). The study sample consisted of 38 breast cancer patients and 38 non-cancer individuals, selected via convenience sampling. Subsequently, participants were assessed using the Wechsler Working Memory Test subtests: Letter-Number Sequencing (assessing the central executive and phonological loop) and Spatial Span (assessing the visual-spatial sketchpad). Data analysis and hypothesis testing were performed using Multivariate Analysis of Covariance (MANCOVA) with SPSS-26 software.

Results : The MANCOVA results revealed significant multivariate effects. Breast cancer patients demonstrated statistically significant and clinically relevant impairments in overall

working memory performance compared to the healthy control group ($p < 0.001$). Crucially, a nuanced analysis of the subsystems indicated that while deficits were present in both domains, participants in the patient group exhibited relatively greater impairment within the auditory-verbal domain (phonological loop) as measured by the Letter-Number Sequencing, compared to the visual-spatial domain (sketchpad) assessed by the Spatial Span. This pattern suggests a differential vulnerability of auditory-verbal processing under conditions of chemotherapy-induced cognitive load.

Conclusion : This detailed comparative analysis confirms that breast cancer patients treated with chemotherapy experience significantly greater working memory impairment than healthy individuals, directly attributable to the neurocognitive side effects of chemotherapy agents. The findings underscore that these patients contend with substantial memory lapses and concentration difficulties, severely impacting cognitive function. The pronounced deficit observed within the phonological loop subsystem, responsible for processing and temporarily holding auditory-verbal information, indicates a specific vulnerability. This may stem from increased susceptibility to distraction, reduced ability to maintain focused attention on auditory stimuli, and consequently, inefficient phonological rehearsal processes. The relative sparing, though not absence, of deficits in the visual-spatial sketchpad warrants further investigation but highlights the complex, domain-specific nature of chemotherapy's impact on cognitive architecture. These results emphasize the need for tailored cognitive rehabilitation strategies focusing on auditory-verbal processing and attention enhancement for this patient population.

Keywords : Visual-Spatial Working Memory, Phonological Loop, Breast Cancer Patients, Chemotherapy-Related Cognitive Impairment.

Count: 130
Abstract ID: 960
subject: Neuropsychiatry and Psychology: Cognitive Disorders
Presentation Type: Poster

Bridging Neurophysiology and Psychometrics: Predicting MCMI Scores from Resting-State EEG Using Machine and Deep Learning

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Background and Aim : The link between brain activity and psychological traits is fundamental to neuroscience, yet translating this connection into objective clinical tools remains a significant challenge. Psychological inventories like the Millon Clinical Multiaxial Inventory (MCMI) provide detailed profiles but are based on selfperception, making them susceptible to response biases and the limits of self-awareness. Electroencephalography (EEG) offers a direct, objective measure of neural dynamics. This research explores whether machine and deep learning can bridge the gap between these two domains by identifying stable patterns in EEG data that correspond to specific clinical personality and symptom profiles as defined by the MCMI.

Methods : Data were acquired from approximately 30 subjects, each providing a resting-state EEG recording from a 19-channel system and a completed MCMI-IV questionnaire. A standardized preprocessing pipeline was applied to the raw EEG signals, including a band-pass filter(1–45 Hz) and applying artifact correction techniques to isolate neural activity. From the cleaned signals, Power Spectral Density (PSD) was computed using Welch's method. The primary features of interest were the mean absolute power within five canonical frequency bands (Delta: 1–4 Hz, Theta: 4–8 Hz, Alpha: 8–13 Hz, Beta: 13–30 Hz, and Gamma: 30–45 Hz) across all channels. Using the continuous MCMI Base Rate (BR) scores as target variables, we employed a supervised learning framework. A comprehensive suite of models are trained on the data, to determine the optimal architecture for mapping neural features to the psychological profiles. The conventional machine learning models include: 1) Random Forests (RF), for its robustness in high-dimensional spaces; 2) Support Vector Machines (SVM), to model complex decision boundaries; and 3) Gradient Boosting Machines (XGBoost), for its state-of-the-art predictive accuracy. For our deep learning approach, we explored: 1) 1D Convolutional Neural Networks (CNNs), to learn topographical patterns across EEG channels;

2) Long Short-Term Memory (LSTM) networks, to model sequential dependencies; and 3) Hybrid CNN-LSTM architectures, to leverage both spatial and sequential feature extraction.

Results : Preliminary analysis confirms a strong, systematic relationship between subjects' neurophysiological data and their MCMI-derived profiles. Initial feature importance analysis, consistent across multiple models, reveals that oscillatory power within the lower frequency bands (Alpha and Theta) consistently emerges as highly influential. Furthermore, topographical analysis suggests that the most salient predictive features are concentrated in specific cortical regions. For assessment of our models we selected regression metrics, including Mean Absolute Error (MAE), Root Mean Squared Error (RMSE), and the coefficient of determination (R2). To ensure our models are both accurate and interpretable, we have selected the SHAP (SHapley Additive exPlanations) approach to identify the most salient brain patterns and quantify their contribution towards driving the predictions.

Conclusion : This study serves as a successful proof of concept, establishing a direct, quantifiable link between objective brainwave signatures from 19-channel EEG and well-defined psychological constructs. Our findings demonstrate that machine learning can effectively exploit neurophysiological features (via EEG signal features) to predict mental health profiles. This approach holds significant potential for developing objective, adjunctive tools to support clinical assessment and offers a new avenue for understanding the neurobiological basis of personality and psychopathology.

Keywords : Resting-State EEG, Deep Learning, Millon Clinical Multiaxial Inventory, Psychometrics, Personality Prediction

Count: 131
Abstract ID: 947
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

Assessing Perceived Social Support in Psychiatric Patients with a History of Suicide Attempt: A Cross-Sectional Study in Kermanshah

Submission Author: Sahel Kanjouri

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Background and Aim : Psychological disorders are considered one of the major risk factors for suicide attempts, with approximately 90% of individuals who die by suicide suffering from some form of psychological disorder. Perceived social support acts as a determinant factor influencing the relationship between psychological disorders and suicide attempts. This research was conducted with the aim of investigating the level of perceived social support among psychological disorders leading to suicide in patients referred to Farabi Hospital in Kermanshah.

Methods : In this cross-sectional descriptive study, all patients diagnosed with psychiatric disorders who visited Farabi Hospital in Kermanshah between the years 2021 and 2024 were examined. Inclusion criteria for the study included a diagnosis of a psychiatric disorder by a psychiatrist based on the criteria of the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), and a history of attempted suicide. The sampling method was convenience sampling, where all eligible patients with psychiatric disorders and a history of suicide attempt were included in the study. The data for the present research included demographic information, medical and clinical information, information related to the history of suicide attempts, and the Perceived Social Support. This 12-item questionnaire was developed by Zimet and Farley (1988) to assess perceived social support from three sources: family, friends, and significant others.

Results : In this study, 450 participants (79.6%) were male and 115 participants (20.4%) were female. Most of the participants were single (49%), held a high school diploma (30%), and were unemployed (37.2%). The mean and standard deviation of perceived social support among the participating patients were 44.19 and 13.75, respectively. The most frequent psychological disorders leading to suicide among the participating patients were, respectively, substance-related and addictive disorders (35.1%), schizophrenia spectrum disorders (24.9%), and bipolar disorder (21.8%). The mean perceived social support score was higher in women, married individuals, educated individuals, and patients with high socioeconomic status compared to others. Furthermore, the difference in perceived social support scores across different psychiatric disorders was statistically significant (p=0.000).

Conclusion : By examining the prevalence of psychological disorders leading to suicide, it was shown that substance-related and addictive disorders pose a serious warning due to their

highest frequency among the existing disorders. Furthermore, perceived social support, which acts as a protective and deterrent factor against suicide attempts, is not in an adequate state.

Keywords : social support; psychological disorders; Suicide; psychiatric disorders.

Count: 132
Abstract ID: 627
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Oral

Brain Structural Changes and Cognitive Impairment in Unipolar Mood Disorders: The Role of Chronic Stress

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Conclusion : Unipolar mood disorders, including depression and anxiety, are increasingly associated with chronic stress response, showing overlapping brain structural and functional changes. The hippocampus, prefrontal cortex (PFC), and memory-related networks are critical for cognitive function and emotion regulation and are highly sensitive to HPA axis dysregulation. Neuroimaging studies consistently reveal reductions in hippocampal volume in unipolar depression, with meta-analyses reporting an 8–10% decrease, particularly linked to the cumulative number of depressive episodes. Cognitive deficits, including working memory and emotion regulation impairments, have been associated with hippocampal and prefrontal cortical dysfunction. fMRI studies in anxiety-inducing contexts show hippocampal hyperactivity, while generalized anxiety disorder (GAD) is characterized by PFC and anterior cingulate cortex (ACC) hypofunction, disrupted cortex-amygdala connectivity, and abnormal activation across memory-related networks, including dorsolateral PFC, parahippocampal gyrus, fusiform gyrus, and cerebellum during cognitive tasks. Structural abnormalities in major depression include large volume reductions in frontal regions, especially the ACC and orbitofrontal cortex, with moderate decreases in the PFC, hippocampus, putamen, and caudate nucleus. These changes correlate with working memory impairments and emotion dysregulation. Chronic HPA axis activation contributes to hippocampal neuroinflammation, oxidative stress, and cognitive deficits. Evidence from treatment studies shows partial normalization of limbic and PFC activity, highlighting the potential reversibility of these alterations. Overall, chronic stress-driven HPA dysregulation mediates hippocampal and prefrontal structural changes, leading to cognitive impairments and sustaining the pathological interplay between anxiety and depressive symptoms. Recognizing the neurobiological overlap between these disorders emphasizes the importance of targeting structural integrity and stress regulation in therapeutic interventions.

Keywords : stress, HPA axis, depression, anxiety, neuroimaging, hippocampus

Count: 133
Abstract ID: 448
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

Investigating the effectiveness of quantitative electroencephalogram-based transcranial direct current stimulation combined with positive psychotherapy on students with major depression

Submission Author: Mohammadreza Gholipourfallahy

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Background and Aim : Based on QEEG studies, it has been shown that people with depressive disorders have decreased activity in the left hemisphere of the frontal cortex. Transcranial direct current stimulation (tDCS) is a non-invasive neuromodulation technique that can modulate the activity of brain regions, and in the present study, it specifically increased the activity of the frontal cortex.

Methods : This study was conducted on 60 individuals with major depressive disorder who were selected after clinical assessment and QEEG diagnosis and were randomly divided into 3 groups for intervention. In this study, 2 intervention methods were used: tDCS and positive psychology therapy (PP) to improve the quality of life of the patients. The present study compared three groups of participants with major depression; the first group received (a) tDCS treatment, the second group received (b) positive psychotherapy, and the third group received (c) combined treatment.

Results : In examining the results obtained using multivariate analysis of variance with pre- and post-test, participants in the combined intervention group showed a greater reduction in depressed mood, improved hope, and optimism than other conditions after 4 weeks and during the 3-month follow-up.In the tDCS intervention group, only depressed mood improved, and in the psychotherapy and recording intervention group, only hope and optimism improved, which shows the effectiveness of the combined method in treatment.

Conclusion : Future studies should (a) use larger sample sizes, (b) consider untreated control conditions, (c) consider double-blind designs, (d) control for prior or current drug therapy, (e) control for Furthermore, future studies should increase theoretical understanding by examining whether the relationship between tDCS and psychotherapy is additive or synergistic.

Keywords : QEEG, Transcranial direct current stimulation, positive psychology therapy , major depression

Count: 134
Abstract ID: 660
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

Evaluation of the Relationship Between Sleep Quantity and Suicidal Behaviors (Ideation, Plan, and Attempt) in the Fasa Youth Cohort

Submission Author: Mohammadsadegh Kamran

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Background and Aim : Sleep is a vital neurobiological process that plays a crucial role in emotional regulation and mental health. Recent research suggests that sleep disturbances may be associated with a higher risk of suicidal behaviors, including suicidal ideation, planning, and attempts. This study aimed to evaluate the relationship between sleep quantity and different forms of suicidal behavior among young adults in the Fasa Youth Cohort, Iran.

Methods : This cross-sectional study utilized data from the Fasa Youth Cohort, including 3,013 participants aged 15–35 years. Data were obtained from standardized cohort questionnaires covering demographic characteristics, psychiatric history, substance use, and sleep patterns. Nighttime sleep duration was used as the main indicator of sleep quantity. Suicidal behaviors (ideation, plan, and attempt) during the past two years were assessed using structured questions. Statistical analyses were performed using SPSS version 24. Chi-square tests and independent t-tests were used for univariate analysis, while multivariate logistic regression identified independent predictors. Adjusted odds ratios (OR) with 95% confidence intervals (CI) were calculated.

Results : Among participants, 7.3% reported suicidal ideation, 5.1% reported suicidal planning, and 4.5% reported suicide attempts. The mean nighttime sleep duration was 9.0 ± 1.6 hours. Participants with suicidal ideation or plan had significantly shorter nighttime sleep compared to others ($p = 0.004$ and $p = 0.003$, respectively). After adjusting for confounders such as depression, anxiety, psychiatric hospitalization, and smoking, reduced nighttime sleep remained significantly associated with higher odds of suicidal ideation (OR = 1.34, 95% CI: 1.10–1.65) and suicide attempt (OR = 1.35, 95% CI: 1.01–1.80). Depression or anxiety (OR = 2.25, $p = 0.001$) and psychiatric hospitalization (OR = 3.01, $p = 0.027$) were strong predictors, whereas daytime sleep showed a mild protective effect (OR = 0.81, 95% CI: 0.67–0.99).

Conclusion : This study demonstrated a significant association between reduced sleep quantity and suicidal behaviors, including ideation, plan, and attempt, among young adults. Given that sleep duration is a modifiable behavioral factor, these findings highlight the importance of early

screening for sleep problems as part of suicide prevention strategies. Further longitudinal and interventional studies are recommended to clarify the causal mechanisms linking sleep and suicidal behaviors.

Keywords : Sleep quantity; Suicidal ideation; Suicide attempt; Suicide plan; Depression; Youth cohort; Mental health

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subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

Pro-Inflammatory Cytokines Level in Depressed Patients with Suicidal Behaviour: A Systematic Review and Meta-Analysis

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Conclusion : Introduction: The study assesses proinflammatory factors in major depressive disorder (MDD) patients with and without suicidal behaviors, and healthy controls, to identify potential biomarkers for suicidal behaviors and prevention strategies. Methods: We conducted a systematic review, which we have registered in Prospero (#CRD42024547764), and searched through five databases until March 2024. We included observational studies in English that reported on depressed patients with and without suicidal behaviors, as well as healthy controls, and measured the levels of pro-inflammatory cytokines. The Newcastle- Ottawa Scale was used for quality evaluation of the studies and the main analysis was performed using Stata 17. Results: One systematic review resulted in 4369 articles but only 24 were selected for the review. The meta-analysis showed depressed patients with suicidal behaviors had significantly higher interleukin (IL)-6 levels compared to the control group with a standardized mean difference (SMD) of 1.26 [0.07, 2.45]; I^2 = 98.81%. There was no significant difference noted

between the depressed patients suffering from suicidal behaviors and those not suffering (SMD 0.42 [-0.58, 1.42]; I^2 = 98.50%). There is no significant difference between the groups in the levels of tumor necrosis factor-alpha (TNF-α) and IL- 10. Further analysis of IL-4, IL-2, IL-1β, IF-γ, and TGF-β1 also indicated no significant group differences. Conclusion: The review found no significant cytokine differences (including IFN-γ, IL-1β, IL-2, IL- 4, IL-8, IL-10, TGF-β1, TNF-α) between depressed individuals with or without suicidal ideation and controls. Variations likely reflect methodological differences, highlighting the need for standardized, longitudinal research.

Keywords : Depression, Suicide, Suicidal attempt, Suicidal ideation, Cytokines, Inflammation.

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subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Oral

The Psychophysiological Mirror: A Framework for Investigating Transdiagnostic Emotional Pathways in Depression, Anxiety, Aggression, and Mania Using Backpropagation from Induced Sadness, Fear, Anger,

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Background and Aim : In the intricate landscape of mental health, the dynamic interplay between emotion and stress provides a critical framework for understanding the etiology and maintenance of psychological disorders. This paper introduces the "psychophysiological mirror" as a novel conceptual model that reflects the bidirectional connections between specific emotional states and their physiological correlates in individuals with depression, anxiety, aggression, and mania. Building on foundational work which establishes emotional regulation as a cornerstone of mental health, this exploration examines how maladaptive processes—such as the heightened stress reactivity seen in anxiety and the ruminative cycles linked to depression —manifest as discernible psychophysiological patterns. By mapping these transdiagnostic pathways, this research aims to provide a comprehensive framework for developing more effective, mechanism-targeted strategies for intervention and support.

Methods : This pilot study employed an intelligent, transdiagnostic framework to investigate the "psychophysiological mirror" in individuals with depression, anxiety, aggression, and mania. The protocol involved the experimental induction of core emotional states (sadness, fear, anger, happiness) to model stress-related dysregulation, guided by the diathesis-stress model (Zuckerman, 1999) and cognitive appraisal theory (Lazarus & Folkman, 1984). The key intervention was the application of Transcranial Photobiomodulation (tPBM), a non-invasive neuromodulation technique that uses specific wavelengths of light to modulate cortical activity. Psychophysiological measures (e.g., EEG, heart rate variability) were recorded concurrently to quantify the emotional and stress responses. Data were analyzed using a backpropagation-based computational model to identify the core neural pathways linking emotional induction to disorder-specific symptomatology, with the aim of developing an intelligent, personalized intervention platform.

Results : This study directly addressed key gaps in the literature on emotion, stress, and psychological disorders. First, by employing a backpropagation-based computational model on our longitudinal psychophysiological data, we were able to map the dynamic interaction between emotional regulation deficits and stress reactivity over time, revealing distinct trajectories for the development of depression, anxiety, aggression, and mania. Second, our transdiagnostic "psychophysiological mirror" framework successfully identified a core circuit of dysregulation common across all four disorders, while also delineating unique, disorder-specific physiological signatures in response to induced sadness, fear, anger, and happiness. This provides a mechanistic explanation for frequently observed comorbidity. Finally, preliminary analysis of demographic data integrated into our model indicated that cultural background significantly modulated the amplitude and expression of the psychophysiological response to stress induction, underscoring the necessity of culturally informed application of interventions like tPBM.

Conclusion : In conclusion, this pilot study successfully established a "psychophysiological mirror" framework, demonstrating its utility as a transdiagnostic model for mapping the intricate pathways between stress-induced emotional states and major psychological disorders. By integrating advanced computational backpropagation with Transcranial Photobiomodulation (tPBM), we have moved beyond theoretical gaps to provide an intelligent, mechanism-based intervention strategy. Our findings confirm that targeting the core psychophysiological dysregulation common to depression, anxiety, aggression, and mania offers a promising avenue for treatment, particularly for complex, comorbid cases. Future longitudinal and culturally-adapted replications of this protocol are warranted to translate these findings into robust, personalized clinical applications, ultimately paving the way for a new paradigm in mental health care that unifies technology, physiology, and psychology.

Keywords : Psychophysiological Mirror, Transdiagnostic, Transcranial Photobiomodulation (tPBM), Emotion Induction, Backpropagation, Computational Psychiatry

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subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

From Cognitive Mechanisms to Clinical Impact: Understanding Punishment and Reward Interactions in Volatile Environments for Major Depressive Disorder (MDD)

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Background and Aim : Major Depressive Disorder (MDD) is a prevalent psychiatric condition marked by cognitive impairments that hinder decision-making and belief updating (LeMoult & Gotlib, 2019). A key feature of MDD is the disruption in belief updating in response to feedback, particularly in volatile environments (Aylward et al., 2019). However, the mechanisms behind this disruption remain unclear due to variations in study methodologies and models. This study investigates the interaction between punishment and reward in volatile environments, a topic rarely explored together, especially in the context of MDD. The findings provide insight into how individuals with MDD process these opposing stimuli, offering new perspectives on decision-making and belief updating. Additionally, our novel computational approach, using probabilistic reversal learning in uncertain conditions, contributes a fresh understanding of cognitive processes in MDD, advancing cognitive neuroscience and mental health treatments.

Methods : We used a computer-based probabilistic reversal learning task to examine belief updating in individuals with MDD. The task was performed twice, once in a reward context and once in a punishment context, with the order of tasks counterbalanced to control for sequence effects. Participants chose between three colored decks and received feedback to guide their decisions. Each task consisted of 160 trials, divided into two blocks: a "simple" block (trials 1–80) with outcome probabilities of 90%, 50%, and 10%, followed by a "complex" block (trials 81–160) with probabilities of 80%, 40%, and 20%. Probability shifts occurred in two ways: every 40 trials and after selecting the best deck in 9 out of 10 trials. To investigate the dynamics of computational parameters in this learning task, we analyzed changes in task parameters between consecutive blocks under both conditions using paired-sample t-tests and

Wilcoxon signed-rank tests, comparing an experimental group of individuals with MDD to a healthy control group.

Results : The experimental group showed significant adaptation in learning rates for both punishment (p=0.024) and reward (p=0.028) conditions, while the control group only exhibited significant changes in the punishment condition (p=0.048). The experimental group also updated their uncertainty about reward-associated options (sigma3_0) in both punishment (p=0.019) and reward (p=0.008) contexts, unlike the control group. Both groups adjusted their uncertainty about punishment-related options (sigma2_0) in reward blocks. The experimental group showed a significant shift in the initial belief regarding punishment options (mu2_0) from Block 1 to Block 2 (p=0.023), indicating a dynamic change in valuation. Additionally, the experimental group exhibited altered exploration-exploitation dynamics, with significant decision noise change during the reward condition (Wilcoxon, p=0.010), while no such change was observed in the control group. The prior belief for reward (mu3_0) and the kappa parameter remained stable across all conditions for both groups, suggesting they are non-adaptive components.

Conclusion : The experimental group demonstrated greater modulation of computational parameters related to learning, belief updating, and uncertainty processing, indicating altered cognitive flexibility and adaptive responses to both reward and punishment feedback. In contrast, the control group showed stable parameters, suggesting potential computational markers for anxiety and depressive disorders.

Keywords : Uncertainty; Reversal Learning; Belief Updating; Major Depressive Disorder; Volatility

Count: 138
Abstract ID: 286
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

Causal Effects of Personality Traits on Psychiatric Disorders: Evidence from a Mendelian Randomization Study on genetic data

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Background and Aim : Personality traits are closely connected with mental health. Among them, Neuroticism and Extraversion have been studied the most and are often reported to relate to psychiatric disorders. The question is whether these links reflect a true causal effect or are simply correlations affected by other factors. To answer this, we used Mendelian randomization, which applies genetic markers as tools to test causality. We investigated the possible causal role of Neuroticism and Extraversion in psychiatric outcomes. Our analysis included ADHD, anxiety, bipolar disorder, insomnia, major depressive disorder (MDD), and PTSD.

Methods : We based our analysis on summary results taken from large genome-wide association studies (GWAS). These datasets are freely available and have been widely used in genetic research. Independent single nucleotide polymorphisms (SNPs) strongly associated with Neuroticism and Extraversion were selected as genetic instruments. We performed two-sample MR analyses across six psychiatric outcomes, employing five complementary estimators: inverse variance weighted (IVW), maximum likelihood (ML), MR Egger regression, weighted median, and weighted mode. Several sensitivity checks were carried out, including Cochran's Q to assess heterogeneity, the MR-Egger intercept to detect directional pleiotropy, and influence analyses such as radial and leave-one-out approaches.

Results : The analyses showed that Neuroticism had clear and consistent effects on several outcomes. The strongest association was with major depressive disorder, where both IVW and maximum likelihood approaches gave very similar results (β around 0.46–0.48, $p < 1\times10^{-50}$). Anxiety also showed a robust link, with IVW estimates of about $\beta = 0.0045$ ($p \approx 2\times10^{-16}$). For ADHD, the evidence was supportive, with effect sizes in the range of $\beta = 0.15$–0.17 and highly significant p-values (<0.001). Bipolar disorder and PTSD also indicated increased risk ($\beta \approx 0.14$ and $\beta \approx 0.34$, both $p < 0.001$), although in bipolar disorder some heterogeneity was noted.

In contrast, no convincing evidence was seen for insomnia, where all methods produced non-significant results. Extraversion showed a different picture. A positive association was detected for ADHD, with IVW estimates close to $\beta = 0.69$ ($p \approx 0.004$), supported by the weighted median method. Bipolar disorder showed a weaker and less certain signal ($\beta \approx 0.36$, p just above 0.02), while for MDD the single available instrument suggested a protective effect ($\beta = -0.68$, $p \approx 3.6\times10^{-4}$). For anxiety, PTSD and insomnia, the effect estimates were close to zero and not statistically significant. Sensitivity analyses indicated that pleiotropy was unlikely to explain these findings, although the bipolar results were more heterogeneous.

Conclusion : Overall, Neuroticism increased psychiatric risk in our MR tests. The clearest effect was for depression and anxiety; smaller effects appeared for ADHD, bipolar disorder and PTSD; insomnia was null. Extraversion showed more selective patterns: higher risk for ADHD, possible risk for bipolar disorder, and lower risk for depression from a single SNP. Pleiotropy checks were negative. Heterogeneity affected the bipolar results, and some Extraversion analyses had limited power. These points support Neuroticism as a broad liability factor, while Extraversion is outcome-specific.

Keywords : Mendelian randomization; Neuroticism; Extraversion; Personality traits; Psychiatric disorders; Causal inference

Count: 139
Abstract ID: 980
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

Effects of a Modified Psychobiotic Diet on Depression, Anxiety, and Eating Behavior in Overweight and Obese Adults with Depression

Submission Author: Reyhaneh Moradabbasi

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Background and Aim : Obesity and depression frequently co-occur and may influence each other through pathways involving the gut–brain axis and gut microbiota–derived metabolites such as trimethylamine-N-oxide (TMAO). The psychobiotic diet, designed to enhance the growth of mood-regulating gut microbes, that was culturally adapted to Iranian dietary habits. This study aimed to assess whether this modified psychobiotic diet could improve mood, eating behavior, and related biomarkers in individuals with depression.

Methods : In this single-blind, randomized controlled trial, adults with mild to moderate depression were assigned to either a psychobiotic diet or control group for 6 weeks. Depression and anxiety scores, eating behaviors, serum leptin and TMAO levels were measured at baseline and post-intervention.

Results : The psychobiotic diet significantly reduced depression scores and improved emotional and uncontrolled eating ($p<0.05$). Lipid profile, leptin and TMAO showed no significant change. Body weight decreased overall ($p=0.003$), but the group × time interaction was not significant.

Conclusion : A modified psychobiotic diet may improve mood and eating behavior in obese adults with depression, highlighting the potential role of gut microbiota modulation in mental health management.

Keywords : psychobiotic diet; depression; gut–brain axis; eating behaviors; anxiety

Count: 140
Abstract ID: 72
subject: Neuropsychiatry and Psychology: Mood Disorders
Presentation Type: Poster

The Dual Role of Nitric Oxide in Stress-Related Anxiety: Interactions with Acute Sleep Deprivation in Mice

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Background and Aim : Chronic stress exacerbates anxiety disorders, but the neurobiological mechanisms remain unclear. Given emerging links between sleep and anxiety, we investigated the role of nitrergic signaling in the effect of acute sleep deprivation on anxiety-like behavior of stressed mice.

Methods : Male NMRI mice (n=8/group) were subjected to chronic restraint stress for 4 weeks (3 hours/day) using rodent restrainers. Twenty-four hours after the final stress session, animals received intraperitoneal injections of either L-NAME (5 or 10 mg/kg) or saline control, followed immediately by 3 hours of acute sleep deprivation using the gentle handling method. Behavioral assessment in the elevated plus maze (EPM) was then conducted to evaluate anxiety-like behaviors

Results : Chronic stress reduced open-arm time [$F(3,28) = 3.24$, $p=0.037$] and entries [$F(3,28) = 3.70$, $p=0.023$], indicating anxiety. Intraperitoneal administration of a nitric oxide synthase inhibitor L-NAME (10 mg/kg) blocked stress-induced anxiety ($p<0.001$), while lower dose (5 mg/kg, i.p.) was anxiogenic. Remarkably, acute sleep deprivation (3h) attenuated the L-NAME response in the stressed mice on anxiety-like behaviors [interaction effect: $F(2,42) = 7.414$, $p = 0.002$). No treatment conditions affected locomotor activity.

Conclusion : This study reveals that nitric oxide mediates stress-induced anxiety in a dose-dependent manner, and acute sleep deprivation paradoxically mitigates this effect via nitrergic modulation. These findings nominate the NO pathway as a potential target for stress-related anxiety disorders, particularly in sleep-disturbed populations.

Keywords : Anxiety; Chronic stress; Sleep deprivation; L-NAME

Count: 141
Abstract ID: 303
subject: Neuropsychiatry and Psychology: Anxiety Disorders and PTSD
Presentation Type: Poster

The Effect of Ketamine on Anxiety- and Depression-Like Behaviors in Male BALB/C Mice Subjected to Chronic Social Defeat Stress

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Background and Aim : Major depressive disorder (MDD) and anxiety disorders are among the most prevalent psychiatric conditions worldwide, often associated with chronic stress exposure and limited responsiveness to conventional antidepressants (1). Ketamine, an NMDA receptor antagonist, has emerged as a promising fast-acting antidepressant, especially in treatment-resistant cases (2). However, its effects on comorbid anxiety and depression symptoms in chronic stress models remain under active investigation (3). The present study aimed to evaluate the behavioral effects of ketamine on anxiety- and depression-like behaviors in male BALB/C mice exposed to the Chronic Social Defeat (CSD) stress paradigm, a validated animal model that closely mimics aspects of human psychosocial stress.

Methods : Twenty-four adult male BALB/C mice were randomly divided into four groups (n = 6 per group): 1. Control (non-stressed, saline-injected), 2. Low-dose ketamine (10 mg/kg), 3. Medium-dose ketamine (20 mg/kg), and 4. High-dose ketamine (30 mg/kg). The CSD protocol was applied for 10 consecutive days using the resident-intruder model, where experimental mice were exposed to aggressive CD-1 resident mice to induce social defeat. Following the stress induction period, ketamine was administered intraperitoneally once daily for 7 consecutive days according to group assignment. Behavioral assessments were conducted using the Elevated Plus Maze (EPM) to assess anxiety-like behavior, the Open Field Test (OFT) to evaluate general locomotion and exploratory behavior, and the Forced Swim Test (FST) to measure behavioral despair associated with depressive phenotypes. Data were analyzed using one-way ANOVA followed by Tukey’s post hoc test in GraphPad Prism 9. A p-value of less than 0.05 was considered statistically significant.

Results : Ketamine administration significantly reduced both anxiety- and depression-like behaviors in a dose-dependent manner. In the EPM test, mice in the high-dose group spent

significantly more time in the open arms (67.4 ± 5.3 s) compared to the control group (30.2 ± 4.1 s), indicating reduced anxiety. In the OFT, the total distance traveled increased from 310.4 ± 21.7 cm in controls to 520.3 ± 28.6 cm in the high-dose group, reflecting enhanced exploratory behavior. In the FST, immobility time decreased from 205.6 ± 13.9 s in the control group to 108.5 ± 9.4 s in the high-dose group, demonstrating an antidepressant-like effect. The medium-dose group also showed significant improvements compared to control but was less effective than the high dose.

Conclusion : The present study demonstrates that ketamine produces significant anxiolytic and antidepressant effects in BALB/C mice subjected to chronic social defeat stress, with a clear dose-response relationship. These findings support ketamine's therapeutic potential for stress-induced affective disorders and encourage further research into its mechanisms of action and clinical applicability. Future investigations should examine molecular biomarkers, neuroplasticity changes, and potential side effects associated with repeated ketamine exposure.

Keywords : Ketamine, Anxiety, Depression, Chronic Social Defeat, BALB/C Mice, NMDA Antagonist, Behavioral Tests

Count: 142
Abstract ID: 391
subject: Neuropsychiatry and Psychology: Anxiety Disorders and PTSD
Presentation Type: Oral

Management of Separation Anxiety in Dogs: A Case Series with Pharmacological and Behavioral Approaches

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Background and Aim : Separation anxiety is one of the most common neurobehavioral disorders in dogs, characterized by excessive vocalization, destructive behaviors, inappropriate elimination, and self-directed behaviors when left alone. This condition significantly impacts both animal welfare and the human–animal bond. Evidence suggests that dysfunction in central neurotransmitters, particularly serotonin, plays a major role in its pathophysiology.

Methods : Three dogs aged between 3 and 6 years with clinical signs consistent with separation anxiety were evaluated. Detailed histories were obtained from the owners, and standardized behavioral assessment questionnaires were completed. Comprehensive medical and internal examinations were performed to exclude possible medical or systemic causes. Final diagnoses of separation anxiety were made. Treatment consisted of pharmacological interventions (fluoxetine, buspirone, or alprazolam at doses adjusted to body weight) combined with behavioral modification programs, including gradual desensitization to owner departure cues and positive reinforcement training.

Results : Over an 8-week follow-up period, all three dogs demonstrated significant behavioral improvement. Excessive barking and destructive behaviors were reduced by an average of 70%. Signs of stress, such as pacing and self-licking, were markedly diminished in two of the cases. The combination of pharmacological treatment with behavioral therapy proved more effective than pharmacological therapy alone. No severe adverse effects related to medication were reported during the study period.

Conclusion : This case series demonstrates that successful management of separation anxiety in dogs requires a multimodal strategy. Combining pharmacological agents targeting central neurotransmitter systems with structured behavioral modification can lead to substantial improvement in clinical signs and overall quality of life. Behavioral assessment tools are essential for both accurate diagnosis and monitoring of treatment outcomes.

Keywords : separation anxiety, canine neurobehavioral disorder, fluoxetine, buspirone, alprazolam, behavioral therapy

Count: 143
Abstract ID: 663
subject: Neuropsychiatry and Psychology: Anxiety Disorders and PTSD
Presentation Type: Oral

AI-Guided Digital Therapeutic with HRV Biofeedback and EMDR for Occupational Trauma in First Responders: A Feasibility Study

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Background and Aim : Post-traumatic stress disorder affects more than 30% of first responders, compared with approximately 20% in the general population (Abbot et al., 2015; Jones et al., 2018). These professionals face substantial barriers to conventional treatment. Stigma remains pervasive in emergency service cultures, specialized trauma-informed providers are scarce, and rotating shifts make regular therapy attendance challenging (Haugen et al., 2017; Stanley et al., 2017). Digital therapeutics could theoretically bridge these gaps (Kuhn & Owen, 2020); however, existing applications reveal significant limitations. Recent meta-analyses show that over one-third of users abandon these interventions (Linardon et al., 2019). Perhaps more concerning, rigid protocols may inadvertently cause harm when they push users beyond their physiological capacity for processing trauma, what clinicians term the window of tolerance (Siegel, 1999; Ogden et al., 2006). Our study investigates whether real-time heart rate variability (HRV) monitoring, combined with adaptive intervention selection, can overcome these limitations.

Methods : This feasibility study is intended to serve as a proof-of-concept investigation, employing multiple-baseline single-case methodology with three first responders who are experiencing work-related functional impairment (Maric & van der Werff, 2024). Rather than targeting specific diagnostic categories, this intervention addresses transdiagnostic mechanisms of autonomic dysregulation, aligning with contemporary dimensional frameworks for understanding psychopathology (Schneider & Schwerdtfeger, 2020). The progressive web application (PWA) combines validated self-report measures (including PCL 5) with continuous physiological monitoring through smartphone camera-based photoplethysmography (rPPG), a method that is validated at 98% sensitivity against clinical-grade equipment (Verkruysse et al., 2008). Most innovatively, we implemented a constrained Retrieval-Augmented Generation (RAG) engine that selects interventions from a library of evidence-based protocols. This

system draws from eye movement desensitization techniques and biofeedback exercises (Gevirtz & Dalenberg, 2018). But critically, it adjusts intervention intensity based on real-time stress indicators, operating as a closed-loop system designed to maintain participants within their therapeutic tolerance window (Siegel, 1999; Ogden et al., 2006).

Results : We set ambitious but achievable feasibility goals, including capturing usable physiological data from at least 70% of sessions, retaining 80% of participants throughout the study period, and, most importantly, experiencing no serious adverse events. The system has several fail-safes because safety is still of the utmost importance. Before being put into practice, every crisis protocol is clinically reviewed. The system is monitoring engagement patterns, perceived therapeutic alliance with the digital system, and initial symptom changes in addition to these key metrics. To make inferences regarding efficacy, more pilots are needed.

Conclusion : This investigation represents a shift from passive monitoring toward active, responsive digital intervention. Existing digital therapeutics often fail because they cannot recognize when users are becoming overwhelmed or disengaged. Our closed-loop system attempts to address this fundamental limitation by continuously adjusting to users' physiological states. While artificial intelligence (AI) guides protocol selection, human clinical judgment shapes every safety parameter and intervention boundary. This interdisciplinary approach, which combines clinical psychology, bioengineering, and computer science, may offer a path toward more effective and safer digital mental health tools. Success here would justify larger trials and eventual pursuit of regulatory approval as a prescription digital therapeutic.

Keywords : Digital Therapeutics; Post-Traumatic Stress Disorder (PTSD); Heart-Rate Variability (HRV); Eye Movement Desensitization and Reprocessing (EMDR)

Count: 144
Abstract ID: 705
subject: Neuropsychiatry and Psychology: Anxiety Disorders and PTSD
Presentation Type: Poster

Transcranial Photobiomodulation (tPBM) a Neurobiologic Protocol for Drug-Resistant Post-Traumatic Stress Disorder (PTSD): A Feasibility and Efficacy Study

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Background and Aim : Post-traumatic stress disorder is a chronic and debilitating psychiatric condition that is prevalent among combat veterans. A significant subset of patients develops a drug-resistant form, creating a pressing need for novel, non-pharmacological interventions. This study investigates the feasibility and preliminary efficacy of a combined protocol using transcranial photobiomodulation (tPBM) and neurobiological reprogramming techniques for war veterans with drug-resistant PTSD.

Methods : A pilot study was designed for 25 veterans with combat-related, drug-resistant PTSD. The intervention consisted of 10 sessions integrating active tPBM (applied to the right frontal and hippocampal regions) with structured neurobiological reprogramming exercises, including infra-low frequency neurofeedback. Primary outcomes were feasibility (recruitment, adherence, safety), while secondary outcomes were changes in PTSD symptom severity measured by the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5).

Results : The combined protocol was found to be feasible and well-tolerated. Preliminary analysis indicated a statistically significant reduction in CAPS-5 scores from baseline to post-intervention, with improvements particularly noted in hyperarousal and negative alterations in cognition and mood.

Conclusion : The integrated tPBM and neurobiological reprogramming protocol is a feasible and potentially effective intervention for war-related, drug-resistant PTSD. These promising results warrant further investigation through larger, randomized controlled trials.

Keywords : Post-Traumatic Stress Disorder, PTSD, Transcranial Photobiomodulation, tPBM, Neurobiological Reprogramming, Drug-Resistant, War Injuries.

Count: 145
Abstract ID: 436
subject: Neuropsychiatry and Psychology: Addiction (Drug, Alcohol, Internet, Food) and Gambling
Presentation Type: Oral

Cerebellum mechanisms associated with addiction

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Conclusion : Cerebellum mechanisms associated with addiction Cerebellum regions including Crus I, Lobule VI, and VIIb are all associated with brain regions involved in cognitive control, including: Dorsolateral prefrontal cortex and dorsomedial prefrontal cortex. Cerebellum may play an influential modulating role in reward/saliency, because it also interacts with dopaminergic systems in the basal ganglia. Functional correlations between these cerebellum regions and brain structures involved in executive control, craving, response selection, and salience further highlight the potential role of cerebellum in addiction. Addiction is a breakdown in motivational homeostasis, in reward and aversion processing and their modulation. Cerebellum can modulate the flow of aversion information and motivational processing. There is a model of cerebellum modulation of circuits involved in aversion and addiction. The main conceptual model for addiction is impaired Response Inhibition and Salience Attribution (i-RISA) model, which is also described as a four-circuit model involving these four interconnected circuits: Memory, reward/saliency, executive control, and motivation. The response to a reward is mediated by the interactions of these four components. The i-RISA is a syndrome characterized by attributing excessive salience to substances and substance-related cues; and reduced sensitivity to non-substance reinforcers and a reduced ability to inhibit maladaptive or undesirable behaviors. The cerebellum model of addiction proposes that cerebellum plays a critical role in maintaining homeostatic balance of four circuits. This model highlights the role of cerebellum as a modulator of emotional and cognitive function in healthy brain. In the brain of an addicted individual, structural gray matter deficits in Lobule VI, VIIb, Crus I, Crus II, and Vermis indicate alterations in the cerebellum's ability to interact and communicate with brain regions associated with the four circuits. Cerebellum is potentially a key modulator that both influences in addiction and is influenced by addiction. Cerebellum is associated with addiction. Alterations in cerebellum both predispose to and may play a key role in the course of addictive disorders. Understanding the cerebellum vulnerability factors for addiction has important implications for prevention and treatment of substance use disorders (SUD) and behavioral addictions.

Keywords : cerebellum, impaired Response Inhibition and Salience Attribution (i-RISA), substance use disorders (SUD), behavioral addictions

Count: 146
Abstract ID: 834
subject: Neuropsychiatry and Psychology: Addiction (Drug, Alcohol, Internet, Food) and Gambling
Presentation Type: Poster

Enduring effects of adolescent morphine exposure on locomotor activity and anxiety-like behavior in adult rats

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Background and Aim : Behavioral experiments and neurochemical data indicate increased anxiety and decreased exploratory behavior as the rat age from adolescence to old age. Adolescence is a critical neurodevelopmental period characterized by heightened risk-taking behavior, and vulnerability to substance use. Exposure to opioids during this period can induce long-lasting alterations in anxiety and exploratory behaviors, which may vary depending on the age at which the behaviors are assessed. However, the temporal trajectory of these behavioral consequences has been poorly understood. Here, anxiety-like behavior and locomotor activity were compared between adult (12 weeks) and older (18 weeks) rats after repeated exposure to morphine during adolescence.

Methods : Adolescent male Wistar rats received morphine sulfate (5 to 25 mg/kg) or saline subcutaneously once daily from postnatal day 28 to 37, and the morphine dose was increased by 5 mg/kg every two days in morphine treated rats. After 7 and 13 weeks without intervention, 12-week-old and 18-week-old adult rats from both groups were subjected to the open field test and elevated zero maze to assess locomotor activity and anxiety-like behavior.

Results : The average speed and total distance traveled in the open field were significantly higher in 12-week-old rats exposed to morphine compared to 12-week-old control rats. The average speed and total distance traveled were significantly decreased in 18-week-old rats exposed to morphine compared to 12-week-old rats exposed to morphine. Cumulative time spent in the open area were significantly increased in 18-week-old rats exposed to morphine compared to 12-week-old and 18-week-old control rats. Cumulative time spent in the closed area were significantly decreased in 18-week-old rats exposed to morphine compared to 12-week-old and 18-week-old control rats.

Conclusion : Results of this study demonstrated that behavioral consequences of adolescent opioid exposure are modulated by aging. Morphine exposure during adolescence resulted in a significant increase in locomotor activity and anxiety-like behavior in 12-week-old rats, while reducing locomotor activity and anxiety-like behavior in 18-week-old rats, indicating differential effects of adolescent morphine exposure on locomotor activity and anxiety-like behavior depending on age. Findings of current study may also help understand the long-term neuropsychiatric risks associated with early-life opioid use.

Keywords : adolescent morphine exposure; age-dependent behavioral effects; locomotor activity; anxiety-like behavior

Count: 147
Abstract ID: 71
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Neurotransmitters and Signaling Molecules
Presentation Type: Poster

The role of high-density lipoprotein and Vitamin D in exercise-induced neuroprotection in epileptic rats

Submission Author: Fariba Karimzadeh

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Background and Aim : Previous studies have reported the neuroprotective effects of exercise in epilepsy. Hence, this study aimed to investigate the potential mechanisms underlying these neuroprotective effects by examining changes in high-density lipoprotein (HDL), low-density lipoprotein (LDL), and Vitamin D levels.

Methods : Wistar rats were divided into four groups: sham, pentylenetetrazole (PTZ), exercise (EX), and PTZ + EX. Seizures were induced in the PTZ and PTZ + EX groups through intraperitoneal administration of PTZ (35 mg/kg) every other day for 1 month. The EX and PTZ + EX groups underwent daily exercise sessions (30 min/session) for 1 month.

Results : Serum levels of Vitamin D, HDL, and LDL were measured, and neuronal damage was assessed in the hippocampus and somatosensory cortex. The PTZ + EX group showed a significant increase in serum HDL and Vitamin D levels compared to the PTZ group. Histological analysis revealed a significant increase in neuronal damage in the PTZ group compared to the sham group. In contrast, both PTZ + EX and EX groups demonstrated a reduction in neuronal damage compared to the PTZ group.

Conclusion : These findings suggest that HDL and Vitamin D may contribute to the neuroprotective benefits of exercise. Furthermore, exercise could potentially serve as a complementary strategy to counteract Vitamin D and HDL deficiencies associated with long-term antiepileptic drug use.

Keywords : Exercise; Vitamin D; Lipoproteins; Hippocampus; Somatosensory cortex; Epilepsy

Count: 148
Abstract ID: 956
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Neurotransmitters and Signaling Molecules
Presentation Type: Poster

Interplay of Neuroinflammation and Epilepsy in Glioblastoma Multiforme: Mechanisms and Therapeutic Implications

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Conclusion : Glioblastoma multiforme (GBM) is the most aggressive form of primary brain cancer in adults and is characterized by poor prognosis and a high incidence of seizures due to tumor-induced alterations in cerebral physiology. This review explores the complex interactions between GBM-induced neuroinflammation and epilepsy, emphasizing the mechanisms of epileptogenesis influenced by blood–brain barrier dysfunction, ion homeostasis, and neurotransmitter dynamics. We discuss the roles of pro-inflammatory mediators such as interleukin-1β and tumor necrosis factor-alpha in exacerbating excitatory synaptic activity while inhibiting inhibitory signaling, thus creating a milieu conducive to seizure activity. Furthermore, we evaluated the efficacy of current anti-seizure medications and emerging therapeutic strategies, including the reprogramming of tumor-associated macrophages, in managing GBM-related epilepsy and tumor growth. This study aimed to elucidate the critical pathways connecting GBM and epilepsy, thereby advancing our understanding of potential interventional approaches to improve patient outcomes.

Keywords : Epilepsy, Glioblastoma Multiform, Neurel Excitability

Count: 149
Abstract ID: 75
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Channels, Receptors, Transporters,
Presentation Type: Poster

The Effect of Combined Treatment with Carbamazepine and Astaxanthin Supplement on the GABA Pathway in a Pentylenetetrazol-Induced Seizure Model

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Background and Aim : Seizure is one of the common neurological disorders in which the balance between excitatory and inhibitory systems of the brain is disrupted. The GABAergic pathway, as the main inhibitory system in the brain, plays an important role in controlling seizures. Carbamazepine is a common antiepileptic drug that acts by enhancing neuronal inhibition. Astaxanthin, a potent antioxidant with neuroprotective effects, may enhance anticonvulsant effects by reducing oxidative stress and strengthening GABA system function. In this study, we investigated the effect of combined treatment with carbamazepine and astaxanthin supplement on the GABA pathway in a pentylenetetrazol (PTZ)-induced seizure model.

Methods : Male Wistar rats were divided into nine groups: sham (receiving olive oil), seizure group, seizure groups treated with carbamazepine (25, 50, and 75 mg/kg), seizure group treated with astaxanthin (AXT, 4 mg/kg), and seizure groups receiving combination therapy of AXT (4 mg/kg) with carbamazepine (25, 50, and 75 mg/kg). Seizure behavior scores were recorded up to 30 minutes after each PTZ injection (35 mg/kg). Immunohistochemistry was performed to assess GAD65 expression in the CA1, CA3, dentate gyrus regions of the hippocampus, and the sensorimotor cortex.

Results : Seizure behavior scores in epileptic rats receiving AXT were reduced compared to the seizure group. Combined treatment with carbamazepine and AXT increased GAD65 expression in CA1, CA3, dentate gyrus, and cortex in the groups receiving AXT (4 mg/kg) with carbamazepine (25, 50, and 75 mg/kg), compared with the seizure group. The results showed that the combined therapy significantly increased seizure onset time and reduced seizure severity. The effects of combination therapy were stronger than monotherapies.

Conclusion : The combination of carbamazepine and astaxanthin can strengthen the GABAergic pathway and reduce seizure severity, suggesting its potential as an effective adjunct therapeutic approach in epilepsy management. The neuroprotective role of astaxanthin through mitigating the adverse effects of carbamazepine may contribute to this improvement.

Keywords : Seizure, Carbamazepine, Astaxanthin,Pentylenetetrazol

Count: 150
Abstract ID: 556
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Channels, Receptors, Transporters,
Presentation Type: Oral

Modulation of Mitochondrial Dynamics by 2-Deoxyglucose as a Neuroprotective Strategy in Temporal Lobe Epilepsy

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Background and Aim : Temporal lobe epilepsy (TLE) is a neurodegenerative disorder characterized by mitochondrial dysfunction, including altered mitochondrial dynamics that contribute to neuronal injury. 2-Deoxyglucose (2DG), known for its metabolic effects and antiepileptic properties, may protect mitochondrial integrity, but its impact on mitochondrial dynamics in TLE remains to be elucidated.
Methods : TLE was induced in rats by intracerebroventricular injection of kainic acid. A single dose of 2DG (125 mg/kg) was administered intraperitoneally 30 minutes prior to epilepsy induction and continued daily. Animals were sacrificed 24 and 72 hours post-induction. Neuronal survival in the hippocampal hilar region was assessed using Nissl staining. Quantitative real-time PCR measured mRNA expression of mitochondrial fission protein Fis1 and fusion protein Opa1. Immunohistochemistry evaluated levels of Drp1 and Mfn1 proteins. Mitochondrial membrane potential was also analyzed
Results : 2DG treatment significantly reduced neuronal necrosis in the hilar region following epilepsy. Epilepsy induced increased expression of mitochondrial fission markers (Fis1, Drp1) and decreased fusion markers (Mfn1, Opa1), indicative of mitochondrial fragmentation. 2DG reversed these changes by downregulating fission proteins and upregulating fusion proteins. Moreover, mitochondrial membrane potential, which was reduced after epilepsy induction, was restored close to control levels with 2DG administration.
Conclusion : Temporal lobe epilepsy disrupts mitochondrial dynamics by promoting excessive fission that contributes to neuronal damage. 2-Deoxyglucose exhibits a neuroprotective effect by modulating mitochondrial fission and fusion balance, preserving mitochondrial function and reducing neuronal necrosis. These findings suggest that regulation of mitochondrial dynamics is a key mechanism underlying the antiepileptic effects of 2DG and offers a promising therapeutic strategy for epilepsy-induced neurodegeneration.
Keywords : Temporal lobe epilepsy; 2-Deoxyglucose; Mitochondrial dynamics; Neuronal necrosis; Mitochondrial membrane potential; Kainic acid model

Count: 151
Abstract ID: 709
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Synaptic Transmission and Synaptic Plasticity
Presentation Type: Poster

Mutual functional interconnection of VTA and LC is important for producing of spike-timing dependent plasticity (STDP) or t-LTP

Submission Author: Firouz Ghaderi

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Background and Aim : Anatomical mutual connection of the ventral tegmental area (VTA) and locus coeruleus (LC) was revealed, but the functional roles of this connection are not well-known. The timing-dependent long-term potentiation (t-LTP) is defined in some brain areas, such as the hippocampus. The hippocampus is the ideal brain region to study LTP in vivo and in vitro, but the time-pairing of synaptic connections, such as spike-timing dependent potentiation (STDP) is a well-defined technique for t-LTP study in the VTA and LC. In this study, the PTSD was evaluated by pair-of-firing between VTA dopaminergic (DA) and LC noradrenergic (NE) neurons.

Methods : Wistar male rats (~270 g body weight) were anesthetized with urethane (1.5 gr/Kg initial dose, i.p., 0.5 g/kg/each hour boosters) and fixed in a stereotaxic frame (Steolting, USA). The bregma and interaural coordinates were used to approach the recording glass microelectrodes to the VTA (perpendicular) and LC (anteroposterior angle). Single neuron firings are amplified by two extracellular AC amplifiers (DAM 80, WPI) and digitized by a USB NI 6009 board, then stored on the PC by software (Wavemetrics Igor Pro) into two-channel recording simultaneously. Neuromatic software is used to evaluate Peri-Stimulus Time Histogram (PSTH) and Inter-Spike Interval Histogram (ISTH). Prazosin was applied ICV (1, 20, 50, 100, 250 µg/10 µL/1 min) for pharmacological manipulation. The spike-timing dependence plasticity (STDP) was studied by time-locking the NE and DA neurons' firing. The cross-correlations with Fisher's or x^2 test were used according to data distribution. The ANOVA and paired t-test were used for evaluation of point of correlation. The $P<0.05$ was the significance level.

Results : The results showed that prazosin (20, 50, 100) increased firing rates of DA-NE neurons amount-dependently, but amount 1 had no significant effect, and amount 250 decreased the firing of both neurons. The results revealed that NE-DA firings paired together. The changes in NE neurons' firing occurred 2.63+-0.34 msec beforehand of DA neurons' firing in average. The STDP data revealed that time-pairing of the NE and DA neurons occurred in 80% of recordings.

Conclusion : VTA and LC interactions exhibit notable neurochemical complexity. Both nuclei can influence prefrontal cortex functioning through dopamine and norepinephrine release mainly. Disruption of VTA-LC connectivity is implicated in disorders such as autism spectrum disorder, major depressive disorder, and drug addiction, where altered arousal or reward processing is observed. Pharmacological inhibition or activation affecting both dopamine and noradrenaline signaling. It revealed that time-pairing of the NE and DA neurons may be important for mutual interconnection and switching loops.

Keywords : Locus Coreless (LC), Noradrenergic (NE), DA releasing neurons (DAergic), Spike-Timing Dependence Plasticity (STDP), Peri-Stimulus Time Histogram (PSTH), Inter-Spike Interval Time Histogram (ISITH)

Count: 152
Abstract ID: 633
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Synaptic Transmission and Synaptic Plasticity
Presentation Type: Poster

Intracerebroventricular prazosin modulates noradrenergic and GABAergic locus coeruleus neurons to influence spike-timing-dependent plasticity

Submission Author: Delnia Mehrafarin

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Background and Aim : The locus coeruleus (LC) is the profound source of the norepinephrine (NE) in the brain. NE may affect mood, attention, and stress response. Projections of LC influence the activity of other regions of the brain and switches their activities in attention, perceptual processing, and memory retrieval. It composed of noradrenergic (NE-ergic) and GABA releasing (GABAergic) mainly. NE neurons have low- and sporadic firing and are modulated by different issues such as pharmacological agents. This study revealed the effect of intracerebroventricular (ICV) microinjection of alpha-1 adrenergic antagonist, prazosin, on the firing patterns of the NE-ergic and GABAergic neurons of LC.

Methods : Wistar male rats (~270 g) were anaesthetized with urethane (1.5 gr/Kg initial dose, i.p., 0.5 g/kg/each hour boosters) and fixed in a stereotaxic frame (Steolting, USA). The bregma and interaural coordinates used to approach the recording glass microelectrodes to the LC with anteroposterior angle. Single neuron firings was amplified by an extracellular AC amplifier (DAM 80, WPI) and digitized by USB NI 6009 board then stored on the PC by software (Wavemetrics Igor Pro). On-line monitoring attended then off-line sorting by Neuromatic software used to evaluate Peri-Stimulus Time Histogram (PSTH) and Inter-Spike Interval Histogram (ISIH) criteria and NE/GABA-ergic populations. Prazosin administered ICV in amounts of 1, 20, 50, 100, 250 µg in 10 µL over 1 minute. The spike-timing dependence plasticity (STDP) was studied by pairing of the NE-ergic and GABAergic neurons' firings. Data analyzed with one-way analysis of variance (ANOVA) and post-hoc Newman-Keuls tests with $P<0.05$ significance level.

Results : The results showed that PSTH and ISIH of the NE-ergic and GABAergic neurons changed and increased after prazosin (20, 50, 100) in an amount-dependent manner but the amount 1 had no significant effect and amount 250 decreased the firing of both neurons. The STDP data revealed that pairing of the NE-ergic and GABAergic neurons occurred between 25% of NE and GABA–ergic neurons. The regular firing of NE-ergic neurons changed to train of impulse and the GABA-ergic neurons initiated firing due to prazosin application. Time pairing of the NE and GABA neurons may indicate the t-LTP by STDP Hebbian relation.

Conclusion : LC neurons project as a diffuse neuronal system and switch many functional behaviors. The NE-ergic neurons change their firings to modulate targets but their modulation remains unclear. intra-LC circuitry between NE-ergic/GABAergic neurons is postulated as a key process of the LC synaptic outputs. The data of the present study revealed that intra-LC circuitry may play some important roles to determine the highly developed neuronal LC outputs. Microcircuits between NE-GABA-ergic neurons proposed as a modular unit.

Keywords : Locus Coreless (LC), Noradrenergic (NE), GABA releasing neurons (GABAergic), Spike-Timing Dependence Plasticity (STDP), Peri-Stimulus Time Histogram (PSTH), Inter-Spike Interval Time (ISIH)

Count: 153
Abstract ID: 634
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Synaptic Transmission and Synaptic Plasticity
Presentation Type: Poster

Investigating the effect of intracerebroventricular injection of Prazosin on modulation and spike-timing-dependent plasticity of the ventral tegmental area neurons in rats

Submission Author: SeyyedeDiman Mahdi

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Background and Aim : The ventral tegmental area (VTA) is an important region for reward, motivation, and goal-directed behavior. It comprises dopaminergic (DAergic) and GABA releasing (GABAergic) mainly. The firing patterns of the VTA DAergic neurons show tonic (slow, steady) and phasic (bursting). The firing of VTA dopamine neurons is closely linked to the inputs but modulated by various factors. Although systemic injection of prazosin modulates the VTA dopamine neurons but the mechanism is not well known. This study evaluated the firing pattern of VTA neurons by intracerebroventricular (ICV) microinjection of alpha-1 adrenergic antagonist, prazosin.

Methods : Male Wistar rats (~270 g) were anaesthetized with urethane (1.5 gr/Kg initial dose, i.p., 0.5 g/kg/each hour boosters) and fixed in a stereotaxic frame (Steolting, USA). The bregma and interaural coordinates used to approach the recording glass microelectrodes to the VTA. Single neuron firings are amplified by an extracellular AC amplifier (DAM 80, WPI) and digitized by USB NI 6009 board then stored on the PC by software (Wavemetrics Igor Pro). On-line monitoring attended then off-line sorting by Neuromatic software used to evaluate Peri-Stimulus Time Histogram (PSTH) and Inter-Spike Interval Histogram (ISIH) criteria and DAergic-GABAergic populations. Prazosin applied ICV (1, 20, 50, 100, 250 µg/10 µL/1 min). The spike-timing dependence plasticity (STDP) was studied by pairing of the DAergic and GABAergic neurons' firings. Data analyzed with one-way analysis of variance (ANOVA) and post-hoc Newman-Keuls tests with $P<0.05$ significance level.

Results : The results showed that PSTH and ISITH of the DA-ergic and GABAergic neurons changed after prazosin (20, 50, 100, 250) in non-dose-dependent manner. Dose 50 and 100

showed profound effects and made burst firing in the DAergic neurons and changed PSTH and ISITH of the DAergic and GABAergic neurons significantly. The STDP data revealed that pairing of the DAergic and GABAergic neurons occurred between 40% of DA and GABA –ergic neurons. 20% of DA neurons showed unchanged PSTH but remaining changed. All DA neurons showed ISITH changing.

Conclusion : Many studies showed that inputs to the VTA are key activators of the intra-VTA DA neurons but intra-VTA circuitry between DAergic-GABAergic neurons is not well-documented. The data of the present study focused on the intra-VTA circuitry and It postulated that intra-VTA GABAergic neurons are the key neuronal elements to change the DA release and may be a goal to different inputs. The changed output of the VTA-DA determined by intra DA-GABA –ergic microcircuits.

Keywords : Ventral Tegmental Area (VTA), Dopaminergic (DA), GABA releasing neurons (GABAergic), Spike-Timing Dependence Plasticity (STDP), Peri-Stimulus Time Histogram (PSTH), Inter-Spike Interval Histogram (ISIH

Count: 154
Abstract ID: 850
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Synergistic Neuroprotective Effects of Sinomenine and Angelica Extract in a Rat Model of Penicillin-Induced Epilepsy

Submission Author: Amirhossein Heidari

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Background and Aim : Epilepsy involves recurrent neuronal hyperexcitability, leading to neuroinflammation, oxidative stress, and behavioral impairments, particularly within cortical and hippocampal regions. Sinomenine, a bioactive plant alkaloid, and Angelica extract, enriched with phenolic and terpenoid compounds, possess potent anti-inflammatory and antioxidant activities. This study investigated their individual and combined neuroprotective efficacy in penicillin-induced epilepsy in rats.

Methods : Epileptic seizures were induced via epicortical or intraventricular injection of penicillin. Animals were allocated into eight groups, including control, penicillin, Sinomenine, Angelica, and their combinations. Behavioral assessments included the Elevated Plus Maze (EPM) and Light–Dark Box (LDT) for anxiety-like behavior, Tail Suspension (TST) and Forced Swimming Test (FST) for depressive responses, and the Morris Water Maze (MWM) for spatial memory. Histopathological analyses of cortical and hippocampal tissues were conducted using H&E, Cresyl Violet, and Luxol Fast Blue staining.

Results : Penicillin-treated rats exhibited pronounced anxiety, depressive behaviors, memory deficits, and neuronal degeneration. Administration of Sinomenine or Angelica significantly ameliorated these alterations, while co-treatment produced superior outcomes, characterized by reduced neuronal pyknosis, preserved hippocampal architecture, and attenuated demyelination.

Conclusion : Combined therapy with Sinomenine and Angelica extract exerts potent neuroprotective, anxiolytic, and antidepressant effects against penicillin-induced epileptic damage. These benefits may arise through modulation of oxidative and inflammatory pathways, supporting their potential as adjunctive agents in epilepsy management.

Keywords : Sinomenine; Angelica extract; Epilepsy; Neuroprotection; Behavioral tests; Hippocampus; Oxidative stress

Count: 155
Abstract ID: 514
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Analysis of Synchronization During the Pre-Ictal And Interictal Periods In Patients with Epilepsy

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Background and Aim : In epilepsy, synchronization between brain region measured by phase alignment in EEG signals varies over time and is linked to seizure onset and spread. Studying these changes before seizures helps predict them and supports seizure warning systems, as well as tracking epilepsy progression after the first seizure.

Methods : Data from five children in the CHB-MIT dataset, who discontinued medications under supervision for scalp EEG recording were analyzed at the Department of Physics of the Institute for Advanced Studies in Basic Sciences. Synchronization was assessed via phase-locking value (PLV) across brain rhythms. Baseline intervals and windows near seizure onset were compared to examine PLV ratio changes. Additionally, synchronization was studied in 556 individuals: 109 with a single seizure and 447 diagnosed with following epilepsy at the stage of the first seizure. Data were collected at the Department of Paediatric Neurology, Belarusian State Medical University. Statistical analysis used Fisher's exact two-tailed test.

Results : PLV analysis across rhythms showed a significant decrease in synchronization approaching the ictal phase in patients with epileptic seizures. This reduction indicates diminished phase coherence between brain signals and may precede abrupt neural network dynamics changes. This decline was more pronounced in patients with epilepsy (3.4% presence of hypersynchronous EEG) compared to patients with a single seizure (6.4% accordingly), ? Fisher<0.005.

Conclusion : Our study suggests that decreased phase locking in the pre-seizure period reflects the nervous system's efforts to inhibit seizure activity through inhibitory mechanisms and dynamic neural network changes.

Keywords : Pre-Ictal, Synchronization, Fisher's Methods, Phase-Locking Value

Count: 156
Abstract ID: 831
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Pre-Seizure ECG Signatures of Different Seizure Types: A Time-Frequency and Nonlinear Machine Learning Approach

Submission Author: SEYEDEH DORSA AKHAVAN MAROUFI

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Background and Aim : Epilepsy affects 50–65 million people worldwide and is marked by unpredictable seizures that can cause serious injuries such as head trauma and fractures. Physiological changes occur before seizure onset and can be detected using wearable technologies. While EEG is the standard diagnostic tool, it is often uncomfortable for continuous monitoring. Wearable devices, such as smartwatches, can reliably capture ECG signals, which may contain hidden information due to complex heart-body interactions. By analyzing these signals in real time with AI models, seizure onset can potentially be predicted before it occurs, offering a non-invasive and practical approach for early warning and intervention.

Methods : To develop our model, we used the SeizeIT2 dataset, which contains 11,640 hours of wearable ECG data from 125 epileptic patients, including 818 seizure events across various seizure types. For analysis, we segmented 400-second windows preceding seizure onset. We focused on the following seizure types: Focal impaired-awareness motor seizures with automatisms, focal impaired-awareness motor seizures with hyperkinetic activity, Focal aware motor seizures with hyperkinetic activity, and Focal impaired-awareness non-motor seizures. Only high-quality, artifact-free segments were included. From each segment, we extracted 105 physiological features related to cardiac and respiratory activity, grouped into four categories: ECG-derived respiratory measures, time-domain features, frequency-domain features, and nonlinear features. For dimensionality reduction, we applied Linear Discriminant Analysis (LDA) and Principal Component Analysis (PCA), finding that LDA provided better visual separation of the data. Classification of seizure types was then performed on the transformed data using Support Vector Machine (SVM), Random Forest, and XGBoost classifiers. Finally, we calculated the importance of each feature to identify those most relevant for distinguishing between seizure types.

Results : We evaluated the performance of three classifiers for distinguishing seizure types. Random Forest achieved a 5-fold cross-validation (CV) accuracy of 0.8692 ± 0.0392, with a test accuracy of 0.9091. XGBoost reached a 5-fold CV accuracy of 0.9000 ± 0.0671, with a test accuracy of 0.8485. Support Vector Machine (SVM) achieved a test accuracy of 0.9091, with a 5-fold CV accuracy of 0.9078 ± 0.0678, indicating that SVM performed best overall. Feature importance analysis of the SVM model revealed that the top five discriminative features were HRV_MFDFA_alpha2_Fluctuation (approximate importance: 501.60), HRV_MFDFA_alpha1_Fluctuation (250.36), HRV_VHF (158.96), HRV_VLF (118.99), and HRV_CVNN (83.12). These results indicate that nonlinear measures of heart rate variability, as well as high- and very-low-frequency HRV features, are the most relevant for distinguishing between seizure types.

Conclusion : The top HRV features identified by our SVM model reflect distinct autonomic signatures preceding different seizure types. HRV_MFDFA_alpha2 and alpha1 capture long- and short-term heart rate correlations, indicating shifts in cardiac complexity depending on motor activity and awareness. VHF and VLF components reflect parasympathetic and sympathetic modulation, while HRV_CVNN highlights overall heart rate variability, together revealing type-specific pre-ictal autonomic patterns. These ECG-derived markers can be applied in smartwatches or specialized devices to predict seizures, reduce injury risk, and guide interventions like vagus nerve stimulation (VNS). Beyond patient care, they hold significant industrial potential for developing next-generation wearable health technologies and seizure-monitoring devices with real-world industrial applications.

Keywords : Seizure; ECG; pre-seizure prediction; Machine learning

Count: 157
Abstract ID: 395
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Oral

The evaluation of small molecules' effects on self-renewal and differentiation of neural stem cells into inhibitory neurons and the assessment of transplantation in a rat temporal lobe epilepsy model

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Background and Aim : Improving neuronal differentiation after stem cell transplantation by limiting astrocyte differentiation is key to advancing epilepsy therapies. Various methods can induce neuronal differentiation. Small molecules effectively target developmental signaling pathways, which are crucial for neuronal maturation. Prioritizing shorter induction times and higher neuronal differentiation rates is essential when selecting an approach. Thus, we explore two hypotheses: first, that small molecules can affect astrogliosis, which is the presence of astrocytes derived from patients with epileptic tissue; and second, that these molecules can enhance neuronal differentiation while decreasing the presence of astrocytes in human embryonic neural stem cells.

Methods : To evaluate the effects of small molecules on astrocyte survival, we isolated and cultured astrocytes derived from human brain tissue. These cells provide valuable insight into pathological changes. To assess the efficacy of small molecules on neuronal induction, we conducted in vitro evaluations, including neurite outgrowth measurements, assessments of stemness properties (neurosphere assay), and differentiation studies (immunocytochemistry). Additionally, to examine the functional integration of stem cells treated with small molecules, we evaluated electrophysiological properties, synaptogenesis, and inhibitory characteristics through electrocorticography and immunohistochemistry (synaptophysin and GAD67). Lastly, astrocyte differentiation was assessed using the GFAP marker in the CA3 area of the hippocampus across experimental groups.

Results : We assessed the effects of three small molecules on the viability of astrocytes derived from human brain tissue affected by epilepsy, as well as on the neuronal differentiation of

human embryonic neural stem cells, both in vitro and in vivo. Our findings revealed that astrocytes from human brain tissue do not express GFAP; however, they do express vimentin and fibronectin. Additionally, the viability of these astrocytes varied in response to the small molecules, with amygdala-derived astrocytes exhibiting higher responsiveness than those from the cortex and hippocampus. Astrocytes in the cortex are more resistant to the small molecules. In contrast, VPA significantly influences nestin expression compared to forskolin and CHIR. Notably, we found that a combination of the three small molecules, referred to as a cocktail, enhanced the diameter of neurospheres. Immunocytochemistry data indicated that in the forskolin-treated groups, MAP2 expression increased while GFAP levels decreased. Furthermore, our data suggest that BDNF can help reduce GFAP expression in vitro. In our in vivo studies, we observed that forskolin minimizes the number of astrocytes while improving the expression of GAD67.

Conclusion : This study explores the impact of small molecules (CHIR, VAP, Forskolin) on astrogliosis and neuronal differentiation in human neurons and astrocytes. Astrocytes derived from epileptic tissue exhibited expression of vimentin and fibronectin but not GFAP, with cells from the amygdala demonstrating greater viability compared to those from the cortex and hippocampus. VPA influenced nestin expression, while the combination of small molecules increased the diameters of neurospheres. Forskolin was found to enhance MAP2 expression and reduce GFAP levels, with BDNF also contributing to the reduction of GFAP. In vivo, forskolin decreased the number of astrocytes and increased the expression of GAD67. Further studies are needed to investigate the effects of small molecules on neuronal differentiation.

Keywords : Epilepsy, Temporal Lobe, Neural Stem Cells, Small Molecule, Cell Differentiation.

Count: 158
Abstract ID: 241
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Oral

The Effects of Seizures on Sperm Parameters and Sperm DNA Damage

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Background and Aim : Seizures, a neurological disorder characterized by abnormal and excessive electrical discharges in the brain, can negatively affect male reproductive health, specifically impacting sperm parameters and causing sperm DNA damage. The present study investigated the effect of seizures on male reproductive health.

Methods : Sperm samples were collected from 20 men with epilepsy who had experienced regular seizures. A comprehensive analysis was performed to assess sperm count, motility, viability, and morphology using standard methods. To evaluate sperm DNA integrity, the Sperm Chromatin Dispersion (SCD) assay was used to determine the level of DNA fragmentation. The results were then compared to a healthy control group.

Results : The findings showed that men with seizures had a significantly lower sperm count, reduced motility and viability, and a higher percentage of abnormal morphology compared to the control group. Furthermore, the level of sperm DNA damage was notably higher in the seizure group. A positive correlation was observed between the frequency of seizures and the degree of sperm DNA damage, suggesting that more frequent seizures may lead to greater DNA fragmentation.

Conclusion : This study concludes that the damage to male fertility caused by seizures is likely the result of a complex and multifactorial process, including oxidative stress, hormonal imbalances, and possibly direct effects on the testes. Understanding these mechanisms is essential for developing more effective therapeutic and counseling strategies for men with epilepsy.

Keywords : Seizures; Male Fertility; Sperm Parameters; Sperm DNA Damage

Count: 159
Abstract ID: 242
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

The Impact of Hypothalamic-Pituitary-Gonadal Axis Dysfunction on Sperm Chromatin Maturation and DNA Damage in Men with Seizures

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Background and Aim : Seizures can negatively affect male reproductive health by disrupting the hypothalamic-pituitary-gonadal (HPG) axis and inducing oxidative stress. This study aimed to investigate the relationship between HPG axis dysfunction, characterized by elevated cortisol levels, and its effects on sperm chromatin maturation and DNA integrity in men with frequent seizures.

Methods : In this study, 20 men with frequent seizures, aged 25 to 37, and with elevated cortisol levels, participated. Sperm samples were collected from each participant. Sperm chromatin maturation was evaluated using the SCMA (Sperm Chromatin Maturation Assay) according to the standard protocol of Aniline Blue staining. Additionally, the Sperm Chromatin Dispersion (SCD) assay was used to determine the level of sperm DNA fragmentation. The results were compared to those of a healthy control group.

Results : Our findings revealed that in the seizure group, sperm chromatin maturation was significantly reduced, which was characterized by a higher percentage of retained histones. Furthermore, the level of sperm DNA damage and fragmentation was notably higher in this group. A significant negative correlation was observed between cortisol levels and sperm chromatin maturation, while a significant positive correlation was found between cortisol levels and the degree of sperm DNA damage.

Conclusion : This study concludes that elevated cortisol, resulting from frequent seizures, suppresses the reproductive axis and increases oxidative stress, leading to reduced testosterone, insufficient chromatin maturation, and sperm DNA damage. These findings emphasize the importance of stress management and hormonal control as key strategies for preserving the reproductive health of men with epilepsy.

Keywords : HPG axis; Cortisol; Seizures; Sperm Chromatin Maturation; Sperm DNA Damage

Count: 160
Abstract ID: 648
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

A meta-analytic evaluation of the efficacy and safety of levetiracetam for treating myoclonic seizures

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Background and Aim : Levetiracetam (LEV) is a broad-spectrum antiseizure medication that is effective and safe in myoclonic seizures. This study aims to systematically review and meta-analyze the literature using LEV in myoclonic seizures and its efficacy and safety profile.

Methods : The included literature is updated till February 2024. Joanna Briggs Institute checklists were utilized for quality assessment. Publication details, general characteristics, and the details of the LEV treatment were extracted from the included papers. Finally, the data was meta-analyzed and publication bias was assessed.

Results : 52 studies were included in the meta-analysis. Considering the reduction of seizures by more than 50% after the administration of LEV as a responder rate, 59.72% of patients with myoclonic seizures significantly responded. Moreover, a significant percentage of the patients (37.82%) became seizure-free after the treatment. 13.86% of the patients discontinued the consumption of LEV due to adverse effects, lack of remission of the seizure, or unwillingness. Pre- to post-data analysis in the LEV group demonstrated a significant decline of 0.9 times in the mean number of myoclonic seizures per month.

Conclusion : LEV significantly reduced the occurrence of myoclonic seizures. Furthermore, it is well-tolerated and responsible for a few side-effects. However, it is necessary to monitor carefully LEV's individual response.

Keywords : Levetiracetam, Myoclonic seizures, Myoclonus, Epilepsy, Antiseizure medications (ASM), Systematic review, Meta-analysis.

Count: 161
Abstract ID: 15
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Oral

Apigenin reduces spontaneous epileptic spikes via suppression of caspase-3 activation in temporal lobe epilepsy model

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Background and Aim : Introduction: Temporal lobe epilepsy (TLE) is one of the most common forms of drug-resistant epilepsy, characterized by spontaneous recurrent seizures and interictal spikes. In addition to abnormal electrical activity, apoptosis is a key pathological feature of TLE. Among the molecular mediators of apoptosis, caspase-3 is recognized as a central executioner enzyme, contributing to neuronal death following seizures. Therefore, this study aimed to investigate the effects of apigenin on the frequency of spontaneous epileptic spikes and caspase-3 activation in a KA-induced model of TLE.

Methods : Method: 28 male Wistar rats were randomly divided into 4 groups: Control/ vehicle, apigenin treated-sham, KA and apigenin+KA. TLE was induced by intracerebroventricular injection of KA. Apigenin was administered by gavage as a pretreatment for 7 days at a dose of 50 mg/kg. intracranial electroencephalographic (iEEG) recordings were used to evaluate the frequency of spontaneous interictal spikes. Caspase-3 expression was evaluated using immunohistochemistry in hippocampal tissues of experimental groups.

Results : Results: Apigenin pretreatment significantly reduced the frequency of spontaneous epileptic spikes compared to the KA group ($P < 0.05$). Immunohistochemical analysis also revealed a marked decrease in caspase-3 expression in the hippocampus of apigenin-pretreated rats ($P < 0.01$).

Conclusion : Conclusion: In conclusion, apigenin demonstrated significant anticonvulsant and neuroprotective effects in a KA-induced model of TLE. the reduction in spike frequency may be attributed to the inhibitory effect of apigenin on caspase-3 activity. These findings support the potential therapeutic role of apigenin in epilepsy management.

Keywords : Keywords: Apigenin, Temporal lobe epilepsy, iEEG, Spontaneous epileptic spikes, Caspase-3

Count: 162
Abstract ID: 179
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Examining Artemisia absinthium Aqueous Extract's Anti-Epileptic Effect on Pentylenetetrazol-Induced Seizures in Male Rats

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Background and Aim : Epilepsy represents a widespread neurological disorder that significantly influences the quality of life of individuals afflicted by this condition. Consequently, the present investigation sought to evaluate the impact of the aqueous extract of Artemisia absinthium on seizures elicited by pentylenetetrazole (PTZ) in male rats.

Methods : A total of thirty male rats were randomly chosen and allocated into five distinct groups: a control group, a vehicle group, and three experimental groups. The treated rats received an aqueous extract of Artemisia absinthium through intraperitoneal injection at varying dosages of 50, 100, and 200 mg/kg prior to the administration of pentylenetetrazole (PTZ). Subsequently, thirty minutes after the administration of the herbal extract or distilled water (for the vehicle group), PTZ (80 mg/kg) was injected intraperitoneally. The subjects were then placed in a specialized enclosure, and a camera monitored their behaviors for a duration of thirty minutes to document their responses. Following this observation period, the latency to seizure onset, the duration of stage 5 seizures, as well as the incidence rate and percentage of mortality, were quantified across the groups. The collected data were subjected to analysis via a one-way ANOVA test alongside Tukey's post-hoc tests. Furthermore, a significance threshold of $P<0.05$ was established for determining statistical relevance.

Results : The results showed a significant delay in reaching stages 4 ($P<0.01$) and 5 ($P<0.001$) of tonic-clonic seizures at a dose of 100 mg/kg hyssop extract, along with a reduction in the duration of stage 5 ($P<0.05$) and a lower mortality rate.

Conclusion : Intraperitoneal administration of the aqueous extract of Artemisia absinthium exhibits anticonvulsant properties against PTZ-induced seizures in a dose-dependent manner. Consequently, further studies are suggested to determine the specific compounds in this plant responsible for its seizure-controlling effects.

Keywords : Aqueous extract of Artemisia absinthium, Pentylenetetrazole Rat, Seizure.

Count: 163
Abstract ID: 497
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Anticonvulsant Effects of Epirizole on Behavioral and Motor Responses in PTZ-Induced Seizure Rats

Submission Author: Helena Salimy

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Background and Aim : This study evaluated the behavioral and motor responses of male Wistar rats subjected to pentylenetetrazole (PTZ)-induced seizures to assess the anticonvulsant effects of Epirizole (EP).

Methods : Twenty male wistar rats (approximately 200 g) were randomly assigned into Four groups (n = 5 per group): control, PTZ, and two EP-treated groups. The control group received 0.3 mL saline injections at a 30-min interval, whereas the PTZ group followed the same protocol with 0.3 mL PTZ (60 mg/kg, ip). The two treatment groups received EP at 5 and 10 mg/kg for the first injection, followed by PTZ after the 30-min interval. Seizure behaviors were recorded using the modified Racine scale, which categorizes behavioral manifestations including head nodding, full-body shakes, forelimb clonus, rearing with forelimb clonus, falling, and jumping.

Results : The PTZ group exhibited frequent myoclonic jerks (NMJ) with short latencies (MJL) and prolonged generalized tonic–clonic seizures (GTCS-D), indicating severe convulsive behavior. Pretreatment with EP at 5 and 10 mg/kg significantly reduced NMJ ($p < 0.05$) and GTCS duration ($p < 0.05$), while markedly increasing MJL and GTCS latency ($p < 0.05$), compared with the PTZ group.

Conclusion : Overall, EP significantly reduced seizure severity and improved seizure thresholds in a dose-dependent manner. Given the high prevalence of drug-resistant epilepsy, EP may represent a promising therapeutic option.

Keywords : Epirizole, Non-steroidal anti-inflammatory drug, Pentylenetetrazol, Seizure, Rat

Count: 164
Abstract ID: 728
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Investigation of the Effects of Nanocuminaldehyde on Anxiety and Depression-Like Behaviors in PTZ-Induced Epileptic Mice

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Background and Aim : Epilepsy is one of the most common chronic neurological disorders, affecting millions of people worldwide. The uncontrolled neuronal depolarization is the main cause of epilepsy, which leads to altered membrane permeability, reduced inhibitory control, and an imbalance in neurotransmitter systems. Epilepsy is often accompanied by seizures and a variety of neuropsychiatric deficits. Evidence shows that patients with epilepsy experience a higher prevalence of anxiety and depressive symptoms compared to the general population. Although conventional antiepileptic drugs are effective for many individuals, adverse effects and drug resistance highlight the need for novel therapeutic strategies. Cuminaldehyde, a natural compound with antioxidant and neuroprotective properties, has been documented to improve behavioral impairments. Moreover, nanocapsulation technology can improve the stability, bioavailability, and targeted delivery of this compound. This study aimed to investigate the antidepressant and antianxiety effects of nanocapsulated cuminaldehyde on behaviors in a pentylenetetrazole (PTZ)-induced epilepsy model in mice.

Methods : In this experimental study, twenty-eight male mice were randomly divided into four groups (n=7 per group): control, nanocapsulated cuminaldehyde, PTZ, and pretreatment with nanocapsulated cuminaldehyde. The control group received distilled water orally. The second group was administered nanocapsulated cuminaldehyde orally (50 mg/kg) every other day for 21 consecutive days. The last two groups received intraperitoneal injections of PTZ (35 mg/kg) for 21 consecutive days, and the last group was pretreated orally by nanocapsulated cuminaldehyde (50 mg/kg) 30 minutes before PTZ injections. Finally, anxiety and depressive-like behaviors were evaluated using the open field test (OFT) and the tail suspension test (TST), respectively. The data were analyzed by one-way ANOVA.

Results : In the TST, the PTZ group showed a significant increase in immobility time compared with the control group ($p<0.001$), indicating enhanced depressive-like behavior. In

contrast, pretreatment with nanocapsulated cuminaldehyde significantly reduced immobility time and restored it to a level close to the control group ($p<0.001$). In the OFT, the PTZ group exhibited significantly increased locomotor activity compared to the control group ($p<0.0001$). while pretreatment with nanocapsulated cuminaldehyde markedly reduced locomotor activity relative to the PTZ group ($p<0.001$), reaching levels comparable to those of the control group. This indicates the occurrence of anxiety-like behaviors.

Conclusion : These findings suggest that nanocapsulation of cuminaldehyde can markedly enhance its neuroprotective effects. Nanocapsulated formulation successfully attenuated anxiety and depressive-like behaviors induced by epilepsy. Therefore, nanocapsulated cuminaldehyde may represent a novel and low-side-effect therapeutic approach for managing anxiety and depression in epileptic patients. Further studies in animal models and eventually clinical trials are warranted to confirm these findings.

Keywords : Epilepsy, Depression, Anxiety, Pentylenetetrazole, Cuminaldehyde

Count: 165
Abstract ID: 472
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Emorfazone's Potential to Attenuate Seizure Related Behavior in Pentylenetetrazol-Induced Seizure Model in Male Wistar Rats

Submission Author: Fatemeh Fayaz Sarcheshmeh

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Background and Aim : Seizure is a sudden, transient disruption of normal brain activity caused by abnormal electrical discharges, leading to involuntary movements, sensory changes, or altered consciousness. Emorfazone (EM) is a non-steroidal anti-inflammatory drug (NSAIDs) that has been used in experimental studies to reduce inflammation and pain symptoms. This study evaluated the anticonvulsant efficacy of EM in male Wistar rats subjected to pentylenetetrazol (PTZ)–induced seizures.

Methods : In this experimental study, 20 male Wistar rats were prepared and randomly allocated into 4 groups, each group contains 5 rats (Weighing between 200-250 g): a healthy control group that only received proper amount of physiological saline, a PTZ-treated group that received physiological saline followed by PTZ and two treatment groups receiving different doses (100 and 200 mg/kg) of EM via intraperitoneal (I.P.) injection, respectively. 30 minutes post-treatment, all groups except the healthy control received PTZ (60 mg/kg) via I.P. injection. Seizure behavior was monitored and recorded for 30 min based on the Racine scale.

Results : The results of the study indicated that pre-treatment with EM significantly prolonged the stages and delay of seizures compared to the PTZ group ($P < 0.05$) and notably reduced the duration of generalized tonic-clonic seizures ($P < 0.05$), as well as decreased the number of myoclonic jerks and seizures ($P < 0.05$).

Conclusion : Based on the results of this research, the EM injection can effectively delay and reduce seizure behavior in treated rats. It is suggested that since EM is an anti-inflammatory drug in this study, inflammatory and molecular pathways should also be investigated to strengthen the research.

Keywords : Emorfazone, Non-steroidal anti-inflammatory drug, Pentylenetetrazol, Seizure, Animal seizure model.

Count: 166
Abstract ID: 735
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Oral

Investigating the Combined Effect of Aerobic Exercise and Topiramate on Effective Drug Dosage, 5-HT1A Receptor Changes, and Depressive-like Behavior in a Pentylenetetrazol-Induced Seizure Model

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Background and Aim : Epilepsy is frequently associated with comorbid depression, necessitating novel therapeutic strategies that address both conditions. This study investigated the combined effects of aerobic exercise and topiramate on seizure threshold, depressive-like behavior, and hippocampal 5-HT1A receptor expression in a pentylenetetrazol (PTZ)-kindled rat model.

Methods : One hundred male Wistar rats (250-300 g) were randomly allocated to ten groups (n=10/group). Groups included: control (vehicle), PTZ-kindled (seizure induction every other day for one month), exercise (30-minute moderate treadmill running every other day), topiramate treatment (25, 50, 100 mg/kg, 30 minutes before PTZ), and combined therapy (topiramate followed by exercise). Depressive-like behavior was assessed using the Elevated Plus Maze (EPM). Seizure latency was recorded following PTZ administration. The density of 5-HT1A receptors in the hippocampus was evaluated using immunohistochemistry. Data were analyzed with SPSS (significance level: $p<0.05$).

Results : In the EPM test, the combined therapy groups showed a significant reduction in the number of entries into and time spent in the closed arms compared to the topiramate-only groups. Furthermore, seizure latency was significantly longer in the combined therapy groups than in the drug-only groups. Immunohistochemical analysis revealed a significant upregulation of hippocampal 5-HT1A receptor expression in the combined therapy groups compared to all other groups.

Conclusion : The combination of aerobic exercise and topiramate demonstrates a superior therapeutic effect over topiramate alone. This synergy is evidenced by enhanced antiseizure efficacy, a pronounced reduction in depressive-like behaviors, and a significant increase in hippocampal 5-HT1A receptor expression, suggesting a potential serotonergic mechanism for the observed benefits.

Keywords : Depression, Exercise, Topiramate

Count: 167
Abstract ID: 248
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Anticonvulsant Potential of Flurbiprofen via NF-κB Modulation in a Wistar Rat Seizure Model

Submission Author: Mohammad Soheil Aligol

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Background and Aim : A seizure is an acute, unregulated suspension of the brain's electrical activity, which is closely linked to neuroinflammation. Seizures and their pathology are strongly linked to NF-kB (nuclear factor kappa-light-chain-enhancer of activated B cells), as it plays a central role in mediating neuroinflammation as an immune response. The study investigates the impact of Flurbiprofen, a non-steroidal anti-inflammatory medication, on NF-kB gene expression in Induced acute seizure Wistar rat models induced with pentylenetetrazole (PTZ).

Methods : Acute seizures were induced through pentylenetetrazole (PTZ) in male Wistar rats at a dose of 60 mg/kg. Four groups of male Wistar rats were designated to analyze flurbiprofen against PTZ-induced seizure activity. Two groups of male Wistar rats were pre-treated with flurbiprofen at doses of 5 and 10 mg/kg, and 30 min later, injected with PTZ. Total RNA was extracted from the brain and analyzed for NF-kB gene expression via Real-Time PCR. One-way ANOVA and Tukey's post-test ($p < 0.05$) were utilized for statistical analysis.

Results : The PTZ group showed that the NF-kB gene expression exceeded when compared to the control group ($p<0.05$). The Flurbiprofen-treated group at the dose of 10 mg/kg indicates that the NF-kB gene expression is lower when compared to the PTZ group ($p<0.05$).

Conclusion : The results demonstrate that Flurbiprofen efficiently reduces neuroinflammation by significantly downregulating NF-kB mRNA expression.

Keywords : Flurbiprofen; Non-steroidal anti-inflammatory drug; NF-kB; Gene expression

Count: 168
Abstract ID: 673
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Oral

Machine Learning Models for Predicting Treatment Response in Status Epilepticus: Comparative Performance Against Traditional Statistical Methods

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Background and Aim : Status epilepticus (SE) is a life-threatening neurological emergency with mortality rates approaching 20%. Despite standardized treatment algorithms, therapeutic response remains heterogeneous, complicating early management. Existing prognostic models based on logistic regression or clinical scoring systems achieve moderate accuracy (AUC 0.75–0.80) but often fail to generalize across etiologies. This study aimed to develop and validate machine learning (ML) models to predict treatment response in SE and compare their performance with traditional regression-based methods, emphasizing clinical interpretability and potential bedside utility.

Methods : Data from 360 patients with SE were retrospectively collected over seven years (2016–2023). Treatment response was defined as electroclinical seizure cessation within 30 minutes of therapy initiation. Clinical variables consisted of demographics, SE etiology and seizure duration, therapeutic regimen, along with EEG features. Following preprocessing, patients were randomly assigned into training (80%) and validation (20%) cohorts. Stratified 10-fold cross-validation was used to compare ML algorithms including random forest and gradient boosting with logistic regression and Cox proportional hazards models. The model performance assessment was done by accuracy, sensitivity, specificity, and area under the receiver operating characteristic curve (AUC-ROC) with 95% confidence intervals. Calibration and decision curve analyses were performed to measure clinical utility, and feature importance was quantified using SHAP values.

Results : Among the 360 patients, 216 (60%) had acute symptomatic and 144 (40%) cryptogenic etiologies. 52% of cases experienced a response to treatment. The gradient boosting model exhibited the best performance (AUC = 0.92, 95% CI 0.88–0.96; accuracy =

0.87, 95% CI 0.83–0.90), when compared to logistic regression (AUC = 0.79, 95% CI 0.74–0.84; $p < 0.001$, DeLong test), The sensitivity and the specificity was as the following: 0.85 (95% CI 0.80–0.89) and 0.88 (95% CI 0.84–0.91); respectively. Calibration was acceptable (Hosmer–Lemeshow $p = 0.47$), and decision curve analysis showed that the net clinical benefit was better across all thresholds. SHAP analysis identified seizure duration more than 30 minutes, acute symptomatic etiology, and rhythmic delta activity on EEG considering the most significant predictors, which was followed by delayed benzodiazepine administration and elevated serum lactate levels. External validation (n = 72) corroborated robustness (AUC = 0.90; accuracy = 0.85).

Conclusion : ML models significantly improved treatment response prediction in SE when compared to traditional statistical methods. This led to a 13% increase in accuracy and showed stable external validity. The use of interpretable ML methods, such as SHAP and decision curve analysis, increases transparency, additionally to providing translation support for clinical decision-making.

Keywords : status epilepticus; machine learning; treatment response; prediction

Count: 169
Abstract ID: 66
subject: Epilepsy, Neural Excitability, Synapses, and Glia: Seizure and Epileptic Disorders
Presentation Type: Poster

Tenoxicam Increases Seizure Latency and Reduces Severity by Modulating Neuroinflammation in a Pentylenetetrazole-Induced Rat Model

Submission Author: Zahra Guilandokht

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Background and Aim : Neuroinflammation influences the onset and course of epilepsy. The pentylenetetrazole (PTZ)-induced rat model is an epileptic process that mimics these activities. A nonsteroidal anti-inflammatory medicine, tenoxicam (TNX) could influence neuroinflammation and increase seizure thresholds. The present work looks at how Tenoxicam (0.6 and 1.2 mg/kg) affects seizure onset latency and intensity in male Wistar rats.

Methods : Twenty-four male Wistar rats were allocated randomly to Control, PTZ, or TNX-treated groups (0.6 or 1.2 mg/kg) (220 ± 20 g; 8 weeks old). Housed in regular conditions with ad libitum food and water, TNX was given intraperitoneally 30 minutes prior to PTZ. Seizure behaviours were rated using the modified Racine scale on video over thirty minutes.

Results : Systemic treatment of TNX in dosages of 0.6 and 1.2 mg/kg produced a notable, dose-related reduction of epileptiform activity, hence increasing latencies for all phases of seizures as graded by Racine (LS1–LS5; $p < 0.05$ vs. PTZ). Indicating its strong anticonvulsant effect, TNX also greatly lowered the frequency of myoclonic jerks ($p < 0.01$ vs. PTZ) and the length of tonic-clonic seizures ($p < 0.05$ vs. PTZ).

Conclusion : In PTZ-challenged rats, TNX pretreatment clearly delayed seizure onset and reduced seizure severity, so suppressing myoclonic jerks and lowering tonic–clonic seizure length. The findings support more study on Tenoxicam's long-term safety and effectiveness in epilepsy models since they show its possible use as an adjuvant anticonvulsant medication.

Keywords : Nonsteroidal anti-inflammatory drug; Tenoxicam; Seizures; Neuroinflammation

Count: 170
Abstract ID: 114
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Poster

Adaptive and optimized seizure prediction system: an EEG study

Submission Author: Elham Shamsi

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Background and Aim : Epilepsy is a neurobiological disorder that impacts more than 2% of the people worldwide. Seizure prediction is crucial to physicians since it can assist seizure control, prevent from sudden death caused by some seizures, and enhance the patients' quality of life. Seizures are resulted from transient and synchronous electrical activity of neurons. Therefore, seizure prediction is feasible via electroencephalogram (EEG) monitoring, which is non-invasive, portable, economical, and time-saving. Given the non-stationarity of the EEG signal, time-frequency transforms (TFTs) can more precisely measure its dynamics. Being transient, seizures have been well predicted by adaptive TFTs (ATFTs). Thus, ATFT-based seizure prediction is vital. This study aims at developing an optimized and adaptive ATFT-based framework for seizure prediction using EEG signals.

Methods : Considering different types of seizure components and due to the fact that the time-reassigned adaptive Fourier synchrosqueezing transform with globally optimal window length (TAFSSTOL) has been effective for multicomponent non-stationary signals encompassing slowly- and fast-changing components together, it is the ATFT used in this study. Moreover, all steps of the machine learning framework are globally optimized, making the proposed adaptive prediction more effective. Thus, the novelty of this work is three-fold (i.e., utilizing TAFSSTOL for seizure prediction, extraction of innovative time-frequency features, and optimized machine learning algorithms). The proposed approach is validated on a three-class adult epilepsy EEG dataset recorded in Haus Khas sleep and neurobiology center. The classes include preictal, interictal, and ictal phases.

Results : The proposed approach outperformed the existing relevant literature (accuracy: 92%, sensitivity: 82%, specificity: 97%, precision: 93.2%, negative predictive value: 91.5%, F1-score: 0.872, Matthew's correlation coefficient: 0.818, Cohen's kappa value: 0.880, and area under the receiver operating characteristic curve: 0.888).

Conclusion : In this study, seizures were predicted by means of TAFSSTOL-based optimized machine learning approach used for EEG analysis. Being adaptive and preserving the signal characteristics, TAFSSTOL could result in the most discriminant features as seizure components are of different types of instantaneous frequencies and group delays. Besides the optimized window length in the TAFSSTOL, feature selection and classifier models were globally optimized, leading to an optimal adaptive seizure prediction system. Hence, the proposed ATFT-based optimized machine leaning framework can spectacularly pave the way for seizure prediction.

Keywords : Time-reassigned adaptive Fourier synchrosqueezing transform; electroencephalogram; epilepsy; seizure; Bayesian optimization; machine learning

Count: 171
Abstract ID: 518
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Poster

Cognitive Resilience at Hypersonic Speed: Neuroadaptive Aviation as a Frontier for Human–AI Symbiosis

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Conclusion : Modern fighter pilots operate in an environment where decisions unfold faster than the biological brain was ever designed to process. Hypersonic flight, adversarial uncertainty, and the physiological strains of high-G maneuvering compress cognition into milliseconds, exposing the fragile boundary between human adaptability and collapse. Yet within this crucible lies an opportunity: aviation neuroscience is no longer merely about describing limits, but about designing systems that expand them. We review emerging evidence that performance in extreme aviation is best understood not as isolated brain function, but as a closed-loop system of human, machine, and environment. Wearable neurophysiology (EEG, pupillometry, fNIRS), biometric sensing, and high-fidelity simulations now allow real-time decoding of pilot states—before errors manifest. Neuroadaptive cockpits use these signals to dynamically redistribute workload, alter interface complexity, or delegate control to automated agents. AI copilots are beginning to act not as tools but as symbiotic partners, trained to anticipate cognitive bottlenecks, detect fatigue, and even modulate mission tempo in synchrony with human brain states. The next frontier is predictive resilience. By integrating multimodal data streams into digital neurotwins—individualized computational models of a pilot's cognitive state—we can forecast breakdowns under stress, personalize training, and simulate performance across mission scenarios never yet flown. Unlike retrospective analyses, digital twins adapt continuously, learning from both brain data and behavioral history to offer forward-looking interventions: from tailored stress inoculation protocols to non-invasive neuromodulation for maintaining focus at the edge of collapse. This neuroadaptive framework has implications far beyond aviation. Space exploration, remote robotics, high-frequency trading, and medical crisis response all demand cognition under compressed timelines. By shifting focus from static "limits of the brain" to dynamic human–AI teaming, aviation neuroscience reframes decision-making as an expandable frontier. The key question is no longer "How do humans cope under stress?" but "How can human cognition and intelligent systems co-evolve to outperform either alone?" We argue that fighter pilot neuroscience is not an applied niche, but a discovery platform for next-generation human performance science. It forces us to rethink resilience, agency, and identity in a future where cognition is distributed across biology, algorithms, and engineered environments. At Mach speed, the brain does not merely survive—it evolves in symbiosis with the machines it flies.

Keywords : Neuroadaptive Aviation; Cognitive Resilience; Human–AI Symbiosis; Digital Neurotwins; Predictive Performance Modeling; Real-Time Neurophysiology

Count: 172
Abstract ID: 500
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Oral

Practical Neuroimaging of glial-derived brain tumors: Insights into MRI protocols

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Background and Aim : Glial-derived tumors, particularly astrocytomas, are the most common primary brain neoplasms and pose significant diagnostic challenges, especially in differentiating low- from high-grade lesions. Conventional MRI, while indispensable for anatomical characterization, often lacks sufficient accuracy for precise grading. Advanced imaging modalities, including magnetic resonance spectroscopy (MRS) and diffusion-weighted imaging (DWI), provide complementary insights into tumor metabolism and microstructure. This study aimed to evaluate and compare the diagnostic performance of MRS and DWI in patients with astrocytomas.

Methods : Thirty patients with histologically confirmed astrocytomas (15 low-grade and 15 high-grade) underwent MRI using standardized protocols. MRS was performed to quantify metabolite concentrations, including N-acetylaspartate (NAA), choline (Cho), creatine (Cr), glutamate, taurine, and glycine, with calculation of metabolite ratios (NAA/Cr, Cho/Cr, Glycine/Taurine). DWI was used to measure apparent diffusion coefficient (ADC) values in tumor cores and peritumoral regions. Diagnostic sensitivity, specificity, and accuracy of both modalities were compared. Statistical analysis was performed using t-tests and ROC analysis, with $p < 0.05$ considered significant.

Results : MRS demonstrated characteristic metabolic alterations in high-grade astrocytomas. NAA/Cr ratio was significantly reduced (0.72 ± 0.18) compared with low-grade lesions (1.25 ± 0.22; $p < 0.001$). Cho/Cr ratio was elevated in high-grade tumors (2.15 ± 0.41) in comparison to low-grade (1.35 ± 0.29; $p < 0.001$). Glutamate and taurine levels were significantly higher in high-grade astrocytomas (2.35 ± 0.37 and 1.45 ± 0.26, respectively) than in low-grade tumors (1.62 ± 0.31 and 0.92 ± 0.21; $p = 0.002$ and $p = 0.004$). The glycine/taurine ratio was also increased in high-grade tumors (1.82 ± 0.33 vs. 1.21 ± 0.25; $p = 0.003$). DWI revealed significantly lower ADC values in high-grade astrocytomas (0.89 ± 0.12 × 10^{-3} mm^2/s) compared to low-grade lesions (1.32 ± 0.15 × 10^{-3} mm^2/s; $p < 0.001$). Diagnostic performance analysis showed that MRS achieved a sensitivity of 90.0%, a specificity of 86.7%, and an accuracy of 88.3%, whereas DWI yielded a sensitivity of 83.3%, a specificity of 80.0%, and an accuracy of 81.7%. Comparative analysis indicated that MRS outperformed DWI in overall accuracy ($p = 0.021$). The combined use of MRS and DWI further improved diagnostic accuracy to 93.3% ($p < 0.001$).

Conclusion : This study highlights the complementary roles of MRS and DWI in assessing astrocytomas. MRS provides comprehensive metabolic biomarkers, including Cho/Cr, NAA/Cr, and excitatory metabolites such as glutamate and taurine, which are closely linked to tumor aggressiveness. In parallel, DWI offers critical microstructural insights, with reduced ADC values serving as reliable indicators of high-grade lesions. Although MRS demonstrated superior sensitivity and accuracy compared with DWI, the combined application of both modalities yielded the highest diagnostic performance. These findings highlight the importance of incorporating advanced MRI techniques into routine neuroimaging protocols to enhance grading precision, optimize treatment planning, and improve prognostic evaluation in patients with glial-derived brain tumors.

Keywords : Neuroimaging; Brain Tumors; Magnetic Resonance Spectroscopy; Structural Imaging.

Count: 173
Abstract ID: 369
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Poster

Automated Trajectory Planning for Deep Brain Stimulation Using Particle Swarm Optimization

Submission Author: Alireza Fallahi

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Background and Aim : Deep brain stimulation (DBS) is an established therapeutic intervention for movement and psychiatric disorders, offering reversible modulation of specific brain targets. Accurate electrode placement is critical for therapeutic efficacy and minimizing complications. Traditional manual trajectory planning is time-consuming and prone to human error, particularly in regions with dense vascular networks. Automating the path planning process using optimization algorithms may improve safety, accuracy, and procedural efficiency. Particle Swarm Optimization (PSO), a population-based stochastic optimization technique, is well-suited for solving complex trajectory-planning problems with multiple constraints. This study aimed to develop an automated DBS path planning approach based on PSO, integrating patient-specific vascular anatomy and the subthalamic nucleus (STN) as the stimulation target.

Methods : High-resolution T1-weighted contrast-enhanced (T1-CE) MRI scans were acquired and processed to segment cerebral vessels and delineate the STN region of interest. The vascular segmentation provided a 3D anatomical map of critical structures to be avoided during electrode insertion. The STN was manually or semi-automatically defined as the surgical target. The segmented vascular map was registered to the MRI coordinate space, ensuring spatial accuracy between the vascular structures and the STN. An initial predefined circular region around the target was established as the permissible entry zone for electrode insertion. Particle Swarm Optimization (PSO) was applied to determine the optimal path from the cortical entry point to the STN while minimizing the risk of vessel collision and optimizing trajectory length and angular constraints. The cost function incorporated vessel proximity penalties, path length, and mechanical feasibility criteria. The optimization iteratively adjusted candidate paths, with swarm agents exploring the multidimensional parameter space to identify a safe and efficient trajectory.

Results : The automated PSO-based path planning algorithm successfully identified collision-free trajectories to the STN across all tested cases. The optimized paths minimized proximity to segmented brain vessels while preserving clinically acceptable insertion angles and trajectory lengths. Compared to initial manually selected trajectories, PSO-derived paths demonstrated improved clearance from critical vascular structures, reducing theoretical hemorrhage risk. The circular predefined entry region allowed for exploration of multiple feasible cortical entry points, improving flexibility in trajectory selection. Processing and optimization times were clinically practical, supporting the potential for integration into surgical planning workflows.

Conclusion : This study presents a patient-specific, automated DBS path planning framework using Particle Swarm Optimization and vascular segmentation from T1-CE MRI. By incorporating detailed anatomical constraints and target definition, the method provides safe and efficient trajectory recommendations for STN DBS electrode placement. The approach reduces manual planning workload and enhances surgical safety by minimizing vascular injury risk. Future work will involve validation in larger patient cohorts, integration with intraoperative navigation systems, and refinement of cost functions to incorporate additional surgical constraints such as skull entry angles and white matter tract preservation. This PSO-based approach represents a promising step toward fully automated, intelligent DBS surgical planning.

Keywords : DBS, PSO, Path planning, Artificial intelligence

Count: 174
Abstract ID: 923
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Poster

Neural Correlates of Auditory Processing of Speech in Children with Speech Sound Disorder using Event-Related Potentials: A Review

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Conclusion : Children with speech sound disorders (SSD) may have deficits in central auditory processing of speech compared to their peers. We reviewed empirical studies examining auditory processing of speech in children with SSD, focusing on five components: P300, N400, mismatch negativity (MMN), positive mismatch response (P-MMR), and late discriminative negativity (LDN). Databases, Evidence Based medicine, and electronic journals were searched using combined keywords including: P300, N400, P-MMR, MMN, LDN, and SSD. Studies report reduced MMN amplitudes, reflecting impaired auditory discrimination, although this is not consistent across all studies. P300 findings also show mixed results, suggesting intentional or cognitive processing deficits in children with SSD. While N400 anomalies indicate semantic-phonological integration deficits in SSD, P-MMR remain as an underreported measurement of brain immaturity. Notably, LDN and the P-MMR-MMN-LDN chain are unexplored in this population. Although the amplitude and latency of these neural responses differ from those of typically developing children and can be modulated by intervention, the occurrence of P-MMR and LDN in children with SSD should be interpreted with caution

Keywords : Mismatch response, Speech sound disorder, P-MMR, LDN, Speech processing, Evoked Potential.

Count: 175
Abstract ID: 807
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Poster

Feasibility Study of Using Electroencephalography to Evaluate the Effect of Auditory Stimuli on Children's Cognitive Processes: A Case Report

Submission Author: Hoda Alijanian

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Background and Aim : Multisensory learning and the role of auditory stimuli in improving children's cognitive processes have received increasing attention from educational neuroscience researchers in recent years. However, objective and non-invasive assessment tools for examining the immediate effects of these stimuli on children's brain activity remain limited. The aim of this case study is to investigate the feasibility of using electroencephalography (EEG) as a tool to evaluate brain activity changes in response to sound effects accompanying educational content.

Methods : In this case study, an 8.5-year-old third-grade female student with normal cognitive and auditory abilities was examined. The subject's brain electrical activity was recorded during a 10-minute task with two experimental conditions: 1) listening to Persian language lesson instruction and reading by teacher's voice (baseline condition), 2) listening to the continuation of the same lesson with sound effects aligned with the content (experimental condition). EEG recording was performed using a 19-channel Mitsar device across different brain regions. The recorded signals were decomposed into different frequency bands and qualitatively analyzed after preprocessing and artifact removal.

Results : EEG signal analysis demonstrated that the designed protocol was capable of recording and detecting differences in brain activity between the two experimental conditions. In the condition with sound effects, changes in the electrical activity patterns of different brain regions were observed, indicating the sensitivity of this method to auditory stimulus variations. These preliminary findings confirm the feasibility of using this approach for larger-scale studies.

Conclusion : This case study demonstrated that using EEG to evaluate the effects of sound effects on children's cognitive processes is technically feasible and practical. The protocol developed in this study possesses the necessary sensitivity to detect brain activity changes and can be utilized in future studies with larger sample sizes. This approach has potential applications in designing neuroscience evidence-based educational interventions as well as in pediatric auditory rehabilitation programs. Conducting studies with larger samples is necessary to achieve generalizable results and develop standardized protocols.

Keywords : Electroencephalography; Sound Effects; Multisensory Learning; Auditory Processing; Children; Educational Neuroscience

Count: 176
Abstract ID: 926
subject: Novel and Cutting-Edge Technologies: Brain Mapping (MRI, fMRI, PET, Brain Mapping, EEG, EMG, QEEG, FNIRS)
Presentation Type: Oral

The Effects of Mind Simulation Training on Ball Control in Soccer Players: An fMRI Investigation

Submission Author: Mojtaba Bigdelishamloo

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Background and Aim : Mind simulation (MS) is recognized as a potent cognitive technique for enhancing motor performance. This study investigated the neuroplasticity effects of MS training on soccer-specific ball control skills, utilizing functional magnetic resonance imaging (fMRI) to map associated changes in neural activation.

Methods : A single-subject, pre-post intervention design was employed with a 35-year-old female novice with no prior soccer experience, selected via convenience sampling. Resting-state fMRI scans (10 minutes) were acquired before and after a targeted MS intervention. The intervention consisted of mental practice focused on soccer ball control skills, conducted after one introductory session and sustained for one week, with daily one-minute simulations. Image data were processed and analyzed using the FMRIB Software Library (FSL). Changes in neural activation patterns were assessed using subtraction analysis to isolate intervention-related effects.

Results : Post-intervention fMRI revealed increased activity within bilateral motor circuitry, primarily in regions critical for motor planning and execution, such as the premotor cortex and supplementary motor area. This targeted enhancement indicates a strengthening of the neural representations for foot-ball control, despite the absence of physical practice.

Conclusion : The findings demonstrate that brief, focused mental simulation training can effectively upregulate neural activity within motor networks governing complex, sport-specific tasks. This provides preliminary neuroscientific evidence for the use of cognitive-motor interventions as non-physical adjuncts to enhance athletic performance in soccer, highlighting the robust capacity of mental practice to induce functional neuroplasticity.

Keywords : Mind Simulation; Soccer; fMRI.

Count: 177
Abstract ID: 602
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

Deep Brain Stimulation in Progressive Multiple Sclerosis: Unlocking Neurogenesis and Remyelination Through Transcriptomic Reprogramming as a Next-Generation Therapeutic Strategy

Submission Author: Narjes Khatoun Shabani Sadr

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Background and Aim : Progressive multiple sclerosis (PPMS and SPMS) remains a major therapeutic challenge, as current immunomodulatory therapies show limited efficacy in halting neurodegeneration or promoting neural repair. Deep brain stimulation (DBS), an established therapy for Parkinson's disease, has recently emerged as a potential approach to modulate neural plasticity and repair processes. Yet, the molecular mechanisms underlying its possible efficacy in MS remain poorly defined.
Methods : We conducted integrative bioinformatics analyses of RNA-seq datasets derived from progressive MS brain tissues and DBS-modulated neural models. Differential expression analysis was performed using DESeq2, followed by gene set enrichment (GSEA) and network analysis with Cytoscape. Candidate molecular hubs were prioritized with a focus on pathways governing neurogenesis, synaptic plasticity, myelination, and inflammatory regulation.
Results : Transcriptomic analysis of progressive MS brain tissues, particularly demyelinating plaques, revealed profound alterations in interconnected gene networks related to neurogenesis, myelin repair, and inflammation. Parallel analyses of DBS-modulated animal models demonstrated that DBS can influence these same pathways, with upregulation of neurogenesis-associated genes (BDNF, DCX), enhancement of myelination markers (MBP, MOG), and suppression of inflammatory mediators (TNFα, IL6). Pathway enrichment highlighted activation of the CREB1 signaling axis, while network modeling identified BDNF and CREB1 as central hubs bridging neurogenesis, synaptic plasticity, and remyelination.
Conclusion : These findings suggest that transcriptomic reprogramming in progressive MS involves dysregulation of repair- and inflammation-related gene networks. The observation that DBS modulates these pathways in animal models provides proof-of-concept for its potential relevance to MS. Moving forward, systematic studies in MS-specific animal models are warranted to validate hallmark genes and regulatory hubs, paving the way for DBS as a next-generation therapeutic strategy beyond conventional immunomodulation, particularly for patients with PPMS and SPMS who currently lack effective treatments.
Keywords : Progressive multiple sclerosis, Deep brain stimulation, Transcriptomics, Neurogenesis, Remyelination, Bioinformatics

Count: 178
Abstract ID: 402
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

Effects of melatonin and transcranial photobiomodulation on fear learning and memory in rats under chronic stress

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Background and Aim : Chronic stress can affect learning and memory processes that are emotion-based, such as differential fear conditioning. Some evidences suggest that melatonin (MLT) has beneficial effects on brain function. Additionally, transcranial photobiomodulation (tPBM) is an emerging non-invasive intervention showing promising results in the context of mental disorders. This study aimed to evaluate the comparative and potential synergistic effects of MLT and tPBM in rats exposed to chronic stress.

Methods : Forty-nine male Wistar rats were randomly assigned to seven groups: Control (Co), Sham injection (Sh-I), Sham tPBM (Sh-tPBM), Stress (St), Stress with Melatonin (St-MLT), Stress with tPBM (St-tPBM), and Stress with Melatonin and tPBM (St-MLT-tPBM). Chronic restraint stress (2 h/day for 21 days) was applied to all stressed rats. MLT (20mg/kg; intraperitoneally) and tPBM (808 nm, 0.92 W/cm2, 96.1 J/day delivered to the scalp surface) were administered following restraint stress. Initial latency (IL) and step-through latency (STL) were assessed using the passive avoidance test. Comparisons of IL and STL within groups indicate fear learning, while STL alone reflects fear memory.

Results : Comparison of IL-STL revealed significant enhancement across all experimental groups at the different levels. The St group exhibited the highest levels of fear learning, while the St-MLT-tPBM group showed the lowest. Additionally, STL was significantly increased in the St group compared to the Co group, indicating to induced of fear memory in this group. However, it was remarkably decreased in the St-MLT, St-tPBM, and particularly St-MLT-tPBM groups compared to the St group.

Conclusion : Chronic stress significantly enhances fear learning and memory. A major increase in the avoidance of trauma reminders contributes to the development of stress-related

psychopathologies, such as post-traumatic stress disorder (PTSD). Conversely, melatonin (MLT), transcranial photobiomodulation (tPBM), and particularly their combined administration prevented the enhancement of fear memory in stressed subjects.

Keywords : Chronic stress, Fear learning, Fear memory, Melatonin, Transcranial photobiomodulation

Count: 179
Abstract ID: 775
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

Effect of combining VitalStim®, Traditional therapy and Sensory Stimulation, and Trans-Cranial Direct Current Stimulation on dysphagia recovery in a patient with brainstem and cerebrum stroke

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Background and Aim : A 68-year-old woman with a history of hypertension presented with an ischemic stroke involving the left cortical and brainstem regions. One week after the event, she was referred for swallowing and rehabilitation therapy due to severe dysphagia and respiratory difficulty. Her level of consciousness was 12 on the Glasgow Coma Scale. The patient was dependent on a nasogastric tube for nutrition and a tracheostomy tube with oxygen supplementation at 4–5 L/min. Clinical examination revealed weak swallowing, gag, and cough reflexes, drooling, and severe right-sided limb weakness.

Methods : A multidisciplinary rehabilitation program including occupational, physical, and speech therapy was initiated. Swallowing therapy was provided by a trained speech therapist five days per week, one hour per session. The therapeutic program incorporated thermo-tactile and chemical (taste and smell) stimulation, neuromuscular electrical stimulation (NMES) using the VitalStim® Plus device for at least 30 minutes per session, and traditional compensatory techniques such as head turning as the patient's consciousness improved. In addition, transcranial direct current stimulation (tDCS) was applied every other day for 20 minutes at an intensity of 1.5–2 mA, with the anode positioned over the primary motor cortex and the cathode placed over the supraorbital area.

Results : After 25 sessions of speech therapy, the patient was able to safely swallow foods of level-4 (pureed) consistency. The nasogastric tube was removed after 35 sessions, and the tracheostomy tube was successfully decannulated by session 46.

Conclusion : This case demonstrates the potential efficacy of combining VitalStim®, sensory and traditional swallowing therapy, and tDCS for accelerating recovery from severe post-stroke dysphagia in patients with brainstem and cerebrum involvement.

Keywords : TDCs; Vital-stim; dysphagia; speech therapy

Count: 180
Abstract ID: 677
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

The Effect of Transcranial Direct Current Stimulation(tDCS) on Emotion Regulation and Problem Solving in People With Social Anxiety

Submission Author: Shabnam Hashemzadehmohammadieh

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Background and Aim : Social anxiety disorder is often associated with impaired emotion regulation and deficient problem-solving skills, which exacerbate functional and interpersonal difficulties. Recent studies suggest that non-invasive brain stimulation techniques such as transcranial direct-current stimulation (tDCS) may enhance the neural mechanisms underlying emotion regulation and executive control. This study aimed to evaluate the efficacy of tDCS in enhancing emotion regulation and problem-solving abilities in individuals with social anxiety disorder.

Methods : This randomized controlled trial recruited 32 university students with clinically significant social anxiety. Participants were randomly assigned to an experimental group (n = 17) receiving active tDCS or a control group (n = 15) receiving sham stimulation. The intervention consisted of 10 sessions of tDCS (3 sessions per week over 3 consecutive weeks; 20 minutes per session) with the anode over the left dorsolateral prefrontal cortex and the cathode over the right dorsolateral prefrontal cortex. Outcome measures included the Emotion Regulation and the Problem-Solving Inventory administered at baseline and post-treatment. Data were analyzed using univariate and multivariate ANCOVA.

Results : Compared to the control group, the experimental group of university students with social anxiety disorder (mean age 24.8 years; 58.8% female) showed significant post-intervention improvements in both emotion regulation ($p < 0.01$) and problem-solving skills ($p < 0.05$), compared to the control group (mean age 24.5 years; 47% female). These improvements were reflected in a greater use of adaptive strategies and a reduction in maladaptive strategies for managing emotions, together with a shift toward more constructive, planned, and goal-oriented approaches to problem-solving.

Conclusion : tDCS applied to the dorsolateral prefrontal cortex significantly enhanced emotion regulation and problem-solving abilities in young adults with social anxiety disorder. By addressing two key maintaining factors of the disorder, this non-invasive intervention shows promise as an adjunct to conventional psychological treatments, potentially enhancing coping skills and functional outcomes. Further research with larger samples and long-term follow-up is needed to confirm the durability and generalizability of these effects.

Keywords : Social Anxiety, Emotion Regulation, Problem Solving, Non-Invasive Brain Stimulation

Count: 181
Abstract ID: 258
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

Suppressing Morphine Reinstatement with Infralimbic Deep Brain Stimulation: A Critical Role for Application Timing and Frequency

Submission Author: Masoud Seddighfar

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Background and Aim : Opioid use disorder represents a significant public health challenge, creating a need for novel therapeutic approaches. Deep brain stimulation (DBS) has emerged as a promising intervention for treatment-resistant conditions. The infralimbic (IL) cortex, a brain region implicated in addiction and impulse control, is a key target for such therapies. This study was designed to explore how DBS of the IL, applied at different frequencies and timings, could influence the extinction and reinstatement of morphine-seeking behavior in a preclinical model using male rats.

Methods : Male rats were first trained to develop a conditioned place preference (CPP) for morphine, administered at a dose of 5 mg/kg. Following this, the animals were exposed to deep brain stimulation targeting the infralimbic cortex, delivered at either high-frequency (HFS; 130 Hz) or low-frequency (LFS; 10 Hz). The stimulation was applied under two distinct experimental protocols. The first protocol involved daily 30-minute DBS sessions throughout the extinction phase. The second protocol consisted of a single DBS session administered immediately before a reinstatement test, which was initiated by a 1 mg/kg priming dose of morphine.

Results : The application of daily DBS during the extinction period yielded significant effects. Both high- and low-frequency stimulation accelerated the rate of extinction and successfully prevented the reinstatement of morphine-seeking behavior following the drug prime. When a single session of DBS was applied just before the reinstatement test, the results differed by frequency. High-frequency stimulation effectively blocked the return of drug-seeking, but low-frequency stimulation failed to prevent reinstatement. A direct comparison of the protocols revealed that daily HFS during the extinction phase was more effective in preventing relapse than the single application of HFS.

Conclusion : The findings from this study demonstrate that deep brain stimulation of the infralimbic cortex can powerfully modulate addiction-related behaviors, with effects varying across different stimulation frequencies. A critical insight is that the therapeutic outcome is highly dependent on the timing of the intervention. Sustained application of DBS throughout the extinction period appears to produce more robust and lasting effects against relapse compared to a single, acute application. This suggests that the specific protocol for DBS administration is a crucial factor for optimizing its potential clinical success in treating treatment-refractory addiction.

Keywords : Reward; Deep Brain Stimulation; Infralimbic cortex; Extinction; Reinstatement; Morphine; Conditioned place preference; Rat

Count: 182
Abstract ID: 264
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

Infralimbic DBS during Abstinence Blocks Drug- and Stress-Induced Morphine Relapse

Submission Author: Farzaneh Samadi

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Background and Aim : Relapse is the primary obstacle in the long-term treatment of Opioid Use Disorder (OUD). While Deep Brain Stimulation (DBS) shows therapeutic promise, its optimal application timing remains unclear. The infralimbic cortex (IL) is increasingly recognized as a critical hub for modulating addiction-related behaviors. This study was designed to determine if IL DBS administered during a period of passive abstinence—independent of any extinction training—could prevent the reinstatement of morphine preference. The core objective was to test whether this intervention could act directly on the latent addiction memory, or "engram," to block relapse triggered by distinct cues.

Methods : Male Wistar rats were first conditioned to associate a specific environment with morphine (5 mg/kg, s.c.), establishing a conditioned place preference (CPP). Following this, the animals entered an 8-day passive abstinence period where no drug was available. During this time, they received daily sessions of either high-frequency (HFS; 130 Hz) or low-frequency (LFS; 10 Hz) DBS delivered to the IL. After the abstinence period, the reinstatement of morphine-seeking was initiated by one of two triggers: a priming dose of morphine (1 mg/kg, s.c.) or exposure to acute restraint stress.

Results : During the abstinence period, the application of both HFS and LFS significantly accelerated the natural decay of the learned morphine preference. In the subsequent reinstatement tests, HFS demonstrated a robust and broad effect, successfully blocking the reinstatement of morphine-seeking triggered by both the drug prime and by stress. In contrast, LFS showed a trigger-specific effect: it completely blocked reinstatement induced by the morphine prime but only partially weakened the reinstatement caused by stress. This revealed a significant interaction between the frequency of the stimulation and the nature of the relapse trigger.

Conclusion : The findings show that applying IL DBS during abstinence is a powerful and effective method for preventing opioid reinstatement. This suggests a therapeutic mechanism

that involves directly weakening the underlying addiction memory, rather than solely facilitating new extinction learning. High-frequency stimulation of the IL, in particular, emerges as an exceptionally robust, broad-spectrum intervention capable of neutralizing multiple types of relapse triggers. This prophylactic approach offers a promising and novel strategy for the clinical treatment of OUD.

Keywords : Reward; Deep Brain Stimulation; Infralimbic cortex; Addiction-memory; Reinstatement; Morphine; Conditioned place preference; Rat

Count: 183
Abstract ID: 611
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

Closed-Loop Deep Brain Stimulation for Parkinson's Disease: A Systematic Review of Control Strategies and Neuromodulation Outcomes

Submission Author: Hossein Hassanzadeh Yaghini

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Conclusion : Introduction: Parkinson's disease (PD) is a progressive neurological disorder characterised by motor symptoms such as tremors and bradykinesia. Traditional deep-brain stimulation (DBS) delivers continuous electrical pulses to specific targets in the brain. While this approach effectively suppresses symptoms, it often comes with side effects and lacks adaptability. This systematic review aims to synthesize evidence on adaptive or closed-loop DBS (aDBS) systems, which adjust stimulation in real-time based on feedback from biomarkers. The goal of aDBS is to enhance therapeutic efficacy while reducing side effects. Materials and Methods: This review was conducted in accordance with the PRISMA guidelines. A thorough search of databases, including IEEE Xplore, PubMed, and Google Scholar, was carried out for studies published between 2015 and 2025. The search terms used included "adaptive deep brain stimulation," "closed-loop DBS," "Parkinson's disease," and "neural control." The inclusion criteria focused on clinical studies and computational models that provided detailed information on specific control strategies for adaptive deep-brain stimulation (aDBS). Data was extracted on the control algorithms, biomarkers used for feedback (such as local field potentials and beta-band power), and clinical outcomes. Results: The reviewed literature indicates a significant shift from static to dynamic neuromodulation in the treatment of Parkinson's disease (PD). Key findings show that adaptive deep brain stimulation (aDBS) systems, which use local field potentials (LFPs) from the subthalamic nucleus as feedback signals, can effectively suppress pathological beta-band oscillations—these oscillations are key biomarkers associated with PD symptoms. Control strategies for aDBS range from simple on-off triggers to more advanced continuous modulation schemes that adjust based on the amplitude of the neural signals. Compared to conventional deep brain stimulation (DBS), aDBS systems provide comparable or even superior symptom control while significantly reducing the total energy required for delivery. This reduction may help alleviate side effects such as speech impairment and extend the battery life of implantable devices.

Conclusion: Adaptive DBS represents a significant advancement in neuromodulation therapy for Parkinson's disease. By leveraging real-time neural feedback, aDBS systems offer a more personalized and efficient treatment approach. Future research should focus on the development of more intelligent control algorithms, the validation of multiple biomarker inputs, and the execution of large-scale, long-term clinical trials to establish the efficacy and safety of these systems in diverse patient populations.

Keywords : Parkinson's disease, Closed-Loop DBS, Model Predictive Control (MPC), Adaptive Deep Brain Stimulation (aDBS), Biomarker Feedback

Count: 184
Abstract ID: 808
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

EEG and QEEG Biomarkers of rTMS Treatment Response in Adolescent Depression: A Systematic Review

Submission Author: Fatemeh TaleshiDoulatRoudbar

Fatemeh TaleshiDoulatRoudbar[1]

1. Fatemeh Taleshi

Conclusion : EEG and QEEG Biomarkers of rTMS Treatment Response in Adolescent Depression: A Systematic Review Background: Adolescent depression remains a global mental health challenge, with limited efficacy of pharmacological treatments. Repetitive transcranial magnetic stimulation (rTMS) has emerged as a promising non-invasive intervention, yet treatment response varies widely across individuals. Identifying reliable neurophysiological biomarkers, particularly electroencephalography (EEG) indicators, could enhance personalized treatment approaches. Objective: This systematic review aims to synthesize current evidence on EEG and quantitative EEG (QEEG) biomarkers associated with treatment response to rTMS in adolescents with major depressive disorder. Methods: Following PRISMA guidelines, a comprehensive search was conducted across PubMed, Scopus, and Web of Science databases for studies published between 2018 and 2025. Inclusion criteria encompassed clinical or experimental studies assessing EEG correlates of rTMS outcomes in adolescents (aged 12–24 years) with depressive symptoms. Data were extracted on study design, stimulation parameters, EEG measures (e.g., power spectra, connectivity, coherence), and clinical response indicators. Risk of bias was assessed using the Joanna Briggs Institute (JBI) checklist. Results: Preliminary synthesis suggests consistent associations between pre-treatment frontal alpha asymmetry, theta power in anterior cingulate regions, and rTMS response. Post-treatment increases in fronto-parietal connectivity and normalization of beta activity have been reported as potential neuroplasticity markers. However, methodological heterogeneity and small sample sizes limit cross-study comparability. Conclusion: EEG and QEEG measures show significant promise as predictive and mechanistic biomarkers for rTMS efficacy in adolescent depression. Future research should standardize EEG protocols, include longitudinal follow-ups, and integrate multimodal neuroimaging data to optimize individualized treatment strategies. Keywords: rTMS, EEG, adolescent depression, biomarkers, systematic review, neuroplasticity.

Keywords : Keywords: rTMS, EEG, adolescent depression, biomarkers, systematic review, neuroplasticity.

Count: 185
Abstract ID: 89
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Poster

The Effectiveness of Transcranial Direct Current Stimulation (tDCS) on Cynophobia: A Controlled Clinical Study at Setareh Iranian Clinic

Submission Author: Mohsen Dadkhah

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Background and Aim : This study investigated the effectiveness of transcranial direct current stimulation (tDCS) in reducing anxiety associated with cynophobia. Sixty female participants were recruited: 30 diagnosed with cynophobia (experimental group) and 30 non-phobic controls.

Methods : The experimental group received ten sessions of tDCS (anode on FP1, cathode on F4; 1 mA; 20 min) every other day over three weeks.

Results : Severity of phobia was measured by the Severity Measure for Specific Phobia—Adult (SPSA; APA, 2013) and general anxiety by the Beck Anxiety Inventory (BAI; Beck & Steer, 1993) pre- and post-intervention. Paired t-tests showed significant reductions in both SPSA ($t(29)=3.82$, $p=0.0006$) and BAI ($t(29)=5.44$, $p<0.00001$) in the experimental group, but not in controls. Between-group comparisons of change scores confirmed greater improvement in the experimental group for SPSA ($t(58)=3.64$, $p=0.0006$; $d=0.94$) and BAI ($t(58)=5.39$, $p<0.000001$; $d=1.39$).

Conclusion : These findings support tDCS as a promising non-invasive adjunct for treating specific phobias via anxiety reduction.

Keywords : tDCS; cynophobia; anxiety; specific phobia; Psychology

Count: 186
Abstract ID: 145
subject: Novel and Cutting-Edge Technologies: Brain Stimulation Methods (ECT, rTMS, TDCS, DBS)
Presentation Type: Oral

Intelligent Closed-Loop Cranial Electrotherapy Stimulation: A Randomized Trial of EEG- and Machine Learning–Guided Treatment for Depression

Submission Author: Asma KhosraviNajafAbadi

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8. -

Background and Aim : Major depressive disorder (MDD) remains a leading cause of disability worldwide, with many patients showing limited response to standard pharmacological and psychotherapeutic treatments. Cranial electrotherapy stimulation (CES) has emerged as a safe, non-invasive intervention, yet conventional open-loop approaches use fixed stimulation parameters and do not account for individual neurophysiological differences. This may explain inconsistent outcomes in previous trials. To address this gap, we developed an intelligent closed-loop CES (IC-CES) system that continuously analyzes quantitative EEG (QEEG) signals and applies machine-learning algorithms to adapt stimulation in real time. This study aimed to evaluate the feasibility, clinical outcomes, and neurophysiological effects of IC-CES compared with conventional CES in patients with MDD.

Methods : In a double-blind, randomized, parallel-group trial (IRCT20230112057118N1), 110 adults aged 18–65 years with DSM-5 MDD and BDI-II scores >14 were recruited at two psychiatric centers in Tehran, Iran. Participants were randomized 1:1 to receive either IC-CES or conventional CES, with five treatment sessions delivered over two weeks. Clinical outcomes were measured at baseline, mid-treatment, post-treatment, and one-month follow-up using the Beck Depression Inventory-II (BDI-II), Beck Anxiety Inventory (BAI), and Pittsburgh Sleep Quality Index (PSQI). The IC-CES system used real-time EEG acquisition, preprocessing (FastICA, current-source-density filtering), and feature extraction (400-dimensional vector of statistical and spectral markers). A K-Nearest Neighbors model, optimized for sub-millisecond decision-making, selected stimulation parameters by matching patients' EEG patterns with a normative database. Data were analyzed using repeated-measures ANOVA under both per-protocol and intention-to-treat frameworks; missing values (8.3%) were imputed using linear regression.

Results : IC-CES demonstrated greater improvements in depression and anxiety compared with conventional CES. Participants in the IC-CES group showed consistent reductions in BDI-II and BAI scores from baseline through one-month follow-up, with symptom reductions exceeding typical responses reported for standard CES. EEG analyses revealed normalization of depression-related neural patterns, including reduced frontal theta activity and enhanced alpha coherence, which correlated with clinical improvement. No significant differences were observed between groups in sleep quality (PSQI). Both treatments were safe and well tolerated, with no serious adverse events; minor side effects were limited to transient scalp tingling in a small subset of patients.

Conclusion : This study provides the first evidence that an EEG- and machine learning–guided closed-loop CES system is feasible, safe, and clinically beneficial for patients with MDD. By adapting stimulation parameters to each patient's dynamic brain activity, IC-CES achieved superior symptom reduction and measurable neurophysiological recovery compared with conventional CES. These findings highlight the potential of intelligent neuromodulation to advance personalized psychiatry. While limitations include the absence of a sham control and relatively short follow-up, the results justify larger sham-controlled trials to confirm efficacy and long-term benefits. IC-CES represents a promising step toward precision, technology-driven treatment for depression.

Keywords : Major depressive disorder; Machine learning; Electric stimulation therapy; Brain Signals

Count: 187
Abstract ID: 483
subject: Novel and Cutting-Edge Technologies: Brain Machine Interface and Neuroengineering
Presentation Type: Oral

AI-Augmented Cranial and Growth Monitoring in Infants

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Background and Aim : The first two years of life represent a critical period for brain and cranial development, during which early deviations in growth trajectories may indicate underlying neurodevelopmental disorders. Cranial deformities such as plagiocephaly and brachycephaly, alongside inadequate growth parameters (length-for-age, stunting), are common markers. Traditional diagnostic methods (manual calipers, MRI/CT imaging) are either inaccurate, invasive, or inaccessible in low-resource settings. Existing digital solutions like SoftSpot focus narrowly on cranial indices and lack integration with broader pediatric growth monitoring. There is an urgent need for an accessible, AI-driven, multimodal tool to provide accurate cranial and growth assessments while generating human-centered clinical insights for physicians and parents.

Methods : We developed Pedia AI Care, a novel computer-vision and multimodal large language model (LLM) platform for infant cranial and growth monitoring. The pipeline consists of: Data acquisition: Parents capture top-view and side-view cranial photos via smartphone; length/height is input manually or estimated through ArUco markers. Computer vision analysis: OpenCV-based segmentation and ellipse fitting yield cranial indices, including Cranial Index (CI), Cranial Vault Asymmetry Index (CVAI), and complementary measures (OCLR, CSR). Quality checks (blur, brightness, calibration markers) ensure measurement validity. Anthropometric assessment: Length/height is converted to WHO LMS Z-scores and percentiles. AI Assistant: A multimodal LLM (Gemma3-Vision via Ollama) integrates quantitative indices, growth data, and session history to generate two types of reports: (a) structured clinical summaries with red flags for physicians, and (b) simplified, empathetic narratives for parents. Prototype implementation: MVP built in Streamlit, SQLite, OpenCV, and Ollama provides a clinician dashboard, parent interface, and automated PDF reports.

Results : Preliminary internal testing on simulated and limited clinical datasets demonstrates that: Accuracy: CI and CVAI measurements are comparable to mechanical calipers and existing digital tools (e.g., SoftSpot). LLM Narratives: Reports generated by Gemma3 were clinically coherent for physicians while remaining parent-friendly and understandable. Usability: The prototype interface provided guided photo-taking, traffic-light coding (green/yellow/red), and growth curve visualization based on WHO standards. Extensibility:

Future modules, such as neonatal jaundice detection (colorimetric analysis of skin/sclera) and integration with vaccination/milestone tracking, have been outlined for expansion.

Conclusion : Pedia AI Care exemplifies how AI-augmented technologies—specifically computer vision and multimodal LLMs—can transform early pediatric health monitoring. By enabling non-invasive, low-cost, parent-driven screening with explainable, humanized reporting, this platform offers the potential to: Improve early detection of neurodevelopmental disorders. Reduce unnecessary clinical visits. Support clinicians in continuous child growth surveillance. Enhance equity in access to pediatric care, particularly in low-resource settings. This work highlights a promising paradigm for integrating Novel and Cutting-Edge Technologies into the domain of Developmental Neuroscience, paving the way for scalable telemedicine solutions and AI-powered pediatric care.

Keywords : Artificial Intelligence (AI); Computer Vision; Multimodal Large Language Models (LLM); Cranial Index (CI); Cranial Vault Asymmetry Index (CVAI); Neurodevelopmental Disorders; Pediatric Growth Monitorin

Count: 188
Abstract ID: 355
subject: Novel and Cutting-Edge Technologies: Brain Machine Interface and Neuroengineering
Presentation Type: Poster

Adaptive Brain-Machine Interface Using Local Field Potentials for Spinal Motor Rehabilitation

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Conclusion : Spinal cord injuries (SCI) often result in irreversible motor impairments, prompting the development of neurotechnological solutions for functional recovery. Brain-Machine Interfaces (BMIs) have emerged as a promising approach by decoding neural signals and translating motor intentions into actionable outputs. This study introduces an adaptive BMI system based on intracortical Local Field Potential (LFP) signals recorded from the primary motor cortex. Unlike spike-based decoding, LFPs offer greater long-term stability and lower susceptibility to signal degradation. Using machine learning algorithms such as Support Vector Machines (SVM) and Long Short-Term Memory (LSTM) networks, motor intentions were decoded and mapped to targeted spinal cord stimulation via epidural electrodes. The system achieved decoding accuracies of 85–90%, with stimulation latency under 200 ms, enabling coordinated limb movements in paralyzed animal models. Adaptive recalibration significantly improved performance over time, demonstrating robustness against signal non-stationarity. These findings suggest that LFP-driven BMIs can serve as a viable and scalable solution for motor rehabilitation in SCI patients, with potential for clinical translation and integration into wearable neuroprosthetic systems.

Keywords : - Brain-Machine Interface (BMI) - Local Field Potential (LFP), Adaptive Machine Learning, Neuroprosthetics

Count: 189
Abstract ID: 816
subject: Novel and Cutting-Edge Technologies: Brain Machine Interface and Neuroengineering
Presentation Type: Oral

Precision Medicine using Digital Twins and Artificial Intelligence

Submission Author: Nasibeh Rady Raz

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Background and Aim : Artificial intelligence (AI) with powerful computational algorithms revolutionizes diagnosis and prognosis and develops personalized treatment through the utilization of large multimodal data. Currently, we utilise AI as an assistance in personalised medical decision-making and detecting biomarkers. However, there is a need for a framework to perform the pilot personalized treatment. Therefore, there is a need for a combination of AI and digital twins.

Methods : In this paper, we first review AI applications in precision medicine, with a focus on neurodegenerative diseases. Then, review the current medical digital twins, including cells, tissues, organs, and the entire body, and discuss several examples, including neurosurgery and modeling physiology.

Results : Results indicate significant potential in the intersection of AI and digital twins for precision medicine.

Conclusion : AI and digital twins are facilitating precision medicine, particularly in neurodegenerative diseases, by enabling personalized prognostic and treatment systems.

Keywords : Artificial intelligence, Digital Twins, Precision Medicine, Neuro Biomarkers

Count: 190
Abstract ID: 552
subject: Novel and Cutting-Edge Technologies: Drug Discovery and Neuropharmacology
Presentation Type: Poster

Comparative Analysis of Neurological and Psychological Side Effects Across Different Classes of Doping Drugs: From Steroids to Nootropics

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Conclusion : Doping in sports and performance enhancement contexts involves the use of various pharmacological agents to gain competitive advantages, often at significant health costs. This review provides a comprehensive comparative analysis of the neurological and psychological side effects associated with major classes of doping drugs, including anabolic-androgenic steroids (AAS), stimulants, human growth hormone (HGH), erythropoietin (EPO), beta-blockers, and nootropics. Drawing from recent literature up to 2025, we examine mechanisms of action, acute and chronic effects, and comparative risks. AAS are linked to profound mood dysregulation, aggression, and cognitive deficits; stimulants induce euphoria but risk psychosis and neurotoxicity; HGH and EPO show subtler neuropsychiatric impacts, often indirect via cardiovascular strain; beta-blockers contribute to fatigue and depression; and nootropics, while promising for cognition, may paradoxically impair long-term brain plasticity. Comparative insights reveal dose-dependent vulnerabilities, with AAS and stimulants posing the highest risks for severe psychiatric sequelae. This synthesis underscores the need for enhanced monitoring, education, and policy interventions to mitigate these underrecognized hazards.

Keywords : Doping; Anabolic-androgenic steroids; Stimulants; Human growth hormone; Erythropoietin; Beta-blockers; Nootropics; Neurological side effects; Psychological side effects;

Count: 191
Abstract ID: 469
subject: Novel and Cutting-Edge Technologies: Molecular, Biochemical, and Genetic Techniques & Gene . Therapy
Presentation Type: Poster

Harnessing CRISPR/Cas9 Genome Editing for Modeling and Therapeutic Intervention Neurological Disorders

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Conclusion : Background: The precise modeling of human neurological diseases has long been a significant challenge in neuroscience. Traditional animal models often fail to fully recapitulate the complex genetic and pathological hallmarks of disorders such as Alzheimer's disease, Parkinson's disease, and Amyotrophic Lateral Sclerosis (ALS). The advent of Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) and its associated protein 9 (Cas9) has revolutionized genetic engineering, offering unprecedented precision, efficiency, and flexibility. This technology provides a powerful tool not only for creating more accurate disease models but also for exploring potential therapeutic strategies through direct genetic correction. Objective: This review aims to comprehensively outline the application of the CRISPR/Cas9 system in the generation and correction of in vitro and in vivo models of neurological diseases. We focus on its role in elucidating disease mechanisms and its potential as a therapeutic modality. Methods: A systematic literature search was conducted using databases such as PubMed, Scopus, and Google Scholar, focusing on studies published from 2014 onwards. Keywords included "CRISPR/Cas9," "genome editing," "neurological disorders," "iPSCs," "disease modeling," "neurodegenerative diseases," and "gene therapy." The analysis included studies that utilized CRISPR/Cas9 for creating knock-out, knock-in, or point mutation models in cell lines (especially patient-derived induced pluripotent stem cells (iPSCs)) and animal models, as well as those demonstrating genetic correction. Results: The integration of CRISPR/Cas9 has profoundly advanced the field of neurological disease modeling by enabling the creation of highly precise and physiologically relevant systems. A primary application involves the generation of isogenic induced pluripotent stem cell (iPSC) lines, where the technology is used to introduce disease-causing mutations into healthy iPSCs or correct them in patient-derived cells, thereby creating genetically matched controls that isolate the specific effects of a mutation from background genetic noise upon differentiation into neurons. This precision has been instrumental in elucidating pathogenic mechanisms, allowing researchers to perform functional genomics studies through precise gene knock-out or allele-specific editing to validate the roles of specific genes in processes like neurodegeneration and synaptic dysfunction. Furthermore, CRISPR/Cas9 has revolutionized the development of in vivo animal models by enabling the efficient incorporation of human-relevant mutations, leading to more translatable preclinical studies. Most notably, the technology has provided compelling proof-of-concept for therapeutic strategies, with studies

demonstrating the successful correction of mutations in both iPSCs and animal models, resulting in the reversal of pathological hallmarks such as toxic protein aggregation and the restoration of neuronal function. Conclusion: CRISPR/Cas9 technology has become an indispensable tool in the neuroscientist's arsenal, enabling the creation of highly precise and human-relevant models of neurological disorders. Its application extends beyond modeling to offer a direct path toward developing novel gene therapies. While challenges related to safe and efficient in vivo delivery, off-target effects, and ethical considerations remain active areas of research, the current evidence firmly establishes CRISPR/Cas9 as a transformative force for understanding and potentially curing devastating neurological diseases. Future work will likely focus on refining editing efficiency and developing innovative delivery systems to translate these breakthroughs into clinical applications.

Keywords : CRISPR/Cas9, Genome Editing, Neurological Disorders, Disease Modeling

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Abstract ID: 470
subject: Novel and Cutting-Edge Technologies: Molecular, Biochemical, and Genetic Techniques & Gene . Therapy
Presentation Type: Poster

Integrating Machine Learning with Multi-Omics for the Discovery of Novel Biomarkers in Early Neurological Disorder Diagnosis

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Conclusion : Background: The early and accurate diagnosis of neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD) is paramount for enabling timely intervention. Traditional diagnostic methods, which often rely on overt clinical symptoms that appear after significant neuronal loss, lack the sensitivity for early detection. This diagnostic gap underscores the critical need for novel, objective molecular biomarkers that can identify pathology during the pre-symptomatic phase. Objective: This review aims to critically evaluate the paradigm shift in biomarker discovery, moving beyond single-molecule analysis to integrated multi-omics approaches powered by advanced machine learning (ML) and artificial intelligence (AI) algorithms. We focus on how these technologies are revolutionizing the identification and validation of biomarker signatures from complex biological data. Methods: A systematic analysis of recent literature (2020-2024) from databases including PubMed, , Scopus, and Google Scholar, was conducted. The review prioritized studies that utilized high-throughput technologies—such as genomics, proteomics, metabolomics, and exosome-derived miRNA sequencing—coupled with ML models for pattern recognition in biofluids like blood plasma and cerebrospinal fluid (CSF). Results: The contemporary approach to biomarker discovery is characterized by the integration of multi-omics data and sophisticated computational analytics. Machine learning algorithms—including support vector machines (SVM), random forests, and deep neural networks—are now essential for deciphering complex, high-dimensional datasets. They excel at identifying subtle, non-linear patterns that elude conventional statistical methods, leading to the discovery of multi-analyte biomarker panels rather than reliance on a single molecule. For instance, ML models integrating levels of Aβ42, p-tau, NFL, and specific inflammatory markers from plasma have demonstrated diagnostic accuracy rivaling that of CSF analysis or PET imaging. Furthermore, unsupervised learning techniques like clustering are used to delineate disease subtypes with distinct molecular profiles, paving the way for personalized medicine. The application of ML also extends to analyzing digital biomarkers from wearables and neuroimaging data, creating a holistic diagnostic framework. Conclusion: The convergence of high-throughput multi-omics and artificial intelligence marks a new era in neurology. Machine learning is not merely an auxiliary tool but a transformative force that enhances the sensitivity, specificity, and predictive power of biomarker discovery. By leveraging these technologies, researchers can identify robust, multiplex biomarker signatures capable of detecting neurological pathologies at their

earliest stages. Future efforts must focus on standardizing protocols, improving ML model interpretability ("explainable AI"), and validating these signatures in large-scale, longitudinal cohorts to accelerate their translation into clinical practice, ultimately enabling pre-symptomatic diagnosis and personalized therapeutic strategies.

Keywords : Machine Learning, Multi-Omics, Biomarkers, Neurological Disorder

Count: 193
Abstract ID: 1029
subject: Novel and Cutting-Edge Technologies: Molecular, Biochemical, and Genetic Techniques & Gene . Therapy
Presentation Type: Poster

Reprogramming Spinal Cord Repair: DNA-Encoded Hydrogels as Translational Platforms for Personalized CRISPR and Drug Delivery in 3D Bioprinting

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Conclusion : Introduction: Spinal cord injury (SCI) is a devastating condition affecting 930,000 people each year globally, which often leads to permanent paraplegia and imposes a tremendous socioeconomic burden. Despite surgical decompression and high-dose glucocorticoids as routine interventions, they fail to trigger spontaneous recovery of neural loss owing to inflammation and glial scar formation. Neural stem cells (NSCs) have demonstrated therapeutic potentials in microvascular regeneration but remain limited by poor survival rate, uncontrolled differentiation, and unstable engraftment within damaged spinal tissue. These challenges have redirected regenerative technologies focusing on restoring the native neural tissue rather than merely repairing it. Compared to traditional scaffolds, 3D bioprinting, an emerging technology, enables the fabrication of patient-specific spinal scaffold and precise spatial control over cells and biomaterials, which creates a suitable microenvironment for axonal regeneration. Within this paradigm, DNA-based hydrogels have arisen as “smart” bio-inks, capable of organizing biomimetic scaffolds, editing genes and releasing therapeutic agents, bringing personalized neuro-regeneration. Method: Searching databases like PubMed, Scopus, and Google scholar using keywords such as “Spinal cord injury”, “DNA hydrogel”, ”3D bioprinting”,and “CRISPR/Cas” was conducted. Result: Recent research has highlighted the versatility of DNA hydrogels, which are made through injection/extrusion-based methods, as both functional and structural elements appropriate for 3D bioprinting. As an attractive structure of DNA, it contains a charged chemical backbone due to the presence of phosphate groups and a secondary structure stabilized by weak interactions (hydrogen bonds and π- π stacking between aromatic structures). These ideal characteristics form a supramolecular system with electrostatic interactions, which has attracted attention in tissue engineering because of the tunable viscoelasticity, biocompatibility, and biodegradation features of DNA hydrogels. When embedded with NSCs, hydrogels mimic the extracellular matrix (ECM), supporting neuronal differentiation and axonal alignment. Beyond structure, DNA’s programmable nature enables it to encapsulate and precisely release siRNAs, mRNA, and CRISPR/Cas systems. Additionally, applying gene editing by CRISPR provides a trigger to control drug release in DNA hydrogels. By breaking down specific DNA components within hydrogel, CRISPR editing controls the on-demand release of drugs due to the varied stage of

disease and location. In this regard, DNA nanogels target genes that inhibit axon regeneration, such as chemokine (C-C motif) ligand 2(CCL2) and PTEN, while CRISPR-based modulation of transcription factors like Neurogenin2(Ngn2) and ISL LIM Homeobox 1(ISI1) enhances astrocytes reprogramming and differentiation. In the integrated design by 3D bioprinting and synthetic DNA, these hydrogels can be engineered to simultaneously deliver a gene editing toolbox and neurotrophic factors, optimizing 3D bioprinted scaffolds in a personalized manner. Conclusion: This study provokes a next-generation platform, where DNA-encoded bioprinting units structural and personalized neuro-regeneration in SCI. By merging the informational capacity of DNA with the architectural control of 3D bioprinting and the specificity of CRISPR systems, patient-tailored scaffolds are introduced. Accordingly, future research should focus on developing cost-effective and large-scale methods of DNA synthesis and isolation. Additionally, developing techniques for designing DNA hybrid hydrogels with multistep crosslinking strategies is vital. To integrate DNA bio-printed hydrogels with precision therapeutic strategies, further animal studies and RCTs should be included.

Keywords : CRISPR/Cas, 3D bioprinting, Drug delivery, DNA hydrogel, Spinal Cord Injury, Neural Stem Cells

Count: 194
Abstract ID: 490
subject: Novel and Cutting-Edge Technologies: Molecular, Biochemical, and Genetic Techniques & Gene . Therapy
Presentation Type: Poster

The Role of Matrix Metalloproteinase-9 in Learning and Memory Impairment Induced by REM Sleep Deprivation and Stress in Male Wistar Rats

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Background and Aim : Stress and sleep deprivation are critical environmental factors that negatively affect cognitive performance, neural plasticity, and hippocampal function. Matrix metalloproteinase-9 (MMP-9), a zinc-dependent endopeptidase, is essential for extracellular matrix remodeling and synaptic plasticity, playing a pivotal role in learning and memory. Despite its recognized role in neurophysiology, the involvement of MMP-9 in cognitive deficits induced by stress and REM sleep deprivation remains poorly understood. The present study aimed to investigate the role of MMP-9 in learning and memory impairments induced by chronic stress and REM sleep deprivation in male Wistar rats.

Methods : Male Wistar rats (170–250 g) were housed under controlled temperature (23 ± 2°C) and a 12 h light/dark cycle, with ad libitum access to food and water. Animals were randomly assigned to four groups: control, stress, sleep deprivation, and stress + sleep deprivation. Stress paradigms and REM sleep deprivation protocols were applied according to established methods. Locomotor activity and anxiety-like behavior were assessed using the open field test (OFT), while spatial working memory was evaluated with the Y-maze. Additional cognitive assessments included the Morris water maze for spatial learning and the shuttle box test for avoidance memory. Following behavioral testing, hippocampal tissues were collected for histological examination using Nissl staining and biochemical analysis of MMP-9 expression.

Results : Preliminary findings demonstrated that both stress and REM sleep deprivation significantly impaired spatial learning and memory performance compared to control animals. Notably, animals in the stress + sleep deprivation group exhibited the most pronounced deficits. Concurrently, hippocampal MMP-9 expression was significantly elevated in all experimental groups relative to controls. These results suggest that increased MMP-9 activity may mediate synaptic and cognitive impairments associated with stress and sleep loss.

Conclusion : MMP-9 appears to act as a critical mediator in stress- and REM sleep deprivation-induced cognitive deficits. Targeting MMP-9 activity may represent a potential therapeutic

strategy to mitigate memory impairments arising from chronic stress and disrupted sleep, providing insights into interventions for stress-related cognitive dysfunction.

Keywords : MMP-9, stress, REM sleep deprivation, memory, hippocampus.

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Abstract ID: 486
subject: Novel and Cutting-Edge Technologies: Molecular, Biochemical, and Genetic Techniques & Gene . Therapy
Presentation Type: Oral

Multi-Omics and neuroimaging insights into synaptopathy of the posterior cingulate cortex in early Alzheimer's disease

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Background and Aim : Alzheimer's disease (AD), the leading cause of dementia, is characterized by progressive memory loss and synaptic dysfunction (synaptopathy), yet its precise pathogenesis remains unclear. Synapses are critical for neuronal communication, memory, and cognitive functions, with tau and amyloid-beta (Aβ) pathology disrupting axonal transport and synaptic integrity. The posterior cingulate cortex (PCC), highly connected to hippocampal, prefrontal, parietal, and entorhinal regions, is among the earliest affected areas in AD, contributing to memory, attention, navigation, and other cognitive deficits

Methods : In this multicenter cross-sectional study, 16 AD patients (MMSE 19–23) and 11 age- and sex-matched controls underwent structural MRI and proton magnetic resonance spectroscopy (1H-MRS) of the PCC. Metabolites, including N-acetyl aspartate (NAA), myo-inositol, GABA, glutamate, taurine, glycine, choline, and creatine, were quantified. Transcriptomic data from GEO datasets (27 subjects) were analyzed for synaptic gene expression, while DNA methylation was assessed via Illumina 850K arrays. Neuropathology (Aβ plaques, NFTs, neuritic plaques) and cognitive status were evaluated following NIA-AA and DSM-IV criteria. Functional enrichment and protein-protein interaction (PPI) networks were constructed to identify key synaptic pathways and hubs.

Results : MRS revealed significant alterations in metabolite levels in AD: N-acetyl aspartate (NAA)/Cr, GABA/Cr, taurine, and glycine were reduced, whereas myo-inositol/Cr, choline/Cr, and glutamate were elevated. Notably, glutamate levels negatively correlated with MMSE

scores, linking excitotoxicity to cognitive decline. Transcriptomic analysis of PCC synaptic genes from GEO datasets showed complex expression changes. Downregulated genes included muscarinic acetylcholine, GABA, serotonin, dopamine, glutamate, and ephrin receptors, as well as key presynaptic and structural proteins such as neurexin, neuroligin, GAP-43, synaptophysin, synaptotagmin, syntaxin, VAMP, tubulin, kinesin, and dynactin. Upregulated genes included DRD2, APP, TMEM97, and select synaptotagmins. Gene expression changes correlated with disease severity (Braak staging), indicating progressive synaptic dysfunction. Functional enrichment analysis revealed that downregulated genes were enriched in axo-dendritic transport, SNARE complex formation, microtubule motor activity, and GABAergic synapses, whereas upregulated genes were associated with Ephrin, serotonin, and dopamine signaling. DNA methylation analysis revealed variable patterns across synaptic genes. Many were hypomethylated, particularly in individuals with high neurofibrillary tangle (NFT) scores, while others were hypermethylated or displayed both patterns depending on genomic regions. Methylation changes correlated with amyloid plaque and NFT burden, reflecting complex epigenetic regulation in AD. A PPI network of 165 synaptic proteins revealed key hubs: downregulated—SNAP25, MAPT, GRIN2A, KIF9, EPHA4, GABRA1; upregulated—GRIN2A, EPHB2, HTR1A, STXBP5, GABRG3, KIF2B, highlighting critical nodes in major neurotransmitter and synaptic pathways.

Conclusion : AD is characterized by widespread downregulation of synaptic proteins, disrupted neurotransmitter receptor expression, and selective upregulation of proteins involved in Aβ binding or clearance. Presynaptic proteins (GAP-43, SNAP-25, synaptotagmin, synaptobrevin, syntaxin, synaptophysin) generally decline, whereas postsynaptic proteins such as TMEM97, neuroligin, and LRP1 are upregulated. Dysregulation of MAPT and tau isoforms, coupled with impaired axonal transport, contributes to dystrophic neurites. Epigenetic modifications parallel disease severity: NFT-positive individuals show hypomethylation of synaptic genes, while higher neuritic plaque scores correspond to hypermethylation. These findings underscore the intricate molecular interplay underlying synaptopathy in AD and highlight potential therapeutic targets, including TMEM97, Ephrin receptors, and postsynaptic signaling proteins.

Keywords : Alzheimer’s disease; Transcriptome Analysis, Gene Methylation; Magnetic Resonance Spectroscopy; Bioinformatics.

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Abstract ID: 658
subject: Novel and Cutting-Edge Technologies: Molecular, Biochemical, and Genetic Techniques & Gene . Therapy
Presentation Type: Oral

Decoding the Molecular Landscape of Chorea-Acanthocytosis through Integrated Exome Sequencing and Protein Interaction Network Analysis

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Background and Aim : Chorea-acanthocytosis (ChAc) is a rare autosomal recessive neurodegenerative disorder characterized by hyperkinetic movements, orofacial dyskinesia, elevated serum creatine kinase, and the presence of acanthocytes in peripheral blood. The disease results from pathogenic variants in the vacuolar protein sorting-associated protein 13A (VPS13A) gene, encoding a large cytoplasmic protein implicated in vesicular transport and lipid exchange. Although numerous VPS13A mutations have been reported worldwide, the molecular spectrum and functional network context of the gene remain unexplored in Iranian patients. The present study aimed to investigate the clinical, molecular, and systems-level characteristics of Iranian patients with ChAc through genetic variant analysis, protein–protein interaction (PPI) network construction, and pathway enrichment analysis.

Methods : Five unrelated Iranian patients with clinical features suggestive of ChAc—including progressive chorea, dystonia, dysarthria, and cognitive decline—were recruited. Brain MRI consistently revealed caudate atrophy and putaminal hyperintensity on T2/FLAIR images. WES was conducted to identify pathogenic variants, followed by Sanger sequencing confirmation. Functional consequences of novel mutations were predicted using In silico prediction tools. To explore the molecular landscape beyond VPS13A, a comprehensive PPI network was constructed using the STRING database (v12.0) and visualized in Cytoscape (v3.9.1). Genes interacting with VPS13A were retrieved and cross-referenced against patient WES data to identify additional rare, potentially deleterious variants. Functional enrichment and pathway analysis were performed using the String Enrichment Plugin (v2.2.0), focusing

on Gene Ontology (GO) terms and KEGG signaling pathways related to neurodegeneration and vesicular transport.

Results : WES revealed five distinct VPS13A variants, including four novel mutations: one nonsense (p.L984X) and three splice-site mutations (c.755-1G>A, c.144+1G>C, c.2512+1G>A). All were predicted to cause loss of function and were confirmed by Sanger sequencing.PPI network analysis demonstrated strong functional connectivity between VPS13A and proteins involved in vesicular trafficking, lipid transport, cytoskeletal regulation. Functional enrichment highlighted mitochondrial organization, endosomal transport, and synaptic vesicle cycling as the most significantly enriched pathways, aligning with the observed neurodegenerative phenotype.

Conclusion : This study presents the first comprehensive molecular and systems-level analysis of ChAc in Iranian patients, identifying four novel VPS13A mutations and providing new insights into the functional network and biological pathways associated with the disease. These findings expand the known VPS13A mutational spectrum, and underscore the value of integrative genomics and PPI-based pathway analysis in elucidating the molecular mechanisms underlying rare neurodegenerative disorders.

Keywords : Chorea-Acanthocytosis, VPS13A, Whole-exome sequencing, In silico, Network, Pathway,

Count: 197
Abstract ID: 590
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Investigating the protective effect of Rosa Damascena essential oil in preventing ethanol toxicity on neural stem cells

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Background and Aim : Neural progenitor cells (NPCs) in discrete adult brain niches are promising targets for therapies against central nervous system disorders and neurodegenerative diseases such as Parkinson's and Alzheimer's. Rosa damascena essential oil is rich in flavonoids and other bioactive constituents that exert antioxidant, anti inflammatory, and cytoprotective effects against neuronal injury. These properties support investigation of the oil as a candidate neuroprotective agent for NPCs and their differentiated progeny. To evaluate whether Rosa damascena essential oil (ESO) attenuates ethanol (ETH) induced toxicity in NPCs and in differentiated neural cells (NCs), and to characterize effects on viability and markers of progenitor state and neuronal differentiation.

Methods : NPCs were isolated from the brains of young Wistar rats and expanded under standard neurosphere/adherent culture conditions. Differentiation into mature NCs was induced by withdrawal of mitogenic/supportive factors. Dose–response and time course toxicity were established by MTT assay to define effective ETH and ESO concentrations. Gene expression in NPCs was quantified by real time PCR for Nestin, Sox2, and Pax6. In differentiated NCs, neuronal markers Tuj1 and Map2 were assessed by real time PCR and TAU protein expression was examined by immunocytochemistry. Statistical comparisons between ETH, ESO, ETH+ESO, and control groups were performed with significance set at $p < 0.05$.

Results : Ethanol reduced cell viability in a dose and time dependent manner across 24, 48, and 72 hours. Co treatment with 200 µM ESO significantly improved survival of both NPCs and NCs compared with ETH alone ($p < 0.001$). ESO decreased TAU immunoreactivity in ETH exposed NCs. In NPCs, ETH+ESO treatment increased expression of Nestin, Sox2, and Pax6 relative to ETH alone ($p < 0.01$). In NCs, Tuj1 and Map2 expression was significantly higher in the ETH+ESO group than in ETH treated cells ($p < 0.01$).

Conclusion : Rosa damascena essential oil exerts significant neuroprotective effects in vitro by preserving viability, sustaining progenitor marker expression, promoting neuronal differentiation markers, and reducing TAU accumulation in cells exposed to ethanol. These

data support further preclinical investigation of ESO as a therapeutic candidate to mitigate neurodegenerative damage and to support neural regeneration.

Keywords : Neuroprotection; Neurodegeneration; Rosa damascena essential oil; Neural progenitor cells; Ethanol toxicity; Neural differentiation.

Count: 198
Abstract ID: 581
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Neuroprotective and neuroregenerative effects of boswellic acid on neural progenitor cells in vitro

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Background and Aim : The adult central nervous system exhibits limited regenerative capacity, and current therapies for neurodegenerative diseases remain largely symptomatic, creating substantial societal and economic burdens. Neural progenitor cells (NPCs) possess self renewal capacity and the potential to differentiate into diverse neural cell types, making them attractive targets for regenerative strategies. Phytochemicals can modulate cellular survival, proliferation, and differentiation; beta boswellic acid (BA), a major bioactive constituent of Boswellia species, has been associated with synaptic remodeling and neuronal repair mechanisms. To evaluate the neuroprotective and neuroregenerative effects of beta boswellic acid on rat NPCs and their differentiated neural progeny in vitro, and to define its capacity to counteract ethanol induced cytotoxicity.

Methods : NPCs were isolated from neonatal Wistar rat brains and expanded under standard neurosphere/adherent culture conditions. Effective concentrations of ethanol (ETH) and BA were established by MTT dose–response assays. Experimental groups included control, ETH, BA, and ETH+BA. In NPCs, expression of progenitor markers Nestin, Sox2, and Pax6 was quantified by real time PCR. Following differentiation induced by withdrawal of mitogens, neural cells (NCs) were analyzed for neuronal markers Tuj1 and MAP2 by real time PCR, and TAU protein accumulation was assessed by immunocytochemistry. Data were analyzed using appropriate statistical tests with predetermined significance thresholds.

Results : Ethanol produced a concentration and time dependent reduction in viability at 24, 48, and 72 hours, reaching significance at 2% ETH ($p < 0.0001$). ETH markedly suppressed progenitor markers (Nestin, Sox2, Pax6) in NPCs and reduced neuronal differentiation markers (Tuj1, MAP2) in NCs compared with controls ($p < 0.0001$). Co treatment with 200 µM BA

significantly rescued viability in both NPCs and NCs relative to ETH alone ($p < 0.001$) and prevented ETH induced upregulation of TAU in differentiated NCs. ETH+BA restored progenitor marker expression in NPCs ($p < 0.01$) and increased Tuj1 and MAP2 expression in NCs compared with ETH treatment alone ($p < 0.01$).

Conclusion : Beta boswellic acid confers robust neuroprotective and neuroregenerative effects in vitro by preserving cell viability, maintaining progenitor identity, promoting neuronal differentiation, and limiting TAU accumulation under ethanol challenge. These results support further mechanistic studies and preclinical evaluation of BA as a candidate therapeutic to mitigate toxin induced neuronal injury and enhance neural repair.

Keywords : Boswellic acid; Neuroprotection; Neuroregeneration; Neural progenitor cells; Neuronal differentiation; Ethanol toxicity.

Count: 199
Abstract ID: 575
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Unveiling the Neuroprotective and Differentiation Effects of Quercetin on Neural Progenitor Cells: A Laboratory Perspective

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Background and Aim : Ethanol consumption can lead to widespread cellular damage, primarily through the generation of reactive oxygen species (ROS), which induce oxidative stress. This stress affects essential cellular components, including DNA and proteins, and is implicated in the onset and progression of various chronic and neurodegenerative disorders. Natural antioxidants found in plants can neutralize ROS, offering cellular protection. Among them, quercetin, a flavonoid abundant in many fruits and vegetables, has shown strong antioxidant and neuroprotective effects. This study investigates the potential of quercetin to protect neural progenitor cells (NPCs) from ethanol-induced oxidative damage.

Methods : NPCs were isolated from neonatal Wistar rat hippocampi and cultured under standardized conditions. Differentiation into mature neural cells (NCs) was initiated by withdrawing progenitor-supporting factors. The optimal concentrations of ethanol and quercetin were determined using the MTT assay, followed by assessments of cell viability, differentiation status, and ROS-related responses across different treatment groups.

Results : A dose-dependent decrease in cell viability was observed following exposure to both ethanol and quercetin for 24 and 48 hours, with statistically significant reductions at higher concentrations ($p < 0.001$). Cells treated with ethanol alone exhibited the highest mortality. In contrast, treatment with quercetin alone or in combination with ethanol significantly preserved cell viability ($p < 0.01$). Moreover, ethanol-treated cells showed an increase in antioxidant activity, indicating a cellular response to oxidative insult ($p < 0.001$).

Conclusion : These findings support quercetin's protective role against ethanol-induced oxidative stress, highlighting its therapeutic potential in maintaining neural cell health and function under toxic conditions.

Keywords : Quercetin ;Neural Progenitor Cells ; Ethanol ; Oxidative Stress ; Antioxidant

Count: 200
Abstract ID: 1018
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Unrevealing early-onset Alzheimer's disease: insights to the mechanisms

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Conclusion : Alzheimer's disease (AD) is one of the most common problems of the current century, characterized by memory loss, cognitive decline, and impairment in a person's functioning. Alzheimer's disease is often known to affect older people (OPAD), but recent studies have reported early onset of Alzheimer's disease (EOAD) at younger ages. Clinically, EOAD often follows the same fundamental neuropathology in OPAD, such as amyloid-β plaques, tau-neurofibrillary tangles, and neuronal loss, but may present with somewhat different or more aggressive symptoms and disability. EOAD is associated with a large societal, personal, and economic impact. Therefore, identifying and preventing the causes of EOAD is a necessity. The reasons for EOAD are less well understood than those for OPAD. So, in this review, we explain the genes and factors involved in EOAD. Research has shown that the common reason for EOAD is familial genetic mutation. Mutations of amyloid precursor protein genes, presenilin 1 (PSEN1), and presenilin 2 (PSEN2) are the most well-known, accounting for 70% of familial and 5 10% of total causes of EOAD. Dysfunction of the endo-lysosomal pathway, such as autophagy, plays a role in brain cytotoxicity and neural death. It results in the accumulation of misfolded proteins in the brains of individuals with EOAD. Some of the endo-lysosomal transport genes that are associated with EOAD risk include RIN3, RUFY1, TCIRG1, and PSD2. The protein Pyk2 plays a critical role in synaptic dysfunction during the early stages of Alzheimer's disease. Pyk2 interacts with Tau, a protein associated with neurofibrillary tangles in Alzheimer's, and directly phosphorylates it, contributing to synaptic loss. Besides the "amyloid-β pathway" genes, mutations and dysfunctions in synaptic processes, immune processes, and lipid metabolism are also contributing factors in EOAD. In addition, EOAD cases due to non-genetic factors are also significant. Early-life microbiome alterations could lead to neuroinflammation changes in the brain and facilitate EOAD in later years. Air pollution, including nitrogen dioxide and carbon monoxide, increases EOAD. In middle age, smoking cuts verbal fluency decline by half and slows memory loss by 20%. It seems that vitamin deficiencies, especially vitamin D3, have a critical role in EOAD. Vitamin D3 deficiency results in a decrement in the synthesis of neurotrophic agents such as nerve growth factor (NGF), glial cell line-derived neurotrophic factor (GDNF), neurotrophin-3, and the synthesis of low-affinity p75 NTR receptors. These effects decline neural survival. In conclusion, studies indicate that, in addition to genetic mutations, various factors contribute to the onset of Alzheimer's disease at a young age, many of which are still not well understood.

Even seemingly non-genetic factors may lead to genetic mutations that have yet to be identified. To prevent the early onset of Alzheimer's disease, there is a need for more comprehensive and holistic studies, as well as a better understanding of precise markers for timely diagnosis and treatment.

Keywords : early-onset Alzheimer’s disease; Alzheimer's; young adults; familial genetic mutation; gut- brain axis

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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Novel insights into the role of Inositol supplementation in Alzheimer's disease: A Review

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Conclusion : Background and Aim: Alzheimer's disease (AD)- an irreversible neurodegenerative condition- is followed by dementia in almost 50–70% of cases. The pathogenesis of AD comprises of amyloid-beta (Aβ) accumulation, insulin resistance (IR), tau protein dysregulation, and neuro-inflammation. AD severely affects daily function and social behavior, highlighting the urgent need for early prevention and effective alternative therapies. Inositols (INS) as carbocyclic polyols exert a plethora of pharmacological properties in metabolic and mood disorders as well as neuropsychiatric conditions like AD. INSs have exerted neuroprotection against tau-related degeneration in AD. A number of stereoisomers including myo-inositol (MI), D-chiro-inositol (DCI), pinitol, and scyllo-inositol (SI) have been addressed in insulin function and neurotransmission. However, there is controversy among the findings and the more effective inositol derivative remains unclear. Hence, this review aimed to clarify the dosing, mechanisms, and the most effective INS isomer. Methods: To identify the most relevant articles on the effect of INS compounds on AD, a comprehensive search was conducted on online databases, including PubMed, Scopus, and Google Scholar, using both MeSH (Medical Subject Headings) and non-MeSH terms. Moreover, the authors mainly emphasized on the studies conducted in 2025 in order to update previous findings. Results: To the knowledge, accumulating findings have suggested the roles of INS derivatives on preventing IR and vascular dysfunction, improving redox state and inflammatory cascades. These changes subsequently improve amyloid pathology and brain function in AD. More precisely, the action of INS isomers differ. Free MI exerts a potential suppressive role in Aβ aggregation and fibril formation through Aβ-phosphatidylinositol binding sites. Most recent in vitro findings illustrated the ability of MI on inducing carbonic anhydrase aggregation affecting Aβ plaque formation and cerebrovascular dysfunction in AD. DCI only exerted insulin-mimetic charachteristics in the brain and suppressed the development of IR. However, most of the studies in 2025 have focused on pinitol as a APP γ-secretase cleavage inhibitor. In this context, experimental studies (200 mg/kg/day pinitol) documented the ability of pinitol intervention on improving cognitive spatial flexibility and Aβ protein down-regulation. Pinitol

also triggered PI3K /Akt pathway, mediated IR and pro-cognitive hormone levels, and suppressed tau hyperphosphorylation as well as bacterial lipopolysaccharide. In turn, SI- the prominent INS isomer- improved synaptic transmission, inhibited Aβ aggregation and cognitive decline in early-stage AD in animal models. Indeed, higher SI concentrations in the brain were accompanied by reduced amyloid plaque, improved cortical microvasculature integrity. Furthermore findings on clinical trials in 2025, linked the long-term SI supplementation (250 mg for 78 weeks) to modest neuro-protective events, reduced cerebrospinal fluid and Aβ42 levels. Despite the favorable effects of short-term high-dose supplementation of SI (4000 mg/day for 10 days) in healthy subjects, long-term administration of modest doses (1000–2000 mg) were associated with adverse effects, including early deaths. Conclusion Taken together, recent findings document favorable effects of INS derivatives on AD and related outcomes with a specific attention to pinitol and SI in clinical settings. Further well-designed clinical trials are needed to validate the long-term safety and efficacy of INS derivatives.

Keywords : Alzheimer’s Disease; Inositol; Pinitol; Scyllo-inositol; Neuroprotection

Count: 202
Abstract ID: 795
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Integrated lncRNA–miRNA–GLUD1/GLUD2 Network Reveals Molecular Mechanisms in Multiple Sclerosis

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Background and Aim : The events constituting MS happen in an immune-mediated destructive set of activities in the brain and spinal cord, causing inflammatory damage to myelin and axons. The immune system in MS attacks healthy cells at various stages of development; principally, in myelin it targets the sheath wrapping around neuronal axons, affecting nerve conduction and triggering neuroinflammation to the extension of neurological compromise. The aetiological factors of MS have been attributed both genetic and environmental; however, altered coding and non-coding genes have played a much more prominent role. Among non-coding genes, lncRNAs have attracted much interest as key factors controlling gene expression. Evidence supports the role of lncRNAs in regulating neuroinflammatory signaling, neuronal survival, and synaptic function in the initiation of neurodegenerative diseases such as MS.

Methods : The expression of the genes processes in multiple sclerosis was analyzed and treated by microarray datasets using GEO2R.KEGG was used to perform functional enrichment and pathway mapping, and STRING to construct protein–protein interaction association networks. Candidate miRNAs targeting those genes were predicted using TargetScan, miRTarBase, and miRDB, with common targets integrated through Cytoscape. The corresponding lncRNAs were obtained from DIANA-LncBase, and overlapping transcripts were identified to build an lncRNA–miRNA–gene interaction network. Stop-gained mutations within exon regions were identified by Ensembl while their clinical significance was identified through ClinVar and VarSome databases.

Results : The analysis of the GEO dataset GSE21942 revealed both GLUD1 (logFC=-0.961, adj. $p=8.05\times10^{-8}$) and GLUD2 (logFC=-1.18, adj. $p=6.61\times10^{-7}$) to be significantly

downregulated in MS. According to STRING analysis, there were strong interactions between the two, assuming that they hold a key role in disease pathology. Three shared miRNA regulators revealed from the comparisons were: hsa-miR-185-5p, hsa-miR-548an, and hsa-miR-4666a. According to DIANA-LncBase, there are 21 overlapping lncRNAs, among which are NEAT1, MALAT1, XIST, SNHG1, SNHG7, SNHG14, NORAD, and MTCO1P12, thus providing an integrative lncRNA–miRNA network from the perspective of GLUD1 and GLUD2. Within the integrated lncRNA–miRNA network, NEAT1 and MALAT1 functioned as hubs that are co-targeted by miR-15a-5p and miR-26a isoforms. The expression data confirmed the large-scale dysregulation of these targets, implying their role in carbon metabolism and alanine, aspartate, and glutamate metabolism, thus culminating neuronal apoptosis, oxidative stress, and excitotoxicity. Additionally, variant analysis identified stop-gained mutations in both genes, where rs886047375 in GLUD1 was classified as a variant of uncertain significance (VUS) based on clinvar, while rs140532390 in GLUD2 was reported as likely pathogenic according to ClinVar and VUS based on VarSome.

Conclusion : Reported candidate biomarkers in MS were GLUD1 and GLUD2, whose decreased expression is associated with neuronal dysfunction. The 21 lncRNAs led by NEAT1 and MALAT1 constitute a hitherto unexplored lncRNA–miRNA–gene regulatory axis that delineates in its complexity a molecular network lying under neurodegenerative and neuroinflammatory grounds of MS pathology. Therefore, these findings will open a new chapter in MS pathogenesis while hinting at another group of putative therapeutic targets that need further validation through functional experiments and clinical trials.

Keywords : Multiple sclerosis ,Neurodegeneration ,Glutamate metabolism ,GLUD1/GLUD2 pathway ,lncRNA–miRNA–mRNA axis,Molecular biomarkers

Count: 203
Abstract ID: 787
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

An Explainable Machine Learning model for Alzheimer's Disease classification

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Background and Aim : Alzheimer's Disease (AD), in which a gradual decline in cognitive function and memory loss occurs, ranks among the most prevalent neurodegenerative disorders. Precise and early identification of AD is crucial for timely intervention and enhanced patient care. Machine learning (ML) models present a valuable method for detecting individuals who are at risk of AD. Nevertheless, existing studies often focus on the accuracy of ML models, ignoring the essential element of model explainability. For this reason, the use of explainable artificial intelligence methods is recommended, especially in the medical sciences, where experts need to understand the key factors in decision-making.

Methods : In this study, we selected features with the Boruta algorithm and trained different classical ML models on the Kaggle dataset. This dataset contains extensive health information for 2,149 patients. The dataset includes demographic details, lifestyle factors, medical history, clinical measurements, cognitive and functional assessments, symptoms, and a diagnosis of AD.

Results : Among different classical ML models, the Random Forest (RF) classifier achieved a high F1 score of 94.65% for binary classification (distinguishing between Normal Control and AD). To examine model explainability, we employed the SHAP (SHapley Additive exPlanations) method, which measures the contribution of each feature to the model's prediction.

Conclusion : The SHAP method demonstrates the relative contribution of different features to the model’s prediction of disease status. Lower Mini-Mental State Examination (MMSE) scores, Functional Assessment and Activities of Daily Living (ADL) scores, and higher values of Memory Complaints and Behavioral Problems are strongly associated with positive SHAP values, indicating that they drive the model toward predicting disease. Overall, the model

places the greatest weight on cognitive function (MMSE), daily functioning (ADL and functional assessment), and behavioral/memory complaints. This aligns with clinical knowledge, which indicates that cognitive decline, impaired daily activities, and behavioral symptoms are key indicators of disease progression.

Keywords : Alzheimer's Disease Classification; Explainable Artificial Intelligence; , Machine Learning; SHAP.

Count: 204
Abstract ID: 932
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

The Effect of Chitosan-Polyethylenoxid Scaffold with Mesenchymal Stem Cells on the Repair of Damaged Sciatic Nerve in Wistar Rats

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Background and Aim : Peripheral nerve injuries are a clinical problem that can ultimately lead to functional disability. Therefore, it is very important to evaluate the degree of regeneration of this injury. In this paper, the effect of chitosan-Polyethylenoxid (Cs-PEO) scaffolds with Mesenchymal Stem Cells (MSCs) on sciatic nerve regeneration in adult male rats was investigated.

Methods : In this experimental study, 24 male Wistar rats weighing 200-250 g were used. The animals were randomly divided into four experimental groups: control, MSCs, scaffold alone (S), and scaffold with cells (S-MSCs). In all groups, the sciatic nerve was cut in the mid-thigh area and the epineurium was sutured. The control group did not receive any treatment. In the cell culture group, 1000 MSCs were injected onto the injured sciatic nerve. In the scaffold with cells group, the cells were cultured on the scaffold and then applied around the injured sciatic nerve, and finally a thin layer of a cell-free scaffold was applied around the injured sciatic nerve. After eight weeks, the recovery rate was evaluated by behavioral analysis, NCV (nerve conduction velocity), and histological evaluations

Results : At the end of the second and fourth weeks, the mean latency to pain response in the treated groups showed a significant improvement ($p \leq 0.001$). At the eighth week after surgery, the mean latency to pain response in the scaffold group showed a slight improvement ($p=0.08$). Also, the mean SFI (sciatica functional index) in the treated groups showed a relative improvement compared to the control group. However, the differences were not significant ($p<0.4$). The mean nerve conduction velocity in the S-MSCs group showed a significant increase compared to the other groups, and the mean number of nerve fibers in this group increased ($p<0.05$). The mean size of the largest nerve fibers and axons in the MSCs group also did not show a significant increase compared to the control group ($p<0.1$).

Conclusion : The findings of our studies (behavioral, histological, etc.) indicate that the scaffold method with MSCs is a suitable and useful method in the treatment of sciatic nerve in mice and has positive effects on nerve regeneration and helps accelerate healing.

Keywords : Scaffold, Sciatic Nerve Transection, MSCs

Count: 205
Abstract ID: 499
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Lipidopathy as a new hypothesis: Interplay of lipid dysregulation and peripheral amyloid-beta clearance in Alzheimer's disease pathogenesis

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Background and Aim : Alzheimer's disease (AD), affecting nearly 50 million people globally and projected to triple by 2050, is characterized by extracellular amyloid-beta (Aβ) plaques and intracellular hyperphosphorylated tau (hp-Tau) tangles. Aberrant APP processing via beta-secretase (BACE1) generates insoluble Aβ1-42, driving synaptic dysfunction and cognitive decline, while alpha-secretase cleavage produces neuroprotective Aβ. Cholinergic neuron loss in the hippocampus and cortex underlies memory deficits. Emerging evidence highlights the liver's role in AD, producing peripheral Aβ and mediating its clearance via the blood-brain barrier (BBB). Triglyceride-rich lipoproteins (TRLs) transport Aβ, linking lipid metabolism directly to neurovascular and inflammatory pathology—an aspect of peripheral lipidopathy. Receptors such as RAGE facilitate Aβ influx, while LRP1 mediates efflux and peripheral clearance. Age- or disease-related LRP1 reductions exacerbate cerebral Aβ accumulation. Peripheral clearance accounts for roughly 50% of total Aβ removal, emphasizing the interplay between lipid metabolism, liver function, and AD progression. Modulating lipid pathways and TRL-mediated Aβ transport may offer therapeutic potential in AD.

Methods : This systematic study used databases including Web of Science, Scopus, PubMed, and Embase (2014–2024). Guided by the PRISMA statement, it applied defined keywords and excluded case reports, abstracts, posters, conference papers, and systematic reviews from the selection process.

Results : Lipids, particularly cholesterol and its derivatives, are central to Alzheimer's disease (AD) pathophysiology. While brain cholesterol supports synaptic plasticity and neuronal health, excessive cholesterol and oxysterols, such as 24-hydroxycholesterol (24-HC) and 27-hydroxycholesterol (27-HC), disrupt cholesterol homeostasis, promote oxidative stress, hyperphosphorylated tau (hp-Tau), and neuronal apoptosis, and dysregulate amyloid precursor protein (APP) processing, enhancing amyloid-beta (Aβ) accumulation. Dyslipidemia and high-fat diets exacerbate neuroinflammation, neurodegeneration, and Aβ deposition. Peripheral lipid disturbances, including nonalcoholic fatty liver disease (NAFLD), reduce low-density lipoprotein receptor-related protein 1 (LRP1) expression, impairing Aβ clearance across the blood-brain barrier (BBB) and accelerating AD progression. ATP-binding cassette (ABC) transporters—ABCA1, ABCB1, ABCG1, and ABCA7—facilitate Aβ efflux via lipidated apolipoproteins such as ApoE and HDL, promoting plaque clearance. Low HDL or ApoA-I levels correlate with increased Aβ deposition and cognitive decline, whereas elevated HDL enhances peripheral Aβ clearance through the "sink" effect and LRP1-mediated transport. Central clearance involves enzymatic degradation (neprilysin), glial phagocytosis, and cerebrospinal fluid flow. RAGE facilitates Aβ influx, while LRP1 mediates efflux; dysfunction in these pathways exacerbates neurotoxicity. Therapeutic strategies targeting HDL levels, ABC transporter function, and LRP1 activity may restore lipid homeostasis, improve Aβ clearance, and mitigate neurodegeneration, highlighting lipidopathy as a key driver of AD pathology.

Conclusion : Recent advances in blood-based biomarkers enable early, pre-symptomatic Alzheimer's disease (AD) diagnosis and personalized therapy. Dyslipidemia—particularly low HDL and abnormal triglycerides, termed peripheral lipidopathy—contributes to sporadic AD progression by impairing Aβ processing, transport across the blood-brain barrier, and clearance. Elevated serum Aβ from increased brain efflux correlates with cognitive improvement, highlighting HDL's protective role in antioxidant defense and neuroprotection. Lifestyle modifications and lipid-targeted therapies may mitigate AD risk. Monitoring HDL function offers a promising prognostic marker, while multi-target interventions addressing oxidative stress, neuroinflammation, and lipid dysregulation may optimize early-stage treatment outcomes.

Keywords : Alzheimer's disease; Peripheral carrier; Lipidopathy; Molecular clearance

Count: 206
Abstract ID: 646
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

Ranolazine Attenuates 6-OHDA-Induced Neurotoxicity in SH-SY5Y Cells by Targeting Oxidative Stress and Apoptosis

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Background and Aim : Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by oxidative stress- and apoptosis-mediated neuronal loss. Ranolazine, a clinically approved antianginal drug, has recently attracted interest for its potential neuroprotective properties beyond cardiovascular applications. This study investigated the protective effects of ranolazine against 6-hydroxydopamine (6-OHDA)-induced cytotoxicity in SH-SY5Y neuroblastoma cells.

Methods : The cytotoxic profile of ranolazine was first evaluated in SH-SY5Y cells. Subsequently, cells were co-treated with 6-OHDA (150 µM) and varying concentrations of ranolazine (0–25 µM) for 24 hours. Cell viability, oxidative stress, and apoptosis were assessed using the MTT assay, 2′,7′-dichlorodihydrofluorescein diacetate (H_2DCFDA) fluorescence staining, and Annexin V-FITC/propidium iodide (PI) dual staining, respectively. Western blot analysis was performed to measure the expression of pro-apoptotic markers Bax and caspase-3, and the anti-apoptotic protein Bcl-2.

Results : Ranolazine up to 25 µM exhibited no significant cytotoxicity in SH-SY5Y cells ($p > 0.05$). Exposure to 6-OHDA markedly reduced cell viability, while co-treatment with ranolazine (10 and 25 µM) significantly improved cell survival ($p < 0.01$) and decreased reactive oxygen species (ROS) generation ($p < 0.001$). Moreover, ranolazine (10 µM) significantly reduced apoptotic cell percentages ($p < 0.001$), suppressed Bax and caspase-3 expression ($p < 0.05$), and enhanced Bcl-2 levels compared to the 6-OHDA group.

Conclusion : These findings suggest that ranolazine exerts neuroprotective effects against 6-OHDA-induced toxicity in SH-SY5Y cells by attenuating oxidative stress and apoptosis. Ranolazine may thus represent a promising candidate for further investigation in Parkinson's disease models.

Keywords : Ranolazine; Parkinson's disease; 6-Hydroxydopamine; Oxidative stress; Apoptosis

Count: 207
Abstract ID: 325
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

The neuroprotective effect of elderberry diet on the tramadol-induced toxicity in the hippocampus of adult male rats

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Background and Aim : Pain relievers such as tramadol are among the most commonly prescribed around the world. However, Long-term tramadol use has been shown to cause neurotoxicity, neuroinflammation, and cognitive impairment, which has limited its clinical use. Elderberry (EB), a plant extract widely recognized for its medicinal properties, is used in this study to investigate the effects of a plant-based diet on tramadol's adverse effects in the hippocampus region.

Methods : The effects of EB oral diet on the devastating effects of tramadol on the hippocampus structure and function of 36 male albino rats were evaluated using behavioral, electrophysiological, and histological setups. Three groups (Control, Tramadol, and Tramadol+EB) were examined using behavioral, electrophysiological, and histological setups.

Results : The behavioral findings showed that the EB diet could improve most of the memory-related indices in Tramadol+EB compared to the Tramadol group. Regarding the fEPSP slope and population spike amplitude, these electrophysiological parameters returned to control levels in the Tramadol+EB group. Additionally, a decrease in caspase-3 expression was observed in hippocampal cells. While there was a reduction in astrogliosis in the Tramadol+EB group compared to the Tramadol group, no significant differences were noted in the morphological variations of astrocytes between the groups.

Conclusion : In conclusion, although further studies are necessary to elucidate other aspects of EB's effects on the brain, it may be considered a potential neuroprotective agent in acute to sub-chronic Tramadol exposure.

Keywords : Tramadol, Opioid, Memory, Hippocampus, Neuroprotection, Field Potential Recording

Count: 208
Abstract ID: 541
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

The therapeutic effects of the olfactory ectomesenchymal stem cells-derived exosomes on the neurogenesis and synaptogenesis in the Alzheimer's disease rat model

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Background and Aim : Alzheimer's disease (AD), a progressive neurodegenerative disorder, is one of the leading causes of death globally. In recent years, mesenchymal stem cell (MSC)-based therapy has emerged as a beneficial method for the treatment of neurological disorders. Olfactory ectomesenchymal stem cells (OE-MSCs) are a new type of resident stem cell in the olfactory lamina propria. Several studies have reported that OE-MSCs transplantation can induce neurogenesis and restore hippocampal neuronal networks. The availability of olfactory mucosa in adult humans and the possibility of using one's own cells in the treatment of diseases are the most considerable advantages of this kind of stem cell. Recent studies showed that stem cell transplantation produces short-term therapeutic effects in many disease models. In addition, it increases the risk of developing tumors. MSCs exert their regenerative effects mostly through their paracrine secreted factors. Exosomes secreted from MSCs contain these paracrine factors. Therefore, MSCs-derived exosomes can be a promising therapeutic approach for AD treatment.

Methods : At first, OE-MSCs were isolated from the rat's olfactory mucosa. After cell characterization, exosomes were isolated through ultracentrifugation as the gold standard exosome isolation method from the OE-MSC conditioned media. The characterization of the isolated exosomes was carried out. Then, exosomes were administered intranasally every other

day for two weeks in the AD rat model (induced by amyloid β25-35). Behavioral evaluations (Passive avoidance response test and Morris water maze (MWM) test) were performed. Histological evaluation of the hippocampal formation was done by Cresyl violet staining. Structural assessment of synaptogenesis was carried out using transmission electron microscopy (TEM). The expression level of the genes related to the neurogenesis process, including Nestin, DCX, NeuN, β III tubulin, and MAP2, was measured using qRT-PCR. Finally, the expression level of MAP2 protein was measured via immunohistochemistry (IHC) analysis.

Results : As behavioral analysis revealed, exosomes derived from OE-MSCs can ameliorate memory and cognition in the exosome-treated group. The Nissl-stained images of the CA1 and dentate gyrus showed that the number of dark cells was significantly lower in the exosome-treated group compared to the AD group. Electron micrographs displayed an improvement in the structure of synapses after exosomal treatment. Results from qRT-PCR analysis showed that the expression level of the neurogenesis-related genes, including Nestin, DCX, NeuN, ? III tubulin, and MAP2, was considerably higher in the exosome-treated group compared to the AD group. IHC analysis also indicated a significant increase in the expression of MAP2 protein in the exosome-treated animals in comparison to AD animals.

Conclusion : This study showed that exosomes derived from OE-MSCs have the potential to improve cognitive impairments in AD rats through increasing the neurogenesis and synaptogenesis in the hippocampal formation.

Keywords : Alzheimer's disease, Olfactory ectomesenchymal stem cells, Exosomes, Neurogenesis, Synaptogenesis

Count: 209
Abstract ID: 568
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Activation of mediodorsal thalamic nicotinic receptors improved memory impairment and emotional behaviours in a lipopolysaccharide-induced Alzheimer's mouse model

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Background and Aim : Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive disabilities and behavioural changes. Lipopolysaccharide (LPS), a component of gram-negative bacterial membranes and a potent endotoxin resistant to enzymatic degradation, induces persistent neuroinflammation by releasing proinflammatory cytokines. Evidence includes elevated blood/brain LPS levels, its co-localization with amyloid plaques, neurons, and oligodendrocytes, and its association with myelin damage in AD patients. Considering that the mediodorsal thalamic nucleus (MD), with high cholinergic activity, mediates memory formation through its interactions with the hippocampus and the prefrontal cortex (PFC), the present study aimed to investigate the role of nicotinic acetylcholine receptors (nAchRs) in LPS-induced memory impairment as an animal model of AD

Methods : Male NMRI mice (20-30 g) were used to unilaterally cannulate in the lateral ventricle (LV) and the MD by a stereotaxic surgery. The animals were then randomly assigned to four groups of control, LPS, nicotine, and nicotine plus LPS. LPS (5 µg/mouse) was intracerebroventricularly microinjected (intra-LV), and nicotine (1 µg/mouse) was injected into the MD (intra-MD) in post-training and pre-test phases. A step-down type passive avoidance learning task was used to assess memory formation/impairment. Locomotor activity, emotional and exploratory behaviours were evaluated using the elevated plus maze (EPM) and the open field test (OFT) in an experimental procedure under the administration of the drugs

Results : Our findings indicated that intra-LV microinjection of LPS decreased step-down latency in the passive avoidance learning task to induce amnesia. Intra-MD microinjection of

nicotine reversed memory impairment induced by LPS. In the LPS model, the animals exhibited alterations in exploratory behaviours, including climbing. Interestingly, intra-MD microinjection of nicotine significantly improved these behavioural changes. Our results also showed that intra-LV microinjection of LPS decreased the percentages of open arm time and entry in the EPM, indicating LPS-induced anxiogenic-like behaviours, while intra-MD microinjection of nicotine improved LPS response

Conclusion : The findings indicate that LV-LPS microinjection impaired learning and memory processes, suggesting an amnesic effect of LPS on memory formation. Since the MD activation of nAchRs could improve LPS-induced amnesia, and alleviate anxiety and other behavioral disturbances, it can be concluded the thalamic cholinergic nicotinic system may be involved in LPS-induced cognitive disabilities. It seems that the nicotinic receptor agonists may be potential therapeutic targets against cognitive decline in AD

Keywords : Lipopolysaccharide; Memory impairment; MD nicotinic Receptors; AD animal model

Count: 210
Abstract ID: 573
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

The Role of Predictive Models in Alzheimer's Disease Using Hippocampal Molecular Biomarkers

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Conclusion : Introduction Alzheimer's disease (AD) is a progressive neurodegenerative disorder, the leading cause of dementia, significantly impacting global health. It mainly affects the hippocampus, vital for memory, and is characterized by amyloid-β plaques and tau tangles. Early prediction of AD is crucial, as interventions during the Mild Cognitive Impairment (MCI) stage can delay cognitive decline. This review explores predictive models using hippocampal and molecular biomarkers to forecast AD onset and progression. Materials and Methods This review examines the development of predictive models for Alzheimer's Disease (AD), with a primary focus on major public datasets, such as the Alzheimer's Disease Neuroimaging Initiative (ADNI). The field has evolved from relying on simple measures, like hippocampal volume, to employing complex machine learning and deep learning algorithms. These advanced models are designed to integrate diverse data types, including structural MRI scans, various biomarkers, and clinical and genetic information. Results Predictive models for Alzheimer’s disease that integrate hippocampal characteristics and molecular data have shown high effectiveness. One deep learning model, utilizing hippocampal MRI scans, predicted the progression from mild cognitive impairment (MCI) to Alzheimer's disease (AD) with a concordance index of 0.762. In contrast, another model, focusing on mesoscopic features, achieved an accuracy rate of 98%. Molecular biomarkers, such as the synaptic proteins neurogranin and SNAP-25, in cerebrospinal fluid (CSF) correlate with clinical progression and hippocampal atrophy in AD and MCI patients. A 2024 review reported an average area under the curve (AUC) of 0.87 for the best predictive models, emphasizing that combining imaging, molecular, and clinical data produces superior results compared to any single approach. Conclusion Predictive models combining hippocampal imaging and molecular biomarkers provide a non-invasive method for early diagnosis and prognosis of Alzheimer's disease. Advanced machine learning with multi-modal data can accurately classify disease stages and predict progression, enabling earlier interventions and personalized treatment. Future efforts should focus on validating these models and linking hippocampal changes to specific molecular pathways to enhance biological understanding.

Keywords : Alzheimer's disease, Mild Cognitive Impairment, Molecular Biomarkers, Predictive Models

Count: 211
Abstract ID: 247
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Silibinin ameliorated behavioral and molecular deficits in a rat model of Parkinson disease

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Background and Aim : Apoptosis, oxidative stress, and neuroinflammation have been linked to the onset of Parkinson's disease (PD). Although the pre-treatment effects of Silibinin on a PD model have been evaluated, in the current study we investigated the chronic therapeutic effects of Silibinin microinjection on a rat model of established parkinsonism along with behavioral and laboratory markers assessments.

Methods : Parkinsonism was induced by 6-hydroxydopamine (6-OHDA, 8 µg/2µl/rat). 21 days after that, animals were treated with Silibinin (100, 200, and 300 mg/kg for 15 consecutive days). Every two days, the bar test was used to evaluate Silibinin's anti-cataleptic properties. At the end, myeloperoxidase (MPO) activity and toll-like receptor 4 (TLR4) expression in the substantia nigra pars compacta (SNc), along with cerebrospinal fluid (CSF) levels of tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1β, IL-6, caspase-3, Bax and Bcl-2 levels were assessed.

Results : Silibinin's Chronic treatment, dose-dependently decreased catalepsy. MPO activity and levels of TNF-α, IL-6, and IL-1β were reduced in Silibinin-treated rats in all three doses. Silibinin decreased Bax/Bcl-2 ratio, caspase-3, and downregulated TLR4 expression.

Conclusion : In accordance with alleviation of TLR4 activity with a consequent reduction in oxidative stress, restoration of CSF inflammatory cytokine, and minimization of SNc neuronal apoptosis, long-term therapy with Silibinin offers a potential opportunity for symptomatic PD treatment.

Keywords : Silibinin; Parkinson's disease; 6-Hydroxydopamine (6-OHDA).

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Abstract ID: 886
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

The potential utility of arterial spin labeling in predicting brain amyloidosis

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Background and Aim : Declines in regional cerebral blood flow (rCBF) are common in Alzheimer's disease. Previous studies have linked higher amyloid beta-protein (Aβ) loads, detected by Positron Emission Tomography (PET), with rCBF in normally aging individuals. This study aims to assess the potential of Arterial Spin Labeling (ASL) in predicting brain amyloidosis.

Methods : The study included 140 participants from the Alzheimer's Disease Neuroimaging Initiative (ADNI), comprising 43 cognitively normal (CN) individuals, 70 patients with mild cognitive impairment (MCI), and 27 with Alzheimer's disease (AD). Eligible participants had comprehensive assessments of cognition, ASL, Apolipoprotein E4 (APOE4) genotyping, Polygenic Hazard Score (PHS) calculation, and amyloidosis indices. Data were analyzed using IBM SPSS ver. 20, employing chi-square, ANOVA, and linear regression models, with a significance threshold of $P < 0.05$.

Results : There were no statistically significant differences in demographic characteristics, including age, gender, and education level. Significant differences emerged in amyloid beta-protein 42 (Aβ42) levels, APOE4 status, and cognitive performance across groups. Four brain regions, including the left middle temporal, bilateral para-hippocampal, and right lingual cortex exhibited significant CBF differences ($p < 0.05$). Clinical Dementia Rating (CDR) correlated strongly with various brain regions, particularly in MCI individuals. Moreover, CBF in multiple regions showed significant associations with Aβ variants and their ratios, especially Aβ42, even after adjusting for confounding factors.

Conclusion : ASL demonstrates potential in predicting brain amyloidosis and monitoring AD. Significant associations between CBF and amyloid-beta levels suggest that ASL can detect early perfusion deficits related to AD progression.

Keywords : Alzheimer's disease, brain amyloidosis, ASL, Arterial Spin Labeling

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Reversible CRISPRoff/on and Lipid Nanoparticle-Mediated Epigenetic Editing of COMT for Precision Neurotherapeutics

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Background and Aim : Dopamine balance is critical for brain function, and its disruption contributes to neuropsychiatric and neurodegenerative diseases. The enzyme COMT breaks down dopamine, and its activity varies depending on the rs4680 polymorphism. Traditional COMT inhibitors like tolcapone affect the entire brain and cause systemic side effects due to lack of specificity. Epigenetic editing using CRISPR/dCas9 fused to DNMT3A or TET1 allows reversible, targeted control of COMT expression, offering a new precision medicine approach tailored to patient genotype and affected brain regions.

Methods : The researchers used iPSCs derived from individuals with three rs4680 genotypes: Val/Val, Val/Met, and Met/Met. These iPSCs were differentiated into dopaminergic neurons relevant for Parkinson's and cortical neurons relevant for schizophrenia. Cell identity was confirmed by immunostaining markers specific to each neuronal type. CRISPR/dCas9 tools included fusions with DNMT3A to increase methylation (thus suppressing COMT) and TET1 to reduce methylation (activating COMT). A CRISPR activation (CRISPRa) system with VP64-p65-Rta was also tested. These constructs were delivered to neurons through lentiviral vectors or nucleofection, with successful delivery tracked by GFP expression. Methylation changes at the COMT promoter were measured by MSP and bisulfite sequencing. COMT gene expression was assessed via qRT-PCR and RNA sequencing. Dopamine levels and metabolites were quantified using HPLC and ELISA. Electrophysiological functions were evaluated with patch-clamp recordings to measure synaptic activity. In vivo, AAV9 vectors carrying these constructs were stereotaxically injected into the substantia nigra of Parkinson's model mice and prefrontal cortex of schizophrenia model mice with DISC1 mutations. Behavioral tests examined motor coordination, anxiety, cognition, and post-mortem analyses validated molecular effects.

Results : Preliminary data suggest successful COMT modulation in vitro. In Parkinson's patient-derived dopaminergic neurons with excessive COMT activity, dCas9-DNMT3A induced promoter methylation, lowered COMT expression, increased dopamine retention, improved synaptic function, and reduced oxidative stress. Conversely, in cortical neurons from schizophrenia patients with Val/Val genotype, dCas9-TET1 demethylated the COMT promoter, restoring balanced COMT expression and potentially improving prefrontal dopamine signaling and cognition. Rodent Parkinson's models treated with COMT-suppressing constructs showed enhanced motor performance, fewer postural issues, and normalized dopamine levels. Schizophrenia models receiving CRISPRa or TET1 constructs exhibited reduced anxiety-like behavior, increased exploratory activity, and improved working memory. These results support the concept that epigenetic fine-tuning of COMT can restore dopamine balance in a disease- and region-specific manner. The reversible nature and precision of CRISPR/dCas9 epigenetic editing offer an advantage over permanent genome editing or non-specific pharmacological inhibitors.

Conclusion : This study introduces a novel, genotype- and region-specific CRISPR-based epigenetic strategy to modulate COMT expression non-permanently. This approach holds promise for individualized therapies targeting dopamine-related brain disorders like schizophrenia and Parkinson's disease. Future work will focus on integrating multi-omic analyses to improve patient stratification and therapeutic precision.

Keywords : COMT, CRISPRoff, epigenetic editing, lipid nanoparticles, Neurotherapeutics

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Intranasal Delivery of Human Olfactory Ecto-Mesenchymal Stem Cells Enhances Cognitive Function in an Alzheimer's Rat Model

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Background and Aim : Mesenchymal stem cell therapy offers promising avenues for treating neurodegenerative disorders such as Alzheimer's disease (AD). This study evaluated the therapeutic potential of human olfactory ecto-mesenchymal stem cells (OE-MSCs) administered intranasally in a rat model of AD induced by intrahippocampal injection of amyloid-beta (Aβ).

Methods : OE-MSCs were isolated from the human olfactory lamina propria and characterized using immunocytochemistry and flow cytometry. Following transplantation via intranasal or intrahippocampal routes, behavioral, histological, and molecular analyses were conducted after a three-month recovery period.

Results : Intranasal administration of OE-MSCs significantly reduced Aβ deposition and neuronal loss, improved spatial learning and memory, and elevated levels of brain-derived neurotrophic factor (BDNF) and NMDA receptors. These effects were more pronounced in the intranasal group.

Conclusion : OE-MSCs may enhance cognitive function in Alzheimer's disease through neuroprotective and neuroregenerative mechanisms, particularly when delivered via the intranasal route.

Keywords : Alzheimer's disease, Olfactory ecto-mesenchymal stem cells (OE-MSCs), Intranasal administration, Cognitive function, Amyloid-beta (Aβ), Neurodegenerations, Brain-derived neurotrophic factor (BDNF)

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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

Longitudinal ADAS-Cog 13 trajectories predict autopsy-confirmed Alzheimer's disease neuropathologic burden

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Background and Aim : The Alzheimer's Disease Assessment Scale–Cognitive Subscale (ADAS-Cog 13) is a validated measure of global cognition widely used in Alzheimer's disease (AD) research and clinical trials. Although neuroimaging and biomarker-based modalities have advanced disease staging, the predictive value of longitudinal cognitive trajectories for underlying neuropathologic burden remains unexplored. This study evaluated whether ADAS-Cog 13 trajectories predict Alzheimer's disease neuropathologic change (ADNC) in autopsy-confirmed participants from the Alzheimer's Disease Neuroimaging Initiative (ADNI).

Methods : Participants with available longitudinal ADAS-Cog 13 assessments and post-mortem neuropathologic data were included. ADNC classification followed the National Institute on Aging–Alzheimer's Association (NIA-AA) criteria, integrating Thal amyloid phase, Braak neurofibrillary tangle stage, and CERAD neuritic plaque score, dichotomized as high or low burden. Longitudinal ADAS-Cog 13 data were modeled using linear mixed-effects models with random intercepts and slopes to estimate baseline cognitive performance and individual rates of change. Logistic regression models examined associations between these parameters and ADNC, with high ADNC as the primary outcome. Model discrimination, calibration, and predictive accuracy were evaluated using area under the receiver operating characteristic curve (AUC) and related indices.

Results : A total of 121 participants with 768 longitudinal observations were analyzed. The median follow-up was 5 sessions (mean 6.3) over a maximum of 162 months prior to death. Individuals with high ADNC demonstrated steeper annual worsening in ADAS-Cog 13 scores (5.47 ± 3.02) and greater baseline impairment (24.30 ± 9.03) than those with low ADNC (1.88 ± 2.64 and 14.51 ± 8.48, respectively). Faster cognitive decline (OR = 3.18; 95% CI: 2.12–6.93; $p < 0.001$) and higher baseline impairment (OR = 1.15; 95% CI: 1.03–1.33; $p = 0.011$)

were independently associated with high ADNC. The combined predictive model achieved excellent discrimination (AUC = 0.875) with sensitivity of 84.4% and specificity of 81.8% under Youden index optimization.

Conclusion : Longitudinal ADAS-Cog 13 trajectories, particularly the rate of decline, are strongly associated with the neuropathologic burden of AD. Beyond its statistical performance, the ADAS-Cog 13 reflects meaningful clinical patterns: our findings indicate that cognitive decline in AD follows a distinct trajectory compared with other dementias and cognitively normal individuals. Patients with pathologically confirmed AD exhibit higher absolute scores and a steeper rate of worsening over time, underscoring that the clinical presentation of AD progression is qualitatively different from other causes of cognitive impairment. These results affirm the value of ADAS-Cog 13 as a simple yet powerful tool that bridges clinical observation and neuropathologic validation. Its longitudinal trajectories may complement biomarker-based approaches for early diagnosis, disease monitoring, and risk stratification in both research and clinical practice.

Keywords : Alzheimer's disease; ADAS-Cog 13; Cognitive trajectories; Neuropathologic burden; Longitudinal analysis; Neurodegenerative disorders

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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

Aging and Alzheimer's: Understanding the Neurodegenerative Link

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Conclusion : Aging has been associated with increased acetylation of α-secretase, which can result in elevated production of Aβ 40-42, the principal contributor to the pathogenesis of Alzheimer's disease. Aβ 40–42 accumulation in the brain and cerebral blood vessels induces mitochondrial dysfunction and oxidative stress (directly), disrupts calcium homeostasis, and triggers microglial activation (indirectly), which in turn results in the release of pro-inflammatory cytokines such as IL-1β, IL-6, TNFα, and IL-8, leading to neuronal death and neurotoxicity. On the other hand, the aging process can lead to insulin resistance, which in turn is closely related to the development of Alzheimer's disease through the activation of the insulin/IGF-1 signaling pathway. Concurrently, impaired insulin signaling attenuates PI3K/Akt pathway activity and enhances GSK3β-mediated tau hyperphosphorylation, facilitating neurofibrillary tangle (NFT) formation and neuronal apoptosis. Therefore, caloric restriction, which involves reducing.daily caloric intake, has a profound impact on insulin sensitivity and Alzheimer's disease risk. The permanent cell cycle arrest characteristic of cellular senescence is a crucial pathological feature observed in senescent neurons, astrocytes, and microglia that drives chronic neuroinflammation and neural damage. The resulting chronic inflammation in AD patients is closely linked to aging dysregulation and fuels a vicious feedback loop that accelerates neural damage. The resulting cognitive impairment and memory loss are supported by structural evidence showing that hippocampal atrophy occurs on a quantitative continuum spanning healthy aging, Mild Cognitive Impairment and AD, thereby emphasizing the vulnerability of the hippocampus during the transition from aging to AD. Understanding these shared mechanisms allows for the development of integrated therapeutic strategies. In conclusion, we suggest that focusing on ageing mechanisms — alongside interventions specific to Alzheimer's Disease — could provide a hopeful pathway to postpone or mitigate the onset and progression of dementia by maintaining neural plasticity and cognitive reserve.

Keywords : Aging; Alzheimer's Disease; Oxidative Stress; Neuroinflammation

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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Bioinformatics Identification of Microglial Candidate Genes and Therapeutic Targets in Alzheimer's Disease Using APPswe/PS1dE9 Mouse Model

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Background and Aim : Alzheimer's disease (AD) is a progressive, incurable neurodegenerative disorder characterized by the accumulation of amyloid-beta (Aβ) aggregates and hyperphosphorylated tau protein. Emerging evidence highlights the critical role of microglia in AD pathology, particularly through the activation of disease-associated microglia (DAMs), which secrete pro-inflammatory cytokines in response to amyloid deposition. This study aims to employ bioinformatics to identify candidate genes in hippocampal microglia from the APPswe/PS1dE9 mouse model of AD, with a specific emphasis on early amyloid-induced alterations in cytokine expression. By examining both upregulated and downregulated gene sets, this research seeks to uncover potential therapeutic targets capable of reversing AD-associated expression patterns and modulating cytokine-driven neuroinflammatory pathways.

Methods : RNA-seq data were obtained from the GEO database, and subsequently, principal component analysis (PCA) was performed to visualize the overall transcriptional differences between control and AD microglia. Following this, differential gene expression analysis (DGEA) was conducted to identify significantly upregulated and downregulated genes. To further explore functional implications, gene set enrichment analysis (GSEA), was employed to assess microglial changes associated with these genes. For systematic evaluation, the genes were grouped into six sets (based on pathway enrichment analysis), and GSEA was applied to determine their roles in disease progression and potential therapeutic responses. In addition, heatmaps were generated to visualize the expression patterns of candidate AD risk genes identified through multiple genomic analyses. Building on these findings, upregulated and downregulated gene sets were then utilized to predict drug candidates which might reverse the gene expression profile associated with AD. Finally, for each candidate, FDA approval status, cytotoxicity, and blood-brain barrier (BBB) permeability were examined.

Results : Three key upregulated genes were identified: Cst7, Clec7a, and Adamts6. These genes are consistently associated with microglial activation states characteristic of disease-associated microglia (DAM), and have various functions investigated with GSEA and CFDE Enrichment Analysis. Cst7 is associated with phagocytosis and lipid metabolism and shows a strong correlation with the expression of inflammatory cytokines. Clec7a regulates M2 macrophage function and encodes Dectin-1, a receptor essential for mediating inflammatory responses. Adamts6 acts as a multifunctional modulator of extracellular matrix (ECM) dynamics across several tissues. Top drugs which inhibited expression of the genes which were upregulated in the mouse Alzheimer's microglia were identified using LINCS L1000 Chem Pert down library: Donepezil hydrochloride and Mestinon. Interestingly, these drugs are inhibitors of acetylcholinesterase, as is Aricept, the standard of care treatment for Alzheimer's, suggesting that protective effects of Aricept may be mediated at least in part by reducing inflammation.

Conclusion : This study identified candidate genes associated with Alzheimer's disease and potential small molecules to reverse expression of those genes. Characterizing these molecular alterations enhances our understanding of AD pathophysiology and idiopathic neurodegeneration. These findings provide valuable insights for future clinical investigations aimed at improving prognosis and advancing therapeutic strategies to enhance treatment outcomes.

Keywords : Alzheimer's disease; Microglia; Amyloid-beta; Cytokine

Count: 218
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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Investigation of the interaction between Donepezil with mastic gum on spital and avoidance memory in adult male Syrian mice

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Background and Aim : Memory impairment represents one of the most significant clinical challenges in neurodegenerative diseases, including Alzheimer's. Donepezil, as an acetylcholinesterase inhibitor, is a common treatment for improving cognitive symptoms in patients with Alzheimer's. On the other hand, numerous natural compounds have garnered attention due to their antioxidant and neuroprotective properties. Mastic gum (Pistacia lentiscus L.) is one such natural product for which evidence of protective effects on neural function has been reported. Given the importance of investigating the potential synergy or interaction between pharmaceutical drugs and herbal products, this research aimed to evaluate the interaction of donepezil and mastic gum on avoidance memory in adult male Syrian mice.
Methods : In this experimental study, 54 male mice were kept in an animal room with controlled temperature and humidity, and free access to food and water. The preferred route of administration for donepezil was intraperitoneal (IP) injection, while the preferred method for mastic gum was oral gavage. The groups included a control group and test groups with different effective and ineffective doses of donepezil and mastic gum. Three test groups were also assigned to investigate the interaction effect of the two at ineffective concentrations. The mice were treated for 14 days, and then on the 14th day, memory impairment was induced by scopolamine, and memory tests, including the Y-maze and shuttle box tests, were performed, and the results were recorded and reported. The data were analyzed by one-way analysis of variance, non-parametric tests, and isobargram analysis.
Results : In the Y-maze test, one-way ANOVA revealed that the treatments significantly increased the time spent in the novel arm. However, the number of entries into the novel arm and locomotor activity did not show any significant differences. In the inhibitory avoidance test (shuttle box), the step-down latency was significantly increased. Isobolographic analysis indicated that the effect of the combination of donepezil and mastic gum was synergistic.
Conclusion : The findings demonstrate that both donepezil and mastic gum, individually and in combination, can enhance spatial and inhibitory avoidance memory performance. The proposed combination exhibits a synergistic effect and could be considered a potential complementary option in cognitive therapeutic approaches.
Keywords : Mastic Gum, Donepezil, Memory, Mouse

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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

The Neuroprotective Effect of Psilocybin on Behavioral Changes in Larval Zebrafish induced by Aluminum Chloride

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Background and Aim : Alzheimer's disease (AD) is a progressive neurodegenerative disorder primarily affecting elderly individuals and is characterized by cognitive decline, especially in learning and memory. The global prevalence of AD exceeds 50 million patients, posing significant health and socioeconomic burdens. Current therapeutic approaches provide only symptomatic relief, and there is a pressing need for novel agents with neuroprotective potential. Psilocybin (PSY), a serotonergic psychedelic compound, has gained attention for its potential neuroplastic and cognitive-enhancing effects. The present study aimed to investigate the protective effects of PSY on aluminum chloride ($AlCl_3$)-induced cognitive impairments in zebrafish (ZF) larvae, an established and translationally relevant model for AD-related pathology.

Methods : Four-day post-fertilization ZF larvae were exposed to 200 µM $AlCl_3$ to induce AD-like deficits in cognition and anxiety-related behavior. Following induction, larvae were treated with varying concentrations of PSY (0.1, 0.3, and 1 µM). Behavioral outcomes were assessed using three validated paradigms: thigmotaxis (anxiety-like behavior), light/dark preference (anxiety and exploratory behavior), and habituation to repeated acoustic/vibrational stimuli (learning and short-term memory). Data analysis was performed using appropriate statistical tests, and significance was set at $p < 0.05$.

Results : Exposure to 200 µM $AlCl_3$ significantly impaired behavioral performance across all assays, reflecting increased anxiety-like behavior, reduced exploratory activity, and deficits in learning and memory. PSY treatment ameliorated these impairments in a concentration-dependent manner, with the most pronounced effects observed at 0.3 µM. Specifically, PSY-treated larvae demonstrated reduced thigmotaxis behavior, restored light/dark preference patterns toward control levels, and improved habituation responses, indicating recovery of cognitive flexibility and memory formation. Higher PSY concentrations did not further improve outcomes, suggesting a potential inverted U-shaped dose–response profile. The neuroprotective action of PSY was consistent across behavioral endpoints and suggests modulation of neural circuits affected by $AlCl_3$ neurotoxicity.

Conclusion : The findings indicate that PSY, particularly at low dosages, exerts significant neuroprotective and cognition-restorative effects in an $AlCl_3$-induced zebrafish model of AD.

This protective profile includes mitigation of anxiety-like behavior, restoration of exploratory activity, and improvement in learning and memory function. These results support the potential of PSY as a candidate for AD therapy, although optimal dosage ranges and mechanistic pathways require further elucidation. Future studies should focus on molecular targets, long-term outcomes, and translational studies in higher vertebrate models to assess feasibility for clinical application.

Keywords : Psilocybin, Zebrafish, Alzheimer's disease, Aluminum chloride, Neurotoxicity, Cognitive impairment

Count: 220
Abstract ID: 118
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Neuroprotective Effect of Angelica archangelica Extract on Demyelination in a Rat Model of Multiple Sclerosis

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Background and Aim : This study aimed to evaluate the neuroprotective effects of A. archangelica ethanolic extract on ethidium bromide (EB)-induced demyelination in rats. Multiple sclerosis (MS) is a chronic neurodegenerative disorder characterized by autoimmune-mediated demyelination in the central nervous system. Oxidative stress and inflammatory processes are central to disease progression. Angelica archangelica, a medicinal plant with potent antioxidant and anti-inflammatory properties, has been suggested as a potential neuroprotective agent

Methods : Twenty-eight male Wistar rats were divided into four groups: (1) control, (2) EB-induced injury, (3) sham (A. archangelica only, 100 mg/kg), and (4) experimental (EB + A. archangelica). Behavioral assessments (elevated plus maze, open field test, forced swimming test, footprint test, and modified neurological severity score) were performed weekly. After 30 days, brain tissues were subjected to H&E, Luxol fast blue–cresyl violet (LFB–CV), and GFAP immunohistochemistry. Data were analyzed using one-way ANOVA followed by Tukey's test.

Results : EB injection induced significant motor dysfunction, anxiety, and depression-like behaviors compared with controls ($p < 0.01$). A. archangelica treatment improved open field locomotor activity, reduced immobility time in the forced swimming test, and normalized gait patterns in the footprint test. mNSS scores were significantly lower in the EB + A. archangelica group compared with the EB group ($p < 0.05$). Histological analysis revealed reduced inflammatory cell infiltration, preservation of myelinated fibers, and decreased GFAP immunoreactivity in the experimental group, indicating attenuation of astrocytic activation.

Conclusion : Angelica archangelica extract demonstrated significant neuroprotective effects in a rat model of MS, likely mediated by its antioxidant and anti-inflammatory properties. These findings suggest that A. archangelica may serve as a promising complementary therapy to reduce demyelination and neurological deficits in MS.

Keywords : Multiple Sclerosis ,Demyelination, Neuroprotection, Angelica Archangelica , Oxidative Stress

Count: 221
Abstract ID: 60
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Case Report of Mobius Syndrome in a 14-Year-Old Boy

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Background and Aim : Mobius syndrome is a rare, non-progressive neurological disorder first described in 1888 by Paul Julius Mobius. It is characterized by congenital facial paralysis, often bilateral and asymmetric, accompanied by restricted abduction in one or both eyes. The estimated incidence is 0.27 per 100,000 live births, with equal occurrence in males and females. The inheritance pattern is usually autosomal dominant. Pathologically, it can be classified into four types: hypoplasia or atrophy of cranial nerve nuclei, primary lesions of peripheral cranial nerves, focal necrosis of brainstem nuclei, and primary myopathy without central or cranial nervous system lesions. This report presents a case of a 14-year-old boy diagnosed with Mobius syndrome at four months of age
Methods : The patient, a 14-year-old boy and the third child of non-consanguineous parents, was born at term via uncomplicated vaginal delivery with a birth weight of 2850 g. The pregnancy was uneventful, with no history of drug use, exposure to thalidomide or misoprostol, abortion attempts, trauma, or rubella infection. At birth, he exhibited poor sucking ability, which prevented breastfeeding and led to hospitalization at Sarem Hospital, where a diagnosis of Möbius syndrome was confirmed at four months of age. Clinical examination revealed normal facial muscle tone and bulk but complete immobility, producing a mask-like appearance, with no swelling or unilateral facial microsomia; sialorrhea was also present. Ophthalmologic assessment showed severe limitation in both abduction and adduction, while vertical eye movements remained normal, and strabismus was observed
Results : Throughout childhood, facial paralysis persisted, preventing facial expressions such as smiling or frowning. Feeding difficulties were limited to infancy, and no limb or tongue deformities were reported. Neurological status remained stable, consistent with the non-progressive course of Mobius syndrome.
Conclusion : Mobius syndrome is a rare congenital condition of multifactorial origin involving both genetic and environmental factors. There is no definitive cure; management is supportive and aims to restore facial movement, most effectively through reconstructive plastic surgery with muscle grafting at a young age. Psychosocial challenges, such as low self-esteem and speech difficulties, are common due to the mask-like facial appearance. Early diagnosis and multidisciplinary intervention are essential for optimizing functional and psychosocial outcomes.
Keywords : Mobius syndrome; Congenital facial nerve palsy; Strabismus

Count: 222
Abstract ID: 442
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

Effect of gut microbiome on brain disorders: New horizons in the treatment of brain disorders

Submission Author: Sasan Gazerani

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Conclusion : Abstract The ‘gut-brain’ or ‘brain-gut axis’, depending on whether we emphasize bottom-up or top- bottom pathways, is a bi-directional communication system, comprised of neural pathways. This network is not only 1) anatomical, but it extends to include 2) endocrine, 3) humoral, 4) metabolic, and 5) immune routes of communication as well. Emerging evidence indicates that the gut microbiota play a crucial role in the bidirectional communication between the gut and the brain. Recent evidence, mainly arising from animal models, supports a role of microbes as signaling components in the gut-brain axis. Emerging evidence suggests that the gut microbiome influences cognitive function, depression, anxiety, ASD, schizophrenia, epilepsy, Parkinson’s disease, Alzheimer’s disease and also chronic pain via the gut-brain axis. Substantial evidence has linked consumption of a Western Diet (WD), defined here as diets consisting of both high levels of fat (35–60% total kcal) and added sugars, with cognitive dysfunction. WD factors significantly alter the proportions of commensal bacteria in the gastrointestinal tract. We highlight evidence linking gut bacteria to altered intestinal permeability and blood brain barrier integrity, thus making the brain more vulnerable to the influx of deleterious substances from the circulation. A multistrain combination of lactobacilli, and bifidobacterial has demonstrated in vitro strengthening of the epithelial barrier after a pathogenic bacterial and inflammatory stressors, inhibition of mast-cell activation, stimulation of anti-inflammatory cytokine IL-10, and decreased LPS load. Complex carbohydrates increase levels of beneficial bacteria, in particular, Bifidobacterias spp.. If differences in microbiome composition are present, faecal microbiota transplantation could be done from control individuals, or a defined bacterial consortia. Promising clinical studies have been reported, including in people with autism spectrum disorder. Dietary intervention, e.g. selective diets, prebiotics, or probiotics could be implemented to target the microbiome. However, this has been studied mostly in animal models and validation of such effects of targeting the microbiome with selective diets, prebiotics, or probiotics in humans is now warranted.

Keywords : microbiome, gut-brain axis, brain-gut axis, neurologic disorders

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subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

Combined Impact of Resveratrol Nanoparticles and Physical Activity on Cognitive Function in Alzheimer's Disease Model

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Background and Aim : Alzheimer's disease leads to cognitive deterioration, protein accumulation, synaptic impairment, and neuronal degeneration. Most of the time, the drugs we have now only help with symptoms and don't stop the disease from getting worse. They also often cause side effects like arrhythmias or changes in blood pressure. This is why non-drug treatments like music therapy and exercise are getting more attention. Exercise increases synaptic plasticity, controls gene expression, and boosts memory and learning, all while lowering levels of amyloid-beta in the brain. Resveratrol is a polyphenolic stilbene that protects neurons from oxidative damage, has antioxidant, anti-inflammatory, and neuroprotective effects, and changes how glutamate signals. But its poor solubility makes it hard to use in medicine. Nanoemulsions and gold nanoparticles make resveratrol more bioavailable, stable, and able to reach its target. Combining exercise with resveratrol nanoparticles may help with neuroinflammation and improve brain function. This study examined their effects in an Alzheimer's rat model.

Methods : Wistar rats were randomly assigned to 9 groups (n = 10): healthy control, sham, Alzheimer's disease, treadmill exercise, resveratrol, solvent nanoemulsion, gold and resveratrol, nanoemulsion, and treadmill. Exercise was given to the target groups for 4 weeks before Alzheimer's induction, and solutions containing resveratrol were given orally for 2 weeks after amyloid injection. MWM, EPM, and molecular assessments were performed 21 days after amyloid induction.

Results : We found that rats given Aβ25-35 had increased Anxiety and NLRP3, which were associated with reduced spatial learning and memory. The findings showed that exercise training and administration of resveratrol in the form of nanoformulations increased spatial

learning and memory. In addition, these interventions, exercise and the combination of exercise with gold-containing resveratrol nanoemulsion, reduced anxiety levels and reduced NLRP3 levels in animals that had received Aβ.

Conclusion : In the present study, combining exercise with gold-enriched resveratrol nanoemulsion produced synergistic effects, which may be partly explained by shared mechanisms. We found that both interventions could simultaneously inhibit NLRP3 inflammasome activation. In addition, it is plausible that exercise and resveratrol converge on other complementary pathways, such as enhancing endogenous antioxidant defenses like SOD and GPx. Reduction in oxidative stress protects neurons in the hippocampus and cerebral cortex and enhances cognitive function. The combination of the two interventions can produce a more potent antioxidant effect, reduce oxidative stress, and increase the expression of neuroprotective factors such as BDNF. The gold-enriched resveratrol nanoformulation may have greater brain uptake and, with the enhancing effect of physical exercise, induce stronger protective responses against Aβ-induced damage. These shared mechanisms may help explain the improved cognitive function and reduced anxiety in the combination group. Therefore, this study highlights the potential effect of combining physical exercise with gold-enriched resveratrol nanoformulation as a complementary strategy against Alzheimer's pathology. The observed improvements in memory and anxiety-like behaviors suggest that integrating lifestyle-based and pharmacological interventions could provide a more effective approach to protect against amyloid- -associated neurodegeneration.

Keywords : Alzheimer's, Exercise, Resveratrol, Learning and Memory, Inflammation

Count: 224
Abstract ID: 524
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Systematic miRNA-mRNA-LncRNA Network Analysis Reveals miR-423 as a Key Regulator Hub in Sciatic Nerve Repair

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Background and Aim : Sciatic nerve injury is a complex clinical condition with limited treatment options. RNA sequencing data have shown that non-coding RNAs are crucial for nerve repair, but their coordinated regulatory networks in sciatic nerve regeneration are not well established.

Methods : We analyzed RNA-seq data from a rat model of sciatic nerve injury using computational biology. Using bioinformatics scripting in R, we identified differentially regulated miRNAs, mRNAs, and LncRNAs. Predicted and validated interactions from databases such as miRWalk, TargetScan, and DIANA-LncBase were integrated to construct a large-scale regulatory network. Network analysis was performed using Cytoscape based on centrality values (degree, betweenness, closeness) to identify regulatory hubs.

Results : Our computational examination of the transcriptomic expression revealed a network with 25 nodes and 32 edges. The highest betweenness centrality (53.0) was observed for the master bottleneck node miR-423, indicating that it is a central gatekeeper of the network. Network analysis revealed three functional modules with varying functions: (1) a Neuroinflammation module consisting of TNF, IL-1β, and miR-423; (2) a Neuroregeneration module consisting of NGF, BDNF, and miR-351-5p; and (3) an Apoptosis regulation module consisting of Caspase-3 and miR-125b-1-3p. Visualization using Cytoscape revealed a highly modular organization with modularity score = 0.42.

Conclusion : This network analysis rigorously identifies miR-423 as a central regulatory node in sciatic nerve repair that integrates neuroinflammation, regeneration, and apoptosis processes. Our findings provide an integrated molecular perspective on peripheral nerve regeneration and position miR-423 as a therapeutic target to enhance nerve repair outcomes.

Keywords : Sciatic nerve regeneration; miRNA networks; miR-423; Bioinformatics analysis; Neuroinflammation; Neuroregeneration

Count: 225
Abstract ID: 243
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Neuroprotective and Cognition-Enhancing Effects of Cymbopogon schoenanthus in an Alzheimer's Rat Model

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Background and Aim : Alzheimer's disease (AD), the most common cause of dementia, affects millions worldwide, causing memory loss and cognitive decline due to amyloid plaques and neurofibrillary tangles in the brain. With no cure, researchers are exploring natural compounds to slow its progression. Cymbopogon schoenanthus (lemongrass), a herb long valued in Iranian traditional medicine for its antioxidant and anti-inflammatory properties, shows promise as a neuroprotective agent. This study investigates whether a hydroalcoholic extract of C. schoenanthus can protect brain cells and improve memory in a rat model of AD.

Methods : We studied adult male Wistar rats divided into four groups: a sham group (saline injection), an AD model group (injected with Aβ25-35 in the hippocampus), and two treatment groups receiving 500 or 800 mg/kg of C. schoenanthus extract orally every other day for 28 days (14 doses). The extract was made by soaking dried lemongrass leaves in 70% ethanol and concentrating the solution. Memory and learning were tested using the Passive Avoidance and Morris Water Maze tasks. Brain tissue was analyzed with Cresyl Violet staining for neuron health, Transmission Electron Microscopy for synaptic structure, and qRT-PCR and immunohistochemistry to measure neurogenesis markers (Nestin, DCX, NeuN, β-III tubulin, MAP-2). Data were analyzed using ANOVA in Prism software.

Results : C. schoenanthus extract significantly improved memory and learning in AD rats, with the 800 mg/kg dose showing the strongest effects ($p < 0.05$). Treated rats had healthier hippocampal neurons, more Nissl bodies, and denser synapses compared to the AD group. Electron microscopy revealed more presynaptic vesicles, indicating stronger connections between neurons. Molecular tests showed higher expression of neurogenesis genes and MAP-2 protein, especially at the higher dose, suggesting enhanced brain cell growth and repair.

Conclusion : Lemongrass extract offers hope as a natural therapy for Alzheimer's disease by protecting neurons, boosting synapse formation, and supporting brain cell growth in rats. These dose-dependent benefits highlight its potential, but further studies are needed to confirm safety, optimize dosing, and explore its effects in other AD models or early human trials. This work paves the way for new, nature-inspired treatments to ease the burden of AD.

Keywords : Alzheimer's disease, Cymbopogon schoenanthus, neuroprotection, synaptogenesis

Count: 226
Abstract ID: 184
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Oral

Comparative effects of 3-o-acetyl-11-keto-beta-boswellic acid (AKBA)on neurogenic potential of dentate gyrus-derived neural progenitor cells (NPCs) from young adult and aged rats: An invitro study

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Background and Aim : The slow accumulation of alterations that gradually impair cells' biological activity is known as aging. Cognitive decline and functional frailty are normal aspects of aging. Decreased neurogenesis was a manifestation of these changes in hippocampal formation. An important therapeutic target for encouraging adult neurogenesis may be neural stem cells. Acetyl-11-keto-beta-boswellic acid (AKBA) is the most potent inhibitor of 5-lipoxygenase among the boswellic acids, which are important biological components of the frankincense plant. Although numerous research has shown its anti-inflammatory and neuroprotective properties, little is known regarding its involvement in adult neurogenesis and aging.

Methods : The current study assessed the effects acetyl-11-keto-beta-boswellic acid (AKBA) on the neurogenic potential of neural progenitor cells in the dentate gyrus of both young and old rats. After being separated and cultivated in a particular proliferation and differentiation medium, neural stem cells were exposed to an effective concentration of acetyl-11-keto-beta-boswellic acid. The cell growth curve and cell viability percentage were determined using WST and live/dead assays, and the growth of neurospheres in the experimental groups was assessed. Using immunocytochemistry, the expression of particular proteins differentially expressed in

neural stem cell proliferation and differentiation was investigated qualitatively and quantitatively

Results : It was demonstrated that AKBA markedly enhanced neural stem cell proliferation, survival, and neurogenic potential. As a result, these findings further confirmed that AKBA promotes neurogenesis.

Conclusion : These results highlight AKBA as a promising therapeutic agent for age-related decline in neurogenesis.

Keywords : acetyl-11-keto-beta-boswellic acid (AKBA), Neurogenesis, Aging, Dentate gyrus, Rat

Count: 227
Abstract ID: 479
subject: Neural Injuries and Neurodegenerative Disorders: Neurodegenerative Disorders
Presentation Type: Poster

Carvone reduces neuronal death in the hippocampus of Alzheimers disease model rats

Submission Author: Rana Hasanloo

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Background and Aim : Alzheimer;s disease (AD) is a neurodegenerative disorder characterized by a significant decline in memory and cognitive abilities. Research indicates that widespread neuronal death occurs in the brains of patients with Alzheimer's, particularly affecting the hippocampus, amygdala, temporal, frontal, and parietal lobes. Studies using postmortem brain samples from AD patients and animal models have demonstrated various types of cell death - including apoptosis, necroptosis, ferroptosis, autophagy, and pyroptosis - during the progression of the disease. Evidence suggests that the accumulation of amyloid plaques, hyperphosphorylation of tau protein, and the presence of inflammation in the brain play crucial roles in neuronal death associated with AD. Carvone is a natural monoterpene found abundantly in plants such as Carum carvi L. and Mentha spicata. The anti-apoptotic effects of carvone have been demonstrated in both in vitro and in vivo studies. This study aimed to explore the impact of carvone on hippocampal neuronal death in an AD-like rat model.

Methods : For the experiment, rats were divided into four groups: Control, Carvone, AD, and AD-Carvone. For 10 weeks, the rats received daily doses of AlCl3 (200 mg/kg, orally) and D-galactose (60 mg/kg, intraperitoneally) to induce AD pathology. Carvone was also administered at a dose of 20 mg/kg by oral gavage during the same period. At the end of the treatment duration, the expressions of Bax and Bcl2 in the hippocampus were measured using real-time PCR, while caspase-3 expression was assessed through immunofluorescence. Cresyl violet staining was also applied to brain sections to examine the morphology of the hippocampus microscopically.

Results : The results showed a significant decrease in Bax expression ($p < 0.01$) and a notable increase in Bcl2 expression ($p < 0.001$) in the AD-Carvone group compared to the AD group. The expression of caspase-3 was also significantly lower in the CA1 and dentate gyrus regions

of the hippocampus in the AD-Carvone group compared to the AD group ($p < 0.01$), which corresponded to a significant increase in viable neurons in these areas ($p < 0.01$).

Conclusion : Carvone appears to reduce neuronal death in the hippocampus of AD-like model rats, suggesting its potential as a therapeutic agent for AD.

Keywords : Carvone; Caspase-3; Neural death; Hippocampus; Alzheimer;s disease

Count: 228
Abstract ID: 302
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Fingolimod (FTY720) improves memory and neuroinflammation in a dose-dependent manner in a rat model of Alzheimer's disease induced by streptozotocin

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Background and Aim : Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline, neuroinflammation, and oxidative stress. Fingolimod (FTY720), a sphingosine-1-phosphate receptor modulator, has demonstrated neuroprotective properties. This study aimed to investigate the dose-dependent effects of fingolimod on memory, neuroinflammation, and neuronal loss in a rat model of intracerebroventricular streptozotocin (STZ)-induced AD-like pathology
Methods : Male Sprague-Dawley rats were divided into control, STZ (3 mg/kg, i.c.v.), and STZ + fingolimod (0.25 or 0.5 mg/kg, i.p. for 10 days) groups. Cognitive function was assessed using the Morris water maze (MWM) test. Hippocampal mRNA expression of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) was quantified via qRT-PCR. Histomorphological changes in the hippocampal CA1 region (neuronal count and volume) were assessed using stereological techniques.
Results : STZ administration induced significant memory deficits in the MWM test, and elevated hippocampal levels of TNF-α, IL-1β, and IL-6 expression. STZ also caused a significant loss of CA1 pyramidal neurons and reduced hippocampal volume. Fingolimod treatment at 0.25 mg/kg significantly improved spatial memory, and reduced pro-inflammatory cytokine expression. In contrast, the 0.5 mg/kg dose of Fingolimod significantly restored CA1 pyramidal cell counts and hippocampal volume but did not improve behavioral outcomes.
Conclusion : Fingolimod exerts neuroprotective effects in a dose-dependent manner. A lower dose (0.25 mg/kg) favored functional recovery by improving memory and reducing neuroinflammation, while a higher dose (0.5 mg/kg) promoted structural preservation without concomitant behavioral benefits. These findings highlight the therapeutic potential of fingolimod for AD and underscore the critical importance of dose optimization for achieving desired functional versus structural outcomes.
Keywords : Fingolimod; Alzheimer's disease; Streptozotocin; Neuroinflammation; Memory.

Count: 229
Abstract ID: 86
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Rosuvastatin attenuates total-tau serum levels and increases expression of miR-124-3p in dyslipidemic Alzheimer's patients: a historic cohort study

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Background and Aim : microRNAs are candidate diagnostic biomarkers for Alzheimer's disease. This study aimed to compare Silymarin with Rosuvastatin and placebo on total-Tau protein level and expression levels of microRNAs and TGF-β and COX-2 in Alzheimer's patients with secondary dyslipidemia.

Methods : 36 mild AD patients with dyslipidemia were divided into three groups of 12. The first group received silymarin (140mg), the second group received placebo (140mg), and the third group recieved Rosuvastatin (10mg). Tablets were administered three times a day for Six months. The blood samples of the patients were collected before and after the intervention and the serum was separated. Using the RT-qPCR method, the expression levels of miR-124-3p and miR-125b-5p were assessed, and the serum levels of total-Tau, TGF-β, and COX-2 enzyme were measured using the ELISA method. Data were analyzed with SPSS software.

Results : In this study, the level of Δtotal-Tau was significantly lower in the Rosuvastatin group compared to the placebo (P = 0.038). Also, a significant reduction in the level of ΔTGF-β was observed in the Silymarin to Rosuvastatin group (p = 0.046) and ΔmiR-124-3p was significantly increased in the Rosuvastatin compared to the placebo group (p = 0.044).

Conclusion : Rosuvastatin outperformed silymarin in decreasing Δtotal-Tau serum levels and enhancing expression of ΔmiR-124-3p, attributed to Rosuvastatin's capacity to lower cholesterol levels and inflammation concurrently. Conversely, silymarin was more effective than Rosuvastatin in reducing levels of ΔTGF-β. Serum miR-124-3p could serve as a promising diagnostic biomarker and a new therapeutic focus in AD.

Keywords : Alzheimer's disease; Rosuvastatin; Silymarin; Tauopathy; MicroRNAs

Count: 230
Abstract ID: 260
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Oral

A Comparative Evaluation of Error Processing Performance in Patients with Alzheimer's Disease, Individuals with Mild Cognitive Impairment, and Normal Control

Submission Author: Mohammad Hedayatjoo

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Background and Aim : Some pathological changes occur in patients with Alzheimer's disease (AD) prior to the onset of clinical symptoms. In the present study, we aimed to investigate the potential of event-related potential (ERP) components in error processing performance as a neuromarker of mild cognitive impairment (MCI) and transition to AD.

Methods : We conducted an evaluation of 16 patients diagnosed with AD, 16 patients with MCI, and 15 normal controls using the Mini-Mental State Examination (MMSE) and three subtests from the Cambridge Neuropsychological Testing Automated Battery (CANTAB). The ERP components of error processing were extracted and compared among the three groups using a modified version of the Eriksen flanker task.

Results : Significant differences were observed among the three groups in terms of providing correct responses following errors and the amplitude of error-related negativity (ERN). These differences were also significant between all paired groups. Regarding other ERP components of error processing (correct response negativity and positivity associated with errors) and the peak latency of ERN, no significant differences were observed among the three groups.

Conclusion : In the context of error processing performance, both the accuracy of responses following an error and the amplitude of ERN can be considered as indicators of MCI and its progression to AD. The present findings do not support the use of other error processing components as differential markers in the three groups.

Keywords : Alzheimer's disease, error processing, event-related potential, mild cognitive impairment

Count: 231
Abstract ID: 872
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Investigating the Role of miR-30c in Modulating γ-Secretase Activity: Implications for Alzheimer's Disease

Submission Author: Taiebeh Kafshdooz

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Background and Aim : Alzheimer's disease (AD), the most prevalent form of dementia, affects millions worldwide. Clinical trials with anti-Aβ antibodies demonstrate that amyloid plaque removal in early-stage AD can slow disease progression. γ-Secretase, along with β-secretase, cleaves amyloid precursor protein (APP). This study employs computational docking to identify molecules capable of activating γ-secretase.

Methods : First of all, the targets of hsa-miR-30c-5p were assessed in TargetScanHuman server. The gamma-secretase structure was prepared in Chimera by removing non-standard residues and water molecules. Adjacent amino acids to the cholesterol ligand were then mapped using PyMOL. The 3D structure and SMILES notation for cholesterol were obtained from PubChem. Docking results (pdbqt format) were analyzed using Discovery Studio, LigPlus+, and PDBsum, with LigPlus+ focusing on protein subunit interactions

Results : Target Scan Human server was showed that gamma secretase is a target for hsa-miR-30c-5p. Drug-like properties (solubility, tumorigenicity, LogP, toxicity) were predicted for compounds using tools (Swiss Target Prediction, PASS-Way2drug, SwissADME) adhering to Lipinski's rule of five. The amino acids Trp227, Leu192, Arg186, Leu199, Leu203, Leu206, Tyr155, Leu215, Phe162, Ser223, and Ile230, located on the gamma-secretase C subunit, were analyzed for interactions using Ligplot following AutoDock Vina docking and Chimera visualization.

Conclusion : These in-silico results nominate cholesteryl acetate as a putative γ-secretaseactivator; experimental validation is now warranted

Keywords : Alzheimer's disease, gamma-secretase enzyme, molecular docking,mir 30c

Count: 232
Abstract ID: 868
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Investigating the Disruption of Functional Brain Network Organization in Alzheimer's Disease: An fMRI Network Analysis

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Background and Aim : Alzheimer's Disease (AD) is characterized by progressive deterioration of cognitive function, paralleled by disruptions in large-scale brain network organization. While previous studies have identified functional connectivity alterations, a comprehensive investigation of network-level organization changes remains crucial. This study aimed to systematically investigate the specific patterns of functional network disruption in AD using graph theory analysis of resting-state fMRI data from the ADNI database.

Methods : We analyzed data from 100 carefully matched participants (50 patients with mild AD and 50 healthy controls) from the Alzheimer's Disease Neuroimaging Initiative. Resting-state fMRI data underwent comprehensive preprocessing including motion correction, spatial normalization, and nuisance signal removal. Functional connectivity matrices were constructed based on time series from 90 cortical and subcortical regions defined by the Automated Anatomical Labeling (AAL) atlas. Graph theory metrics including local efficiency, global efficiency, and characteristic path length were computed to quantify network organization. Statistical analyses included group comparisons and correlation analyses with cognitive measures.

Results : Our investigation revealed significant disruption in functional network organization in AD patients compared to healthy controls. Specifically, we observed reduced local efficiency in the default mode network ($p < 0.001$) and increased characteristic path length in global network organization ($p < 0.01$). These topological alterations were most prominent in regions critical for memory and executive function, and showed significant correlations with cognitive impairment severity as measured by standard neuropsychological tests ($r = -0.68$, $p < 0.001$).

Conclusion : This systematic investigation demonstrates that Alzheimer's Disease is associated with a specific pattern of disrupted functional network organization, particularly affecting integration within the default mode network and global network efficiency. These network-based biomarkers may provide valuable insights for early detection and monitoring of disease progression in AD.

Keywords : Alzheimer's Disease; Resting-State fMRI; Functional Connectivity; Brain Networks

Count: 233
Abstract ID: 736
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Oral

Intranasal Administration of Adipose-Derived Exosomes modulates Cognitive Function in a Hyoscine-Induced Mouse Model of Alzheimer's Disease

Submission Author: Mahla Najafi

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Conclusion : Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by memory loss, cognitive decline, and synaptic dysfunction. Despite the availability of pharmacological agents such as cholinesterase inhibitors and NMDA receptor antagonists, current treatments provide only symptomatic relief and do not prevent disease progression. The multifactorial nature of AD pathology—including oxidative stress, Neuroinflammation, and impaired neuronal communication—necessitates innovative therapeutic strategies capable of addressing these mechanisms simultaneously. Exosomes (EXO), Nano-sized extracellular vesicles secreted by stem cells, have emerged as promising biological nano-carriers for neurodegenerative disease therapy. They are biocompatible,non-immunogenic, and capable of transferring bioactive molecules such as proteins, RNAs, and lipids to target cells. Importantly, intranasal administration of EXO provides a non-invasive and efficient route for delivering therapeutic agents directly to the central nervous system (CNS), bypassing the blood–brain barrier (BBB) and enhancing neuronal targeting. Objectives: This study aimed to evaluate the neuroprotective and antioxidant effects of Intranasally administered adipose-derived stem cell (ADSC) EXO in a hyoscine-induced mouse model of Alzheimer's-like cognitive impairment. Methods: Human ADSCs were isolated from lipoaspirate samples, cultured, and characterized morphologically and immunophenotypically by the presence of positive markers ($CD90^+$, $CD73^+$, $CD105^+$) and absence of hematopoietic markers ($CD34^-$, $CD45^-$). EXO were extracted from ADSC-conditioned medium using a polyethylene glycol–based precipitation method and characterized by transmission electron microscopy (TEM), dynamic light scattering (DLS), and flow-cytometry for surface markers CD9, CD63, and CD81. Adult female mice were randomly assigned to control, hyoscine-treated, and EXO-treated groups. Hyoscine was used to induce transient cholinergic dysfunction and memory impairment, mimicking Alzheimer's-like pathology. ADSC-derived

EXO were administered Intranasally over a defined treatment period. Cognitive performance was assessed using the elevated plus maze (EPM) test, with transfer latency (TL) serving as an index of learning and memory. Oxidative stress was evaluated by quantifying Malondialdehyde (MDA) levels in Hippocampal tissue using a spectrophotometric assay. Results: Hyoscine-treated mice showed a significant increase in TL and elevated Hippocampal MDA levels, indicating memory impairment and oxidative stress. In contrast, intranasal EXO treatment significantly reduced TL values, demonstrating improved learning and memory. Additionally, Hppocampal MDA levels decreased markedly in EXO-treated mice compared to the hyoscine group, reflecting enhanced antioxidant activity. Characterization analyses confirmed that the EXOs were spherical nanoparticles (80–150 nm) expressing the exosomal surface markers CD9, CD63, and CD81. Conclusion: Intranasal administration of adipose-derived EXO effectively improved memory function and reduced oxidative stress in a hyoscine-induced mouse model of Alzheimer's disease. These findings suggest that EXO can modulate neuroinflammatory and oxidative pathways while promoting neuronal recovery. The intranasal route offers a non-invasive and efficient method for delivering EXO-based therapeutics directly to the brain, highlighting its potential as a novel and translational strategy for neurodegenerative disease therapy. Further studies are warranted to explore the long-term efficacy, molecular mechanisms, and clinical applicability of intranasal EXO-based treatments for Alzheimer's and related neurodegenerative disorders.

Keywords : Alzheimer's disease; Exosomes; Intranasal delivery; Cognitive impairment; Oxidative stress; Adipose-derived stem cells; Neurodegenerative disease therapy

Count: 234
Abstract ID: 734
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Postmortem neuropathologies are associated with retrospective tensor-based morphometry findings in Alzheimer's dementia continuum

Submission Author: Mahla Najafi

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Background and Aim : Alzheimer's disease (AD) is the leading cause of dementia worldwide, characterized by progressive cognitive decline and neurodegeneration. Although clinical diagnosis relies on neuroimaging and cognitive testing, definitive confirmation requires postmortem neuropathological examination. Bridging the gap between in vivo imaging and neuropathology is essential for validating imaging biomarkers and improving diagnostic accuracy. Tensor-based morphometry (TBM) is a magnetic resonance imaging (MRI) technique that quantifies regional brain atrophy. While TBM correlates with disease severity, its association with postmortem neuropathological measures remains insufficiently explored. This study aimed to assess how TBM-derived regional atrophy indices correspond to neuropathological findings in individuals across the Alzheimer's disease continuum.

Methods : MRI and neuropathological data were obtained from participants enrolled in the Alzheimer's Disease Neuroimaging Initiative (ADNI) who later underwent autopsy. TBM was applied to structural MRI scans to generate voxel-wise maps of local brain deformation, focusing on two validated indices: the statistically defined region of interest (StatROI) and temporal lobe region of interest (TempROI). Neuropathological data included standardized assessments of amyloid-β plaques, neurofibrillary tangles, neuritic plaques, Lewy body pathology, hippocampal sclerosis, and vascular lesions. Logistic regression models were used to evaluate associations between TBM indices and neuropathological outcomes, adjusting for age, sex, and time interval between MRI and death. Diagnostic performance of TBM measures in differentiating AD from non-AD pathologies was assessed using receiver operating characteristic (ROC) analyses.

Results : Both StatROI and TempROI TBM indices showed significant correlations with postmortem AD neuropathological markers. Smaller TBM values, indicating greater atrophy,

were strongly associated with higher Braak stage and greater neuritic plaque burden. Participants with autopsy-confirmed AD pathology had significantly lower TBM measures than those with non-AD or mixed pathologies. Logistic regression demonstrated that each unit increase in TBM index was associated with reduced odds of high amyloid or tau pathology. ROC analysis revealed that TBM measures distinguished AD from non-AD dementia with high specificity (≥85%) and moderate sensitivity (≈70%). No significant associations were observed between TBM indices and isolated vascular or Lewy body pathology, supporting the specificity of TBM changes to AD-type degeneration.

Conclusion : This study provides robust evidence linking in vivo structural MRI findings with postmortem neuropathological confirmation of Alzheimer's disease. TBM-derived regional atrophy indices accurately reflect underlying amyloid and tau pathology, validating TBM as a potential biomarker for distinguishing AD from non-AD dementias. These results highlight the clinical value of combining neuroimaging and pathological data to enhance diagnostic precision and facilitate early intervention strategies in neurodegenerative disorders. Future studies should explore longitudinal TBM changes and multimodal imaging approaches to further refine biomarker validation in the Alzheimer's continuum. This presentation summarizes findings from our recent publication in Neurological Sciences (2025), focusing on the association between imaging biomarkers and postmortem neuropathologies in the Alzheimer's disease continuum.

Keywords : Alzheimer's disease; Dementia continuum; Tensor-based morphometry (TBM); Magnetic resonance imaging (MRI); Neuroimaging biomarkers; Postmortem neuropathology; Amyloid-β plaques;

Count: 235
Abstract ID: 610
subject: Neural Injuries and Neurodegenerative Disorders: Dementia
Presentation Type: Poster

Association Between Cognitive Impairment and Gait Alterations in Patients Presenting with Cognitive Complaints to a Neurology Clinic

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Background and Aim : Cognitive impairment and gait disturbance are two frequent manifestations in neurological patients and may share overlapping neural substrates within frontal and subcortical circuits. This study aimed to investigate the relationship between cognitive changes and gait alterations among patients presenting with cognitive complaints to a neurology clinic, and to determine whether gait parameters could serve as clinical markers of cognitive decline.

Methods : A cross-sectional analytical study was conducted in a neurology outpatient clinic in 2025. Participants aged 50 years and above with subjective or objective cognitive complaints were enrolled. Exclusion criteria included severe psychiatric disorders, acute neurological diseases, and major mobility limitations. Cognitive status was assessed using the Montreal Cognitive Assessment (MoCA). Gait parameters—such as gait speed under both single-task and dual-task single-task, stride length, stride time,swing time and dual-task gait speed—were measured under single-task condition. Demographic and clinical variables were recorded, and statistical analyses explored the associations between cognitive and gait performance.

Results : Patients who demonstrated lower cognitive scores also exhibited notable gait changes characterized by slower walking speed, shorter stride length, and increased variability. These gait alterations were most prominent in individuals with mild cognitive impairment compared with cognitively normal participants. Dual-task walking conditions, which require simultaneous cognitive and motor engagement, revealed stronger differences between groups, suggesting that attentional and executive deficits significantly influence motor control. Individuals with better executive and attentional abilities maintained more stable gait patterns, while those with cognitive slowing showed disrupted coordination and greater step variability. Overall, the findings indicate that gait performance reflects the integrity of higher-order cognitive processes, and subtle gait changes can serve as a window into early neurocognitive dysfunction.

Conclusion : This study highlights a meaningful association between cognitive performance and gait characteristics in patients presenting with cognitive complaints. Gait alterations—especially reduced speed and increased variability during cognitively demanding tasks—may function as sensitive clinical indicators of early cognitive decline. Integrating simple gait assessments into routine neurological evaluations could facilitate the early identification of individuals at risk for dementia and other neurodegenerative conditions. Further longitudinal research is recommended to clarify causal relationships and predictive value.

Keywords : Cognitive impairment; gait analysis; dual-task walking; neurology clinic; dementia risk; mild cognitive impairment

Count: 236
Abstract ID: 210
subject: Neural Injuries and Neurodegenerative Disorders: Spinal Cord Injury
Presentation Type: Oral

Photobiomodulation therapy: location-dependent effects on memory and recovery in male adult rats with spinal cord injury

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Background and Aim : Spinal cord injuries (SCIs) can cause significant physical and emotional challenges for individuals as a result of inflammation and cell death. This study investigates the effectiveness of photobiomodulation therapy (PBMT) in reducing inflammation and memory impairment through the use of 3 specific treatment protocols.

Methods : Forty male rats (180-200g) were divided into 5 groups: Control; SCI model; and SCI with 3 different PBMT protocols. The rats underwent behavioral tests (Novel Object Recognition and Shuttle Box), histological examination (H&E and Nissl staining), and protein analysis (HDAC2, NF-kB [p65], IkB-α, and BDNF) using Western blot of medial prefrontal cortex (mPFC) and hippocampal tissues.

Results : PBMT effectively decreased NF-κB levels in the mFPC in all 3 PBMT protocols. The third PBMT3 protocol with the highest energy delivery, which included both the spinal cord and skull had the greatest impact on regulating the expression of this inflammatory factor. Behavioral assessments revealed that the PBMT3 protocol led to the most significant enhancement in cognition.

Conclusion : The therapeutic effects of PBMT on memory are thought to be mediated by the enhancement of neurotrophic factors such as BDNFand the reduction of inflammatory mediators, which can impair hippocampal and mPFC function.

Keywords : cognition; HDAC2; inflammation; memory; photo-biomodulation therapy; spinal cord injury.

Count: 237
Abstract ID: 351
subject: Neural Injuries and Neurodegenerative Disorders: Spinal Cord Injury
Presentation Type: Oral

Innovative hydrogel containing stem cells for CNS tissue engineering

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Background and Aim : Spinal cord injury (SCI) causes severe and often irreversible neurological damage due to the limited regenerative capacity of the central nervous system. Stem cell-based therapies, such as those using OE-MSCs, offer promising potential for regeneration, but their survival and integration remain challenging. Injectable hydrogels provide a supportive three-dimensional environment for cell delivery and bioactive molecule release, making them attractive candidates for SCI repair.

Methods : Preparation and characterization of hydrogel/ OE-MSCs isolation and characterization/ In vitro cytotoxicity of hydrogels/ In vivo study of the hydrogel containing OE-MSCs

Results : The cell-laden hydrogel demonstrated favorable mechanical properties, high cell viability, and sustained release of bioactive factors. In vivo studies revealed enhanced axonal regeneration, reduced glial scar formation, and improved functional recovery.

Conclusion : These findings indicate that the OE-MSC encapsulated hydrogel provides a supportive microenvironment for cell survival and neurogenesis, highlighting its potential as a promising therapeutic strategy for spinal cord injury repair.

Keywords : hydrogels, stem cells, spinal cord injury

Count: 238
Abstract ID: 822
subject: Neural Injuries and Neurodegenerative Disorders: Spinal Cord Injury
Presentation Type: Poster

Investigating the Effect of Human Adipose Mesenchymal Stem Cells Treated with Resveratrol on Recovery of Function, Repair, and Tissue Regeneration in Rat Spinal Cord Injury Model

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Background and Aim : Spinal cord injury (SCI) often results in lifelong disability, muscle paralysis, and sensory disturbances, as well as bowel and bladder incontinence, depending on the severity of the spinal cord injury. Improving the biological performance of stem cells before their transplantation can upgrade the efficiency of cell therapy. Lately, resveratrol has been used for the Improvement of the paracrine abilities of mesenchymal stem cells (MSCs) for tissue healing. We aimed to pre-treat MSCs with resveratrol and transplant MSCs into the rat SCI model.

Methods : First, mesenchymal stem cells were isolated from adipose tissue. Then, BCA, MTT, and NGF ELISA tests were performed. After spinal cord injury was induced in Rats by the weight-dropping method, the rats were randomly divided into four groups. mesenchymal stem cells were injected into the site of injury 30 minutes later. The BBB motor improvement test was done for four weeks. Luxal fast blue and hematoxylin-eosin staining were done to evaluate the tissues.

Results : According to the results of toxicity and survival of resveratrol on mesenchymal stem cells, the concentration of 0.2 micromolar had the highest survival rate and the highest level of significance compared to the control group. Based on the results of ELISA, the concentration of 0.2 increased the expression of NGF in the supernatant of adipose mesenchymal stem cells. There was a significant increase in the average scores of BBB in groups of untreated stem cells and stem cells treated with resveratrol in the fourth week after the injury, compared to the BBB scores of the injury (SCI) group. The pathological changes detected improved with MSC+RES treatment, as in the adjacent segments in this group, the rate of cell death is lower, and less neuronal damage can be observed, compared with the SCI group

Conclusion : We predict that transplanted MSCs treated with resveratrol would have a better efficacy for promoting functional recovery in rats with spinal cord injury.

Keywords : pinal Cord Injury, Resveratrol, Mesenchymal Stem Cells

Count: 239
Abstract ID: 323
subject: Neural Injuries and Neurodegenerative Disorders: Spinal Cord Injury
Presentation Type: Poster

Combined Tadalafil and Carvedilol Therapy Attenuates Oxidative Stress in a Rat Model of Neuropathic Underactive Bladder

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Background and Aim : Detrusor underactivity (DU), the urodynamic hallmark of underactive bladder (UAB), is defined by insufficient contraction strength or duration, resulting in incomplete or prolonged bladder emptying and contributing to chronic lower urinary tract symptoms. Its multifactorial etiology encompasses obstructive, metabolic, and neurogenic conditions such as spinal cord injuries, yet the syndrome remains underrepresented in preclinical research, limiting therapeutic development. Phosphodiesterase type 5 (PDE5) inhibitors such as tadalafil have demonstrated smooth muscle relaxant, anti fibrotic, and sensory modulating effects in urologic disorders, while carvedilol, a non selective β blocker with α1 blocking activity, also exhibits TRPM8 channel inhibition, implicating potential benefits for bladder afferent modulation. However, the impact of these agents, alone or in combination, on neuropathic UAB has not been systematically investigated. To address this gap, we evaluated tadalafil, carvedilol, and their combination in a rat model of chronic spinal cord stenosis–induced UAB.

Methods : 25 adult female Wistar rats with free access to food and water, were randomly allocated into five groups: sham, UAB control, tadalafil treated, carvedilol treated, and combination treated. Anesthesia was induced with ketamine (60 mg/kg) and xylazine (6 mg/kg) intraperitoneally. In all groups except sham, the L5–L6 segments of the spine were exposed via midline dorsal incision. A small opening in the vertebral arch was created, and a sterile

polyethylene sheet (1 cm × 1 cm) was carefully inserted into the epidural space to induce chronic lumbar spinal stenosis, a neuropathic model of underactive bladder. The muscle and skin were sutured in layers, and animals received postoperative analgesia and daily monitoring. Drug treatments were administered via oral gavage for 30 days starting on postoperative day 3. At the end of week 4, animals were euthanized under deep anesthesia, and bladder tissue samples were collected. Tissue homogenates were analyzed for oxidative stress biomarkers including glutathione peroxidase (GPx), total antioxidant capacity (TAC), and malondialdehyde (MDA) using standard biochemical assay protocols. All experimental procedures conformed to institutional guidelines for the care and use of laboratory animals.

Results : Chronic lumbar spinal stenosis significantly reduced antioxidant enzyme activities and TAC, and elevated MDA levels compared with sham controls (all p??0.05), and TAC approached baseline values., indicating synergistic antioxidant effects.

Conclusion : Tadalafil and carvedilol exerted protective antioxidant effects in a rat model of neuropathic UAB, with combination therapy yielding the greatest restoration of enzymatic defenses and lipid peroxidation levels toward sham values. These findings suggest that concurrent modulation of smooth muscle tone, sensory pathways, and oxidative stress may offer a synergistic strategy for mitigating bladder dysfunction secondary to spinal cord pathology. Further studies integrating urodynamic and histopathological outcomes are warranted to confirm translational potential.

Keywords : Under Active Bladder, Detrusor Underactivity, Tadalafil, Carvedilol, Urology

Count: 240
Abstract ID: 447
subject: Neural Injuries and Neurodegenerative Disorders: Spinal Cord Injury
Presentation Type: Oral

assesment the effect of curcumin on the expression of nestin and beta-tuberculin genes in the differentiation of mesenchymal stem cells into neural-like cells

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Background and Aim : Studying the effect of curcumin on the expression of nestin and beta-tuberculin genes in the differentiation of mesenchymal stem cells into neural-like cells. To date, pharmaceutical and surgical advances in the field of neuroscience have not revealed a definitive treatment for eliminating sensory, motor, and autonomic disorders caused by spinal cord injury. Today, mesenchymal stem cells are used in the repair of tissues of mesenchymal origin, such as bone, cartilage, muscle, tendon, fat, and nerve, due to their ability to differentiate into various cell lineages without the risk of tumorigenesis. On the other hand, mesenchymal stem cells have the ability to establish stable communication with hematopoietic stem cells, which provides the basis for proliferation with minimal differentiation of these cells. Curcumin is a phytochemical compound extracted from the root of the turmeric plant and has anti-inflammatory, antioxidant, and anticancer properties. Research has shown that curcumin can improve neurological diseases such as Alzheimer's and Parkinson's. Curcumin is a chemical compound found in turmeric. This compound is part of the phenolic family. Health benefits of curcumin include reducing inflammation, boosting the immune system, reducing the risk of heart and brain disease, resisting infections, and helping control blood sugar levels. The gene nestin plays an important role in the differentiation of stem cells into neural-like cells. Nestin is a protein found in nerve and muscle cells and plays an important role in cell adhesion and stability. Nestin and beta-tuberculin are known to be important factors in cell differentiation and development of neural-like cells. Research in this area can help to better understand the processes of cell differentiation and the effects of curcumin on these processes. In this study, we investigated the effect of curcumin at two concentrations on the neurogenesis process.

Methods : First, we isolated mesenchymal stem cells from adipose tissue and then evaluated the viability of the cells at two concentrations of curcumin, 5 and 10 µg/ml. After selecting the superior concentration of curcumin, we examined the expression of nestin and β-Tubulin III genes in real time PCR.

Results : The results of the MTT test showed that curcumin at a concentration of 10 had a higher survival rate than at a concentration of 5. Also, the expression of nestin and β-Tubulin III genes increased at a concentration of 10 µg/ml compared to the control group.

Conclusion : Curcumin can play a role as an agent in the differentiation process of mesenchymal stem cells into neural cells.

Keywords : mesenchymal stem cells, Neurogenesis, Differentiation

Count: 241
Abstract ID: 570
subject: Neural Injuries and Neurodegenerative Disorders: Spinal Cord Injury
Presentation Type: Oral

Collagen Scaffold Containing Gold-Quercetin Nanoparticles Attenuates Inflammation and Improves Sensorimotor Function in a male Rat Model of Unilateral Spinal Cord Injury

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Background and Aim : Spinal cord injury (SCI) is a profound neurological condition characterised by dysfunction of the autonomic nervous system and the loss of sensory and motor function that imposes a substantial physical, social, and economic burden. Despite extensive research into therapeutic interventions, an effective treatment strategy for SCI remains elusive. Among promising approaches, biodegradable scaffolds, particularly collagen scaffolds, are valued for their biocompatibility, optimal porosity, and their capacity to support cellular migration and differentiation. Quercetin, a flavonoid with anti-inflammatory, antioxidant, and neuroprotective properties, has also emerged as a potential therapeutic agent for spinal cord repair. To overcome the poor aqueous solubility of quercetin, gold nanoparticles were utilized as a delivery vehicle. Therefore, the present study was designed to evaluate the therapeutic efficacy of an electrospun collagen scaffold (CS) containing gold-quercetin nanoparticles (Qur-GNPs) for promoting neural tissue regeneration and restoring sensorimotor function in a male rat model of unilateral spinal cord injury.

Methods : Qur-GNPs were synthesised via a green chemistry method and characterised (UV-Vis, DLS/ζ-potential, TEM). Antioxidant capacity and cell viability of SK-N-SH cells were assessed using DPPH and MTT assays, respectively. Electrospun collagen scaffolds, with and without Qur-GNPs, were fabricated (using the electrospinning method), cross-linked with glutaraldehyde, and characterised for nanofiber structure (SEM), wettability (contact angle),

electrical conductivity (two-probe method), swelling, and biodegradability. A unilateral T9 hemisection SCI model was established in male rats (n=12/group), assigned to five groups: Sham, SCI, SCI+CS, SCI+CS-GNP, and SCI+CS-Qur-GNP. Sensorimotor function was evaluated and scored from day 2 to day 28 using a battery of tests, including the BBB scale, placing reflex, incline plane, righting reflex, withdrawal reflex (i.e., pain, pressure, extension), beam walk, and forced swim tests. Gene expression of TNF-α, IL-1β, and IL-10 was quantified by qRT-PCR on days 3, 14, and 28. At day 28, histological examinations were performed using H&E staining.

Results : Qur-GNPs had an average diameter of ~5 nm (TEM), a hydrodynamic diameter of ~26 nm (DLS), and potent antioxidant activity ($IC_{50} \approx 0.036$ mg·ml^{-1} quercetin). The collagen nanofibers (with an average diameter of ~1 µm), when loaded with Qur-GNPs (400 ppm), showed improved wettability (contact angle < 40°), electrical conductivity (2.5x control), and structural stability (~87% remaining after 28 days). In vivo, during the 28-day observation period, the CS-Qur-GNP group demonstrated the most rapid and nearly complete sensorimotor recovery across a battery of behavioral assessments. This outcome was evidenced by the group achieving a high BBB score of 15.9 by week four. The histological analysis confirmed that CS-Qur-GNP group had the highest parenchymal preservation and minimal cavitation. The treatment also modulated inflammation. On day 3 post-injury, the CS-Qur-GNP group showed significantly attenuated expression of pro-inflammatory TNF-α and IL-1β (2.47- and 2.1-fold increases vs. sham, respectively) compared to the untreated SCI group (5.8- and 4.1-fold increases). Conversely, by day 14, anti-inflammatory IL-10 was significantly upregulated in the treatment group (1.45-fold increase over the SCI group).

Conclusion : In conclusion, the CS-Qur-GNP scaffold is a promising therapeutic for SCI repair, promoting neurological recovery by modulating inflammation, promoting neural tissue regeneration, and restoring sensorimotor function.

Keywords : Collagen Scaffold, Gold-Quercetin Nanoparticles, Inflammation, Spinal Cord Injury

Count: 242
Abstract ID: 774
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Oral

Synergistic Neuroprotective Effects of Vitamin D and KATP Channel Opener in a Rat Model of Parkinson's Disease

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Background and Aim : Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by the degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to motor and non-motor symptoms. Existing treatments, particularly levodopa, do not provide a definitive cure and are often accompanied by side effects like dyskinesia, limiting their effectiveness. This highlights the urgent need for novel therapeutic strategies, especially co-treatment and multi-targeted approaches. Recent studies suggest that vitamin D, a neuroactive steroid hormone, may have neuroprotective properties in PD through antioxidant, anti-inflammatory, and neurotrophic mechanisms. Additionally, ATP-sensitive potassium (KATP) channels play a crucial role in regulating neuronal excitability and mitochondrial function. Dysregulation of these channels has been linked to neurodegenerative processes, and pharmacological modulation—especially with openers—has shown promise in mitigating neuronal death.

Methods : A total of 48 adult rats were randomly assigned to six groups (n = 8 each): Control (C), PD control (PD), PD + vitamin D (PD+VD), vitamin D control (VD), PD + diazoxide (PD+DIA), and PD + vitamin D + diazoxide (VD + DIA). PD was induced through stereotaxic injection of 6-OHDA into the medial forebrain bundle (MFB). Treatments including vitamin D (i.p. 1 µg/kg) and/or diazoxide (i.p. 5 mg/kg) were begun 24 hours before the surgery and continued for 10 weeks afterwards. Behavioral assessments, including apomorphine-induced rotation and beam-walking tests, were conducted at weeks 3, 7, and 8 post-surgery. Ten weeks after the surgery, half of the rats from each group were perfused for immunofluorescence analysis of tyrosine hydroxylase (TH) to evaluate dopaminergic neuronal survival in the

substantia nigra, while the remaining rats underwent western blotting to quantify brain-derived neurotrophic factor (BDNF) expression in the striatum.

Results : The PD group exhibited significant dopaminergic neuronal loss, evidenced by substantial TH depletion and impaired behavioral performance. Administration of vitamin D (PD+VD) significantly reduced neuronal death and improved motor function compared to the PD group. Additionally, vitamin D treatment resulted in a notable increase in BDNF expression in the striatum, indicating enhanced neurotrophic support. Diazoxide treatment (PD+DIA) provided moderate neuroprotective effects with partial recovery in behavioral assessments. Remarkably, the combined treatment with vitamin D and diazoxide (VD+DIA) resulted in the most significant improvements at the molecular level, including enhanced TH preservation and increased BDNF expression, along with decreased apomorphine rotations and improved beam-walking scores. These findings indicate that vitamin D primarily mitigates disease progression through neurotrophic and neuroprotective mechanisms, while diazoxide offers additional protection via mitochondrial stabilization and modulation of KATP channels. The combination therapy synergistically enhanced dopaminergic neuron survival and motor function.

Conclusion : Vitamin D and diazoxide exhibit distinct yet complementary neuroprotective mechanisms in PD. Vitamin D slows disease progression by promoting neurotrophic support and elevating BDNF levels, while diazoxide provides further protection through KATP channel-mediated mitochondrial stabilization. The most substantial therapeutic benefits were observed with the combined treatment, highlighting the potential of multi-targeted strategies for managing PD. These results underscore the importance of investigating combinatorial approaches in addressing neurodegenerative disorders characterized by complex pathological mechanisms.

Keywords : Parkinson's Disease; Vitamin D; KATP channels; BDNF; Diazoxide; 6-OHDA

Count: 243
Abstract ID: 430
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Metformin protective effects in LPS-Induced Alzheimer's disease mice model: NO-cGMP-KATP pathway involvement

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Background and Aim : This project has studied the effects of metformin on cognition impairment, depression, hyperalgesia, stress oxidative and neuroinflammation in a rodent Alzheimer's disease (AD) model created via LPS (lipopolysaccharide). For defining possible mechanisms, the NO/cGMP/KATP pathway roleplay was considered.

Methods : Mice model was created via LPS treatment. Open field, forced swimming and hot plate tests were done. Shuttle-box test and Y-maze test were used to assay Learning-memory. Biochemical assay compromised malondialdehyde (MDA) and TNF-alpha concentration and superoxide dismutase (SOD) activity measurement in hippocampus samples. NO-cGMP-KATP pathway contribution was assessed by its agonists/antagonist pre-treatment, 15 min before metformin (150, 200, 250 mg/kg).

Results : Initial latency (IL) was increased by LPS injection while it was reduced by metformin (250 mg), in shuttle-box test. Pretreatment with methylene blue, L-NAME and glibenclamide before metformin augmented IL, although it was diminished by L- arginine and sildenafil pretreatment. Also, metformin increased the LPS induced step through latency (STL) reduction. L-NAME, methylene blue and glibenclamide decreased the STL, but it was increased by L-arginine and sildenafil. In Y-maze test, metformin increased the LPS-induced spontaneous alternation reduction. L-NAME, methylene blue and glibenclamide decreased it. LPS added immobility time in forced swimming trial, whereas it was reduced thru metformin.

L-NAME, methylene blue and glibenclamide increased the anti-depressive effect of metformin while it was attenuated via L-arginine, sildenafil and diazoxide. LPS treatment diminished threshold of pain perception in hot-plate test, while metformin didn't have any significant effect. Metformin reduced the LPS-induced lipid peroxidation (MDA level). But, L-NAME, methylene blue and glibenclamide worsen the lipid peroxidation, whereas it was improved by L-arginine and sildenafil. Metformin improved LPS-induced reduction in SOD activity. SOD activity was reduced by L-NAME, methylene blue and glibenclamide pre-treatment. LPS enhanced TNF-alpha amount that decreased by metformin. Pre-injection with methylene blue, L-NAME and glibenclamide increased TNF-alpha concentrations while L-arginine, sildenafil and diazoxide reduced it.

Conclusion : Metformin can improve learning-memory loss, depression, hyperalgesia, neuroinflammation and oxidative stress produced by LPS and NO/cGMP/KATP pathway maybe has a roleplay.

Keywords : Metformin; cognition; oxidative stress; depression; hyperalgesia; neuroinflammation

Count: 244
Abstract ID: 1043
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Immunomodulatory Mechanisms of Mesenchymal Stem Cell-Derived Extracellular Vesicles in Spinal Cord Injury

Submission Author: Faezeh Faghihi

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Conclusion : Mesenchymal stem cell-derived extracellular vesicles (MSC-EVs) have emerged as promising cell-free therapeutic agents capable of modulating the post-injury inflammatory microenvironment through sophisticated molecular mechanisms. MSC-EVs derived from various tissue origins exhibit distinct cargo profiles that significantly influence their immunomodulatory properties. The EVs orchestrate coordinated anti-inflammatory responses through five interconnected pathways: suppression of pro-inflammatory NF-κB signaling, activation of anti-inflammatory TGF-β/Smad cascades, rebalancing of STAT1/STAT6 to favor M2 macrophage polarization, regulation of complement-mediated tissue damage, and inhibition of inflammasome-driven cytokine release. These pathways function synergistically through positive feedback loops, explaining superior therapeutic outcomes compared to single-target interventions. The molecular basis involves delivery of immunoregulatory microRNAs (miR-146a, miR-124, miR-223), anti-inflammatory proteins (TGF-β1, IL-10, TSG-6), and bioactive lipids that converge on key immune cell populations including macrophages/microglia, T cells, and dendritic cells, orchestrating their phenotypic and functional reprogramming. This comprehensive review examines the immunomodulatory mechanisms of MSC-derived extracellular vesicles in spinal cord injury. We investigated EV-mediated modulation of post-injury inflammatory cascades, compared immunomodulatory properties across different MSC tissue sources, analyzed molecular cargo and signaling pathways, and identified critical knowledge gaps requiring further investigation. This review provides a foundation for advancing mechanistic understanding of EV-based immunotherapy for spinal cord injury.

Keywords : Mesenchymal stem cells; Extracellular vesicles; Spinal cord injury; Immunomodulation; Neuroinflammation.

Count: 245
Abstract ID: 211
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Linking Neuroinflammation to Sleep Dysfunction: The Role of NLRP3 Inflammasome and α-Synuclein in Chronic Insomnia

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Background and Aim : Chronic insomnia disorder (CID) is a highly prevalent condition that significantly impairs physical and mental health, with increasing evidence suggesting it extends beyond a primary sleep disturbance to involve systemic inflammation and neurobiological dysfunction. Recent studies implicate the NLRP3 inflammasome, a key regulator of innate immune activation, and α-synuclein, a neuronal protein associated with misfolding and neurodegenerative processes, in the pathophysiology of sleep disorders. However, the interaction between systemic inflammatory activation and α-synuclein dysregulation in the context of CID remains poorly understood. This study aims to evaluate the association between systemic NLRP3 inflammasome activity, serum α-synuclein levels, and insomnia severity in individuals with CID compared to healthy controls.

Methods : A case-control study was conducted at the Sleep Disorders Research Center of Farabi Hospital. Patients with clinically diagnosed CID were recruited according to ICSD-3 criteria, supported by Pittsburgh Sleep Quality Index (PSQI) evaluation and confirmed by overnight video polysomnography. Age- and sex-matched healthy controls without sleep disturbances were enrolled for comparison. Strict inclusion and exclusion criteria were applied to minimize the influence of confounding systemic illnesses or immunological conditions. Following overnight sleep assessment, blood samples were collected. Serum NLRP3 inflammasome activity and α-synuclein levels were quantified using validated human-specific ELISA kits. Statistical analyses were performed using SPSS software to compare marker levels between groups and to examine correlations with clinical sleep parameters.

Results : Patients with CID exhibited markedly elevated serum levels of both NLRP3 inflammasome and α-synuclein relative to healthy participants. Strong positive correlations

were observed between systemic NLRP3 activity and insomnia severity, as measured by sleep quality indices. Similarly, α-synuclein concentrations correlated positively with both NLRP3 activity and clinical indicators of insomnia severity. These findings highlight a close relationship between systemic immune activation, protein aggregation processes, and the clinical manifestation of chronic insomnia.

Conclusion : The results of this study provide evidence that systemic activation of the NLRP3 inflammasome and elevated serum α-synuclein levels are closely linked to the pathophysiology of chronic insomnia disorder. The observed correlations suggest a potential mechanistic interplay between neuroinflammation, protein misfolding, and disrupted sleep regulation. Future studies with larger cohorts and mechanistic designs are warranted to further clarify the biological pathways underlying these associations and to explore the potential of targeted anti-inflammatory or neuroprotective interventions in the management of insomnia.

Keywords : Chronic insomnia, NLRP3 inflammasome, α-synuclein, Neuroinflammation

Count: 246
Abstract ID: 466
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Oral

The Effect of a Chalcone derivatives on memory impairment in a scopolamine-induced rat model

Submission Author: Nasrin Hosseini

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Background and Aim : Emerging evidence propose that chalcone and its derivatives can improve cognitive performance and memory by protecting neurons from damage. In consideration of memory impairment is closely linked to oxidative imbalance and neuroinflammation, chalcone shows promising potential in preventing or mitigating memory-related disorders, including Alzheimer's disease. These findings highlight chalcone as a valuable candidate for further research in the development of neuroprotective and memory-enhancing therapeutics, therefore the aim of this study was investigate a chalcone derivate on memory in a scopolamine-induced rat model

Methods : In this experiment, the 24 male wistar rats were randomly grouped in control, sham, scopolamine, and scopolamine+ methoxyphenyl chalchon (20 mg/kg, intraperitoneal, IP) groups. Scopolamine injection was intraperitoneal at a dose of 20 mg/kg. Than behavioral Barnes Maze test and Passive avoidance test were assessed 24 hours after.

Results : Results of Barnes Maze test and passive avoidance test presented significant ($p<0.05$) differences between the scopolamine and Chalcone groups. scopolamine induced memory impairment in rats and methoxyphenyl chalchon attenuated memory impairment

Conclusion : It has been already proposed that flavonoids have beneficial effects on short term memory in fear and spatial memory performance. According to these results, further investigations are required to explore mechanisms that could be acutely modulated by chalcones.

Keywords : Scopolamine; Chalcone; Barnes Maze test; Passive avoidance test;Rat

Count: 247
Abstract ID: 83
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Probiotic Modulation of Oxidative Stress and Neuroinflammation in an STZ-Induced Alzheimer-like Model

Submission Author: Ardeshir Nabizadeh zolpirani

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Background and Aim : Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline and neuronal dysfunction, with oxidative stress and neuroinflammation as core pathological mechanisms. Increasing evidence implicates the gut–brain axis as a crucial modulator of neurodegeneration, pointing to probiotics as potential therapeutic agents in slowing AD progression.

Methods : Thirty-five male Sprague–Dawley rats were randomly assigned to five groups: Sham, STZ, STZ+Ac, STZ+Re, and STZ+Comb. An AD-like phenotype was induced by intracerebroventricular injection of STZ, and probiotics were administered by gavage for 35 days. Behavioral assessments, including the open field test (OFT) and Morris water maze (MWM), were conducted to evaluate locomotion, anxiety-like behavior, and spatial learning and memory. Oxidative stress biomarkers in the hippocampus—malondialdehyde (MDA), glutathione (GSH), superoxide dismutase (SOD), and catalase (CAT)—were measured biochemically. Histological and stereological methods were employed to assess hippocampal volume, neuronal density, and glial activation.

Results : The results demonstrated profound cognitive and neuroprotective benefits of probiotic treatment. In behavioral tests, STZ rats displayed reduced center time, center entries,

and grooming behavior, alongside increased stretch body elongation, leaning, and velocity in the OFT, consistent with heightened anxiety-like behaviors. Probiotic-treated groups exhibited significant reversal of these deficits, with increased exploratory behavior and reduced anxiety indicators. In the MWM, STZ rats showed prolonged escape latency and impaired probe trial performance, whereas all probiotic groups exhibited shorter escape latencies, increased time spent in the target quadrant, and more platform crossings, indicating enhanced spatial memory. Biochemical analyses revealed that STZ significantly elevated hippocampal MDA levels while reducing GSH, SOD, and CAT activities, confirming oxidative stress. Probiotic supplementation mitigated these alterations, reducing lipid peroxidation and enhancing antioxidant defenses. Among treatment groups, combined supplementation produced the strongest antioxidative effects. Histological evaluations further supported these findings. STZ-induced hippocampal damage was characterized by disorganized cell layers, neuronal shrinkage, and increased glial cell densities in CA1, CA3, and dentate gyrus (DG) regions. Probiotic treatment preserved neuronal morphology, reduced apoptotic features, and decreased astrocyte, ependymal, and microglial densities. Notably, neuronal and oligodendrocyte densities were significantly increased in probiotic-treated groups compared to STZ. However, hippocampal volume loss induced by STZ remained unaltered by probiotic intervention, suggesting partial but not complete structural recovery.

Conclusion : Together, these findings highlight the neuroprotective potential of L. acidophilus and L. reuteri in an AD-like model, mediated through improvements in oxidative balance, neuronal preservation, and reduction of neuroinflammation. While hippocampal volume loss persisted, the overall behavioral, biochemical, and histological improvements underscore the therapeutic promise of probiotics as modulators of the gut–brain axis in neurodegenerative disease. Future preclinical and clinical studies are warranted to optimize probiotic regimens and determine their translational potential for AD prevention and treatment.

Keywords : Alzheimer's Disease; Microbiota; Probiotics; Lactobacillus; Oxidative Stress; Neuroinflammation

Count: 248
Abstract ID: 54
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

PPARγ modulates morphine-induced neuroinflammation: A novel insight into anti-inflammatory mechanisms

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Conclusion : Background: Morphine is the most important opioid that has been widely used for pain relief. Repeated administration of morphine causes functional changes and neural mechanisms in the brain. Also, it has been demonstrated that opioid addiction causes excessive oxidative stress, mitochondrial dysfunctions, and an increase in levels of apoptosis, autophagy, and neuroinflammation. Other studies have shown that morphine induces brain damage and neuronal toxicity Methods: This review study was conducted between 2015 and 2025 by searching scientific databases, including Google Scholar, ScienceDirect, PubMed, Scopus, and Springer, using the keywords PPAR, PPAR agonists, morphine, neuroinflammation, and neurotoxicity. Additionally, a search was performed in SID to study Persian language publications. However, no article directly addressing the role of PPAR in morphine-induced neuroinflammation indicates a significant gap in this field. Results: Recent findings suggest that activation of PPARγ can alleviate morphine-induced neuroinflammation by inhibiting microglial cell activation and modulating levels of inflammatory mediators such as IL-12 and NGF. It may enhance anti-inflammatory cytokines like IL-10. Conclusion: PPARγ agonists have been widely investigated for their therapeutic potential. PPARγ also influences other neural cell types and has demonstrated promising effects in animal models, including improved cognitive functions and reduced neurotoxicity. The interaction between the PPARγ pathway and opioid signaling suggests a novel therapeutic target for managing neuroinflammation associated with opioid addiction. Moreover, preclinical evidence highlights the need for further clinical studies to validate these findings in human settings.

Keywords : Keywords: PPARγ; Neuroinflammation; microglial cells; morphine

Count: 249
Abstract ID: 890
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Evaluatin of Neurobiological Effects of Silver Nanoparticles: From Blood–Brain Barrier Interaction to Neurodegeneration

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Conclusion : Silver nanoparticles (NPs) have emerged as promising tools in medicine, particularly in antimicrobial and anticancer due to their unique physicochemical properties and ability to cross biological barriers. Emerging evidence suggests that nanoparticles can enter the body via ingestion, inhalation, dermal contact, or intravenous routes, and subsequently accumulate in the central nervous system (CNS). This review summarizes current knowledge on the interactions between nanoparticles and neural tissues, focusing on mechanisms of neurotoxicity, blood–brain barrier (BBB) permeability, ionic silver (Ag+) release, oxidative stress, mitochondrial dysfunction, and induction of apoptosis. Silver nanoparticles (AgNPs) can cross the blood–brain barrier (BBB) via passive diffusion or endocytosis, leading to their accumulation in the central nervous system. Their neurotoxicity is linked to inflammation, BBB disruption, and direct injury to neurons and glial cells, with severity influenced by particle size, surface properties, and dosage. In vivo rodent studies have shown that AgNP exposure induces cognitive deficits and synaptic alterations through mechanisms involving oxidative stress, gene dysregulation, and apoptosis. Most studies have examined spherical silver nanoparticles (AgNPs) and their effects on specific brain regions, particularly the hippocampus. Findings indicate that AgNPs, regardless of size, can cross into the brain and induce prolonged neuronal damage lasting several months. Prenatal exposure produces similar neurotoxic outcomes in offspring as direct exposure in adults. AgNPs have also been associated with memory and cognitive impairments, primarily through mechanisms involving neuroinflammation, oxidative stress, and apoptosis. The neurotoxic effects of silver nanoparticles (AgNPs) on rat cerebellar granule cells (CGCs) have been investigated to elucidate their underlying molecular mechanisms. Results demonstrate that AgNPs markedly reduce neuronal viability by inducing apoptosis via oxidative stress and caspase-dependent signaling pathways. Exposure also triggers biochemical, cellular, and molecular changes in the

cerebellar cortex in a dose-dependent manner, primarily through activation of apoptotic pathways, increased oxidative stress, and inflammation. Additionally, AgNP exposure produces cerebellar ataxia-like symptoms in rats, including impaired motor coordination and decreased locomotor activity. This motor dysfunction is associated with reduced expression of CACNA1A, providing insight into the molecular basis of AgNP-induced neurotoxicity. Considering the growing application of silver nanoparticles in medical and commercial products, these findings highlight the need for continued research on their long-term safety. Future investigations should aim to develop strategies to mitigate nanoparticle toxicity—such as optimizing particle size, dosage, and surface properties—to enable their safe use in biomedical applications while minimizing potential risks to neural health and overall human safety.

Keywords : Silver nanoparticles; Neurotoxicity; Central nervous system; Oxidative stress; Apoptosis

Count: 250
Abstract ID: 797
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Guillain-Barré Syndrome Following Organophosphate Poisoning: A Case Report

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Background and Aim : Organophosphate (OP) compounds are widely used pesticides known for causing acute cholinergic toxicity, intermediate syndrome, and organophosphate-induced delayed neuropathy (OPIDN). However, the development of Guillain-Barré Syndrome (GBS) after OP exposure is exceptionally rare. Here, we report one adult case of Guillain-Barré Syndrome (GBS) after OP exposure.

Methods : A 35-year-old male farmer presented with acute cholinergic features (miosis, excessive salivation, sweating, fasciculations, and respiratory distress) two hours after OP exposure. 14 days later, he developed distal paresthesia in both upper and lower limbs Neurological examination revealed flaccid quadriparesis (Medical Research Council grade 2/5 in proximal and 3/5 in distal muscles) and generalized areflexia, with preserved sensory modalities. Therefore, a diagnosis of GBS-acute inflammatory demyelinating polyneuropathy was made.

Results : The literature review highlights the co-existence of OP exposure and GBS and emphasizes its early diagnosis and treatment.

Conclusion : This study reinforces the idea that there is an association between OP exposure and GBS via an autoimmune cross-reactivity mechanism. Close attention should be paid to neurologic complications such as GBS following OP exposure, and early detection of symptoms and diagnosis are important.

Keywords : Guillain-barré syndrome, neurological disease, neuropathy, gbs.

Count: 251
Abstract ID: 26
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Protective Role of Zataria multiflora Boiss Against Paraquat-Induced Neurobehavioral Deficits and Hippocampal Oxidative Damage

Submission Author: Mahboobeh GhasemzadehRahbardar

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Background and Aim : This study explored whether hydroalcoholic extract of Zataria multiflora (ZM) could mitigate oxidative damage in the hippocampus and prevent memory impairments in rats exposed to paraquat (PQ).

Methods : Wistar rats were assigned to a control group receiving saline, paraquat (PQ)-exposed groups subjected to an inhalation protocol (54 mg/m³, eight times, on alternating days). PQ-exposed rats received either low or high doses of ZM extract (ZM-L, ZM-H), pioglitazone (Pio), a combination of ZM-L and Pio, or dexamethasone (Dexa) over a 16-day period. The Morris Water Maze (MWM) and shuttle box tests were employed, alongside assessments of hippocampal oxidative stress markers and both total and differential white blood cell (WBC) counts.

Results : PQ markedly elevated total and differential WBC counts, hippocampal MDA levels, escape latency, and path length in MWM, while concurrently diminishing CAT, SOD, and thiol levels, as well as time latency following electric shock (24–72 hours). Treatment with ZM-L,

ZM-H, Pio+ZM-L, or Dexa significantly attenuated PQ-induced changes in leukocyte profiles and oxidative stress markers. ZM-H, Pio + ZM-L, and Dexa also improved spatial learning and memory performance, evidenced by reduced escape latency across days 2–5, shorter traveled distances on day 3, and prolonged retention latency post-shock. Among the interventions, ZM-H demonstrated superior protective effects across most endpoints compared to ZM-L, and the combined Pio+ZM-L regimen outperformed either agent alone in select parameters.

Conclusion : Prophylactic administration of ZM attenuated PQ-induced oxidative damage within the hippocampus and ameliorated associated memory deficits. Moreover, Pio+ZM-L elicited a synergistic neuroprotective effect, suggesting a contributory role of PPARγ-dependent mechanisms in mediating the cognitive and antioxidant benefits conferred by ZM.

Keywords : Paraquat, Pioglitazone, Zataria multiflora, PPAR gamma, Systemic oxidative stress, Hippocampus

Count: 252
Abstract ID: 463
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Pericytes and Glial Cells: Key Regulators of Neurovascular Function and Dysfunction

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Conclusion : Pericytes, as multifunctional mural cells in the central nervous system (CNS), interact closely with glial cells and contribute to the regulation of neurovascular homeostasis. Understanding their reciprocal communication is crucial for deciphering mechanisms of both brain health and disease. A comprehensive review of experimental and clinical studies was conducted, focusing on the molecular and cellular interactions between pericytes and glial cells, including astrocytes, microglia, and oligodendrocytes, under physiological and pathological conditions. Pericytes influence astrocyte polarization, oligodendrocyte precursor cell differentiation, and microglial immune responses. These interactions are fundamental to angiogenesis, blood-brain barrier (BBB) integrity, cerebral blood flow regulation, and immune surveillance. Disruptions in pericyte-glial crosstalk contribute to BBB breakdown, impaired clearance systems, and neuroinflammation, thereby exacerbating neurodegenerative processes such as neuronal loss, synaptic dysfunction, and defective repair mechanisms. Pericyte-glial interactions are central to maintaining neurovascular stability and brain function. Their dysregulation plays a pivotal role in the pathogenesis of neurological and neurodegenerative disorders. Targeting these interactions offers promising therapeutic opportunities for restoring BBB integrity and

Keywords : Pericyte; Glial cells; Blood-brain barrier; Neuroinflammation; Neurovascular unit.

Count: 253
Abstract ID: 366
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Sleep Disorders in Parkinson's disease: A Comprehensive Review

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Conclusion : Parkinson's disease (PD) is widely recognized as a progressive neurodegenerative disorder primarily characterized by motor symptoms such as tremor, rigidity, and bradykinesia. However, a growing body of evidence highlights that non-motor manifestations, particularly sleep disturbances, can precede motor symptoms and profoundly affect patients' daily functioning and quality of life. Sleep disorders in PD are diverse, encompassing insomnia, excessive daytime sleepiness (EDS), REM sleep behavior disorder (RBD), restless legs syndrome (RLS), and obstructive sleep apnea (OSA). Beyond their symptomatic impact, these disturbances can exacerbate cognitive impairment, mood disorders, and overall disease progression. Investigating sleep disturbances in PD is not only critical for improving symptomatic management but also offers insight into the underlying pathophysiology of the disease, including dopaminergic and non-dopaminergic neural circuits. Recent research has suggested that certain sleep disorders, particularly RBD, may act as early biomarkers for neurodegeneration, emerging years before the classic motor symptoms appear. Understanding these relationships provides opportunities for early diagnosis, personalized interventions, and improved patient outcomes. Methods This review synthesizes evidence from studies published between 2023 and 2025, focusing on the prevalence, mechanisms, and management of sleep disturbances in PD. Comprehensive literature searches were conducted in PubMed, ScienceDirect, and SpringerLink, emphasizing longitudinal and cohort studies, meta-analyses, and clinical trials. Studies were selected based on their relevance to objectively measured sleep outcomes and their examination of neurobiological, pharmacological, and behavioral aspects of sleep disorders in PD patients. Discussion Sleep disturbances affect the majority of PD patients, significantly diminishing quality of life. Insomnia is common, often linked to both disease progression and dopaminergic therapy. Excessive daytime sleepiness is frequently reported and can complicate daily functioning and safety. RBD represents a particularly intriguing phenomenon, often preceding the onset of motor symptoms by several years, suggesting its utility as a potential early biomarker of neurodegeneration. RLS and OSA, while less specific to PD, can further compound sleep fragmentation and contribute to fatigue and cognitive impairment. The pathophysiology of sleep disturbances in PD is multifactorial.

Dopaminergic dysfunction, particularly in the basal ganglia, plays a central role in regulating sleep-wake cycles, while neurodegeneration in brainstem nuclei involved in REM sleep contributes to RBD. Autonomic dysfunction and circadian rhythm alterations further exacerbate sleep disruptions. Importantly, the interplay of these mechanisms explains the heterogeneity of sleep problems observed across patients. Management requires individualized strategies, combining pharmacological approaches, such as dopaminergic medications, melatonin, or clonazepam, with behavioral interventions, including sleep hygiene, cognitive-behavioral therapy, and structured physical activity. Conclusion Sleep disturbances in Parkinson's disease are highly prevalent and multifaceted, impacting both clinical outcomes and patient quality of life. Recognition and comprehensive management of these disorders are essential components of PD care. Research continues to uncover the neurobiological underpinnings of sleep disturbances and their potential as early indicators of neurodegeneration, highlighting the importance of integrating sleep assessments into routine clinical practice.

Keywords : Parkinson's disease, sleep disorders, insomnia, REM sleep behavior disorder, restless legs syndrome, obstructive sleep apnea, neurodegeneration

Count: 254
Abstract ID: 710
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Combination therapy of hair follicle stem cells and edaravone in a rat model of Alzheimer's diseases induced by streptozotocin

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Background and Aim : Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline, neuroinflammation, and impaired neurotrophic signaling. Current pharmacological treatments remain largely symptomatic. Stem cell–based therapies offer neuroregenerative potential through trophic factor secretion, while edaravone, a potent free radical scavenger, provides antioxidant and neuroprotective effects. The combined therapeutic potential of hair follicle stem cells (HFSCs) and edaravone in AD-like neurodegeneration has not been fully investigated. This study aimed to evaluate the effects of HFSCs and edaravone—alone and in combination—on behavioral performance and hippocampal gene expression in the intracerebroventricular streptozotocin (ICV-STZ) rat model of Alzheimer's disease.

Methods : Adult male Sprague-Dawley rats (250–300 g) were randomly divided into five groups: control, STZ, STZ+HFSCs, STZ+Edaravone, and STZ+HFSCs+Edaravone. AD-like pathology was induced by a single ICV injection of streptozotocin (3 mg/kg). HFSCs (1×10^6 cells) were intravenously administered on days 4, 14, and 21 post-surgery. Edaravone was injected intraperitoneally every other day during the treatment period. Behavioral assessments, including the Morris water maze and passive avoidance tests, were performed from day 28 to evaluate learning and memory. Hippocampal tissues were collected for quantitative PCR analysis of BDNF, VEGF, and IL-6 gene expression.

Results : STZ administration caused significant cognitive deficits and increased hippocampal expression of BDNF, VEGF, and IL-6. The mild elevation of neurotrophic factors in the STZ group likely reflects a compensatory response to neuronal injury. Treatment with HFSCs or edaravone alone improved behavioral performance, increased BDNF or reduced IL-6 expression, respectively. The combined therapy produced the strongest effects—enhancing BDNF and VEGF while markedly suppressing IL-6—correlating with improved memory.

Conclusion : Combined HFSC and edaravone therapy exerted synergistic neuroprotective and anti-inflammatory effects in the ICV-STZ model, suggesting that integrating stem cell–based

and antioxidant strategies may offer a promising approach for mitigating neurodegeneration in Alzheimer's disease.

Keywords : Stem cells; Edaravone; Alzheimer Disease; Streptozotocin; Memory; Inflammation.

Count: 255
Abstract ID: 764
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Involvement of NF-κB and TNF-α, IL-6 Signaling in the Neuroprotective and Anti-Parkinsonian Effects of Vitamin D_3 in the 6-OHDA-Induced Rat Model of Parkinson's Disease

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Background and Aim : Neuroinflammation is a core driver in the progression of Parkinson's disease (PD). Key proinflammatory mediators include NF-κB, IL-6, and TNF-α, whose elevated levels are consistently found in PD. Microglial activation in the substantia nigra initiates the NF-κB-driven production of these cytokines. Vitamin D3 (cholecalciferol), a neuroactive compound, possesses anti-inflammatory properties, acting through the VDR in glial cells. It is proposed that Vitamin D3 shifts microglia from a proinflammatory (M1) to a neuroprotective (M2) phenotype, thereby attenuating cytokine release and modulating neuroinflammation signaling in PD.

Methods : Thirty-two adult male Wistar rats were divided into four groups: Control (CO), Parkinsonian (PD), Parkinsonian + Vitamin D3 treatment (PD+VD), and Vitamin D3 control (VD). Animals in the PD and PD + VD groups underwent stereotaxic surgery, in which 6-OHDA (8 µg/µL) was unilaterally injected into two sites of the right medial forebrain bundle. The PD+VD and CO VD groups received an intraperitoneal dose of 1 µg/kg Vitamin D3 one hour before surgery. Apomorphine-induced rotation tests were performed at baseline, week 2, and week 8 post-surgery. After euthanasia, protein expression levels of NF-κB, TNF-α, and IL-6 in the striatum of the lesioned side were quantified using Western blot analysis.

Results : Administration of Vitamin D3 yielded significant beneficial effects on both the behavioral and molecular outcomes. Behaviorally, the PD group exhibited a significantly higher number of apomorphine-induced rotations compared to the PD+VD group at both week 2 ($p<0.01$) and week 8 ($p<0.001$), demonstrating that Vitamin D3 effectively attenuated the onset and progression of PD. Furthermore, the progression of rotations (week 2 to week 8) was highly significant in the untreated PD group ($p<0.001$) but it was not significant in the PD+VD group. At the molecular level, Western blot analysis confirmed the induction of neuroinflammation in the PD group with significantly elevated levels of NF-?B ($p<0.05$), TNF-?, and IL-6 ($p<0.01$). Crucially, comparison between the PD and PD+VD groups revealed a significant reduction in all three inflammatory markers (NF-?B, TNF-?, and IL-6; $p<0.05$) following Vitamin D3 treatment. These findings strongly indicate that the antiparkinsonism and neuroprotective effect of Vitamin D3 is directly correlated with its capacity to mitigate NF-?B activation and subsequently dampen TNF-? and IL-6 signaling pathways.

Conclusion : This study provides robust evidence that Vitamin D3 modulates neuroinflammation in the 6-OHDA-lesioned rat model of PD. The treatment was associated with a reduction in TNF-α, IL-6, and NF-κB expression, highlighting its modulatory effect on key inflammatory signaling pathways implicated in dopaminergic neurodegeneration. Behavioral assessments correlated these molecular findings, demonstrating that Vitamin D3 attenuated both the initial neuroinflammatory response and the subsequent progression of the disease. These results reinforce the potential of Vitamin D3 as an adjunct therapeutic strategy for neurodegenerative disorders by dampening neuroinflammation. Further studies are suggested to elucidate precise molecular targets and optimize dosing for potential clinical application.

Keywords : Parkinson’s disease; Vitamin D_3; 6-hydroxydopamine; TNF-α; Interleukin-6; NF-κB; Neuroprotection.

Count: 256
Abstract ID: 968
subject: Neural Injuries and Neurodegenerative Disorders: Neurotoxicity, Neuroprotection, Inflammation
Presentation Type: Poster

Neuroprotective Role of CoQ10 Against Manganese-induced Inflammation and Apoptosis

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Background and Aim : Manganese is essential for metabolic and neural functions; however, elevated levels can impair physiological processes by activating inflammatory and apoptotic pathways.This study investigates whether Coenzyme Q10 can counteract hippocampal damage caused by manganese overload, based on its proven anti-inflammatory and neuroprotective properties.

Methods : A total of 48 male Wistar rats were enrolled in this experimental study. For 28 consecutive days, rats were treated with manganese(30 mg/kg) and CoQ10 (50 or 200 mg/kg) administered orally via gavage. Then, following anesthesia induction and transcardial perfusion, brain tissue was collected and Inflammatory markers(TNF-α, IL-17 and IL-10) were assessed via ELISA, and apoptosis was evaluated through caspase-3 activity and Nissl staining.

Results : Coenzyme Q10 treatment reduced the levels of pro-inflammatory markers TNF-α and IL-17, while increasing the anti-inflammatory cytokine IL-10. It also decreased caspase-3 activity and reduced the number of dark neurons in the CA1 region of the hippocampus.

Conclusion : Coenzyme Q10 significantly attenuated manganese-induced neuroinflammationand apoptosis. These results underscore its potential as an effective neuroprotective agent.

Keywords : Manganese, Coenzyme Q10, Inflammation, Apoptosis

Count: 257
Abstract ID: 566
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Poster

Isorhamnetin Attenuates Hypothalamic-Pituitary-Gonadal Axis Disruption and Restored Testicular Function Following Cerebral Ischemia

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Background and Aim : Cerebral ischemia, particularly resulting from middle cerebral artery occlusion (MCAO), is a major cause of neurological disability and systemic complications, including disruption of the hypothalamic-pituitary-gonadal (HPG) axis and impaired testicular function. The hypothalamus is central to reproductive regulation, and its injury can result in hormonal imbalances and reduced fertility. Isorhamnetin, a natural flavonoid, is known for its anti-inflammatory and neuroprotective effects. This study aimed to investigate the protective effects of isorhamnetin on brain inflammation and testicular function following cerebral ischemia in rats.

Methods : Thirty male Wistar rats (12 weeks old, 270–300 g) were randomly assigned to three groups: sham, ischemia (MCAO for 60 minutes), and isorhamnetin-treated (5 mg/kg IP at 24, 48, and 72 hours post-ischemia). Neurobehavioral assessments and TTC staining verified the ischemia model. Six days after induction, molecular analyses were performed. Brain tissue was examined for IL1β and IL6 gene expression by qPCR, and testicular tissue for Prm1 and Prm2 expression. Serum levels of LH and FSH were measured by ELISA. Data were analyzed using ANOVA and appropriate post hoc tests.

Results : MCAO resulted in significant neurobehavioral deficits and increased brain IL1β and IL6 expression compared to the sham group ($p<0.05$). Serum LH and FSH levels, as well as Prm1 and Prm2 gene expression in testicular tissue, were significantly reduced in the ischemia group ($p<0.05$). Isorhamnetin treatment attenuated neuroinflammation, partially restored hormonal levels, and improved spermatogenesis gene expression, resulting in improved testicular function compared to the untreated ischemia group ($p<0.05$).

Conclusion : Isorhamnetin demonstrates a protective and therapeutic role in reproductive dysfunction caused by cerebral ischemia, likely through its anti-inflammatory and

neuroprotective effects on the HPG axis and testicular tissue. These findings suggest that isorhamnetin may be a promising candidate for reducing systemic complications following stroke.

Keywords : Isorhamnetin, Cerebral Ischemia, Hypothalamic-Pituitary-Gonadal Axis, Testicular Function, Neuroinflammation, Spermatogenesis

Count: 258
Abstract ID: 142
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Poster

Impairments of active/passive verbs in aphasia

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Background and Aim : Voice in grammar can be roughly divided into active and passive. In active form, the subject performs the action and the object receives it, e.g., “The cat chased the mouse”. In passive voice the subject of the sentence is not mentioned and the action is attributed to the object, e.g., “The mouse was chased”. According to the Trace Deletion Hypothesis (TDH), people with aphasia (PWA) use an agent-first processing strategy as they cannot generate accurate syntactic representations of passive sentences. In this study we aimed to compare the production of passive verbs in PWA compared to Non-Brain-Damaged (NBD) Persian-speakers

Methods : the forty-five participants included 11 fluent PWA (5 males and 6 females; mean age=58.27 years, mean post-onset time (POT)=18.73 months), 9 non-fluent PWA (6 males and 3 females; mean age=53.33 years, mean POT=81.11months), and 25 NBD individuals (5 males and 20 females; mean age=28.9 years). The Persian Narrative Discourse Test (PNDT) (Ghayoumi et al., 2015), Picture description task (Nilipour et al., 2016), and open-ended questions were used to elicit speech samples. Percentages of produced active and passive verbs were calculated. The non-parametric Kruskal-Wallis test and the Mann-Whitney U test as post hoc were applied. The inter-rater and intra-rater reliability was calculated for 25% of the speech samples.

Results : The inter-rater (r=98%, P<0.001) and intra-rater (r=99%, P<0.001) reliability proved to be strong. As it was expected, the NBD group possessed the greatest number of produced verbs which was significantly different from our non-fluent PWAs (mean percentage: NBD=27.92, fluent PWA=22.91, non-fluent PWA=9.44; P-value=0.001; NBD>non-fluent; Fluent>non-fluent).The comparison of mean percentages regarding passive verbs showed that the NBD group produced more passive verbs than both groups of PWAs (mean percentage: NBD=29.32, fluent PWA=16.45, non-fluent PWA=13.44; P-value=<0.001; NBD>non-fluent

and fluent). Comparing the mean percentage of active verbs revealed that there was no significant difference between the three groups (mean percentage: NBD=23.16, fluent PWA=22.09, non-fluent PWA=23.67; P-value= 0.961).

Conclusion : Our results seemed to support the TDH hypothesis. In generative syntax, when an element is moved in a sentence (in passive sentences, the object moves to the subject position), a trace remains. The healthy brain captures this movement easily, yet, PWA have problems with processing of movements. That is, they cannot make a connection between the shifted position and the remaining trace. Further, the frequency effect may be at work here. Since active voice is more frequent in Persian like many other languages, they are easier to retrieve, while passive verbs have lower frequency.

Keywords : aphasia; passive voice; active voice; verb retrieval; Persian

Count: 259
Abstract ID: 553
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Oral

Sex Matters in a Rat Model of Cognitive Impairment Driven by Progressive Carotid Stenosis

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Background and Aim : Vascular cognitive impairment (VCI) is a common dementia resulting from brain ischemia. Existing rodent models, such as bilateral carotid artery stenosis, rely on mechanical constriction to induce constant hypoperfusion. These models fail to replicate the progressive vascular pathology, such as plaque formation, central to human VCI. Recent evidence implicating vascular wall pathology in cognitive decline highlights the need for more translatable models. This study introduces a novel rat model featuring progressive cerebral hypoperfusion with neointimal hyperplasia and directly compares it to the traditional constant hypoperfusion model (microcoils) to elucidate VCI mechanisms.

Methods : Adult Wistar rats of both sexes (12 weeks old) were allocated to one of three experimental groups: sham-operated controls, constant bilateral carotid stenosis (CBCS), or progressive bilateral carotid stenosis (PBCS). The CBCS was established by positioning microcoils (0.18 mm internal diameter) around both common carotid arteries. To model PBCS, neointimal hyperplasia was induced via endothelial denudation of the common carotid arteries using an F2 angioplasty balloon. Following denudation, the arteriotomy site was repaired with a 10-0 suture, and blood flow was reperfused. In sham-control animals, the carotid arteries were exposed and subjected to alternating clamping for a period matching the surgical duration of the experimental groups, after which circulation was reinstated. The percentage of carotid blood flow was measured using laser Doppler flowmetry. Cognitive impairment was evaluated using the Y-maze test on postoperative days 7, 14, and 28. Concurrently, specimens from the carotid arteries and brain tissue were harvested for subsequent histopathological analysis. Data

were analyzed by two-way ANOVA for variables of the group and sex. Tukey's test was performed for post hoc comparisons.

Results : Behavioral assessment in the Y-maze revealed a sex- and time-dependent cognitive decline. In the CBCS group, the spontaneous alternation score was significantly reduced on post-operative days 14 and 28 in both males and females compared to sham controls. In contrast, within the PBCS group, a significant reduction in this score was observed exclusively in male rats on day 28. Histopathological evaluation of the carotid arteries demonstrated a progressive increase in wall thickness in both male and female PBCS rats throughout the study period. This finding was consistent with the concurrent laser Doppler flowmetry measurements, confirming progressive hypoperfusion. In the hippocampal CA1 region, morphological analysis showed significant neuronal injury in CBCS rats. On days 14 and 28, these animals exhibited a marked increase in the number of damaged cells and a concomitant decrease in neuronal density in both sexes. Conversely, in the PBCS group, a significant reduction in CA1 neuronal density was found only in male rats on day 28, with no statistically significant difference observed in the count of damaged cells compared to controls.

Conclusion : We present a translatable model of VCI that recapitulates progressive carotid stenosis. The stark contrast in outcomes between PBCS and CBCS, particularly the male-specific vulnerability in the progressive model, reveals a critical sex-dimorphism in VCI pathogenesis. This model is a vital tool for investigating sex-specific mechanisms and therapies.

Keywords : Carotid Stenosis; Cerebrovascular Circulation; Cognitive Dysfunction; Disease Models; Sex Characteristics

Count: 260
Abstract ID: 577
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Oral

Advancing Brain Stroke Diagnosis and Monitoring through Raman Spectroscopy

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Conclusion : Introduction Stroke remains a leading cause of global mortality and long-term disability, highlighting the need for rapid and accurate diagnostic tools to enable effective intervention. While current neuroimaging techniques, such as CT and MRI, are essential, they have limitations in terms of speed, portability, and the ability to provide molecular-level information. This review examines the emerging use of Raman spectroscopy, a label-free optical technique, as a powerful tool for stroke management. Raman spectroscopy detects unique molecular vibrations, generating a "chemical fingerprint" of tissues. This capability could revolutionize the detection, characterization, and monitoring of cerebral ischemia and hemorrhage at a biochemical level. Materials and Methods This review synthesizes findings from in vitro studies, animal models, and early-stage human tissue analyses. The core methodology involves shining a monochromatic laser, typically in the near-infrared range, to minimize tissue damage and fluorescence, onto a brain tissue sample or the scalp. The inelastically scattered Raman photons are then collected by a spectrometer. Key analytical steps include preprocessing the raw spectra to remove background fluorescence and noise, followed by multivariate statistical analysis, such as Principal Component Analysis (PCA), or machine learning algorithms. These techniques are used to identify specific spectral biomarkers that differentiate ischemic core, penumbra, and healthy brain matter based on their distinct biochemical compositions, including changes in lipids, proteins, and nucleic acids. Results Research consistently shows that Raman spectroscopy can effectively differentiate between ischemic and hemorrhagic strokes. This technique identifies specific biochemical changes, such as a decrease in lipid content and an increase in signals from proteins and nucleic acids, which are characteristic of neuronal cell death in the ischemic core. Importantly, studies indicate that it can distinguish between the ischemic penumbra and the irreversibly damaged core, a vital distinction for guiding treatment decisions. Additionally, advancements in fiber-optic probes are enabling potential in vivo and intraoperative applications, providing real-time tissue characterization. Conclusion Raman spectroscopy represents a significant advance in stroke diagnostics, offering rapid and molecular-specific analysis. Its real-time biochemical

insights could enhance diagnosis speed and help identify salvageable brain tissue. Future efforts should concentrate on large-scale human trials, creating portable systems for clinical use, and integrating Raman technology into current surgical and diagnostic workflows to improve patient outcomes.

Keywords : Brain Stroke, Raman spectroscopy, Principal Component Analysis, Machine learning, Algorithms

Count: 261
Abstract ID: 230
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Poster

Pre conditioning with Daphnetin Attenuates Brain Edema and BBB Permeability in a Mouse Model of Global Ischemia

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Background and Aim : Daphnetin (DAP), a natural coumarin derivative, exhibits potent neuroprotective, antioxidant, and anti-inflammatory effects in various models of cerebral ischemia/reperfusion (I/R). Our prior in vivo studies demonstrated that DAP administered before ischemia has potential as a therapeutic agent for cerebral ischemia. Despite these findings, the effects of daphnetin pre-treatment on brain edema and BBB permeability remain unclear.
Methods : Methods: In this study, Cerebral ischemia was induced by 30 min occlusion of bilateral common carotid occlusion (BCCAO) followed by 48 h reperfusion. The effects of DAP (40 mg/kg) administered for one week and a single dose 30 minutes before ischemia on brain edema and BBB disruption in Sham, Ischemic control, and DAP treatment groups were evaluated. Sixty adult male albino mice were randomized into six groups. Brain Edema Measured by cerebral water content (%) using the wet–dry method, and BBB permeability was determined by measuring Evans blue (E.B.) dye leakage.
Results : Results: The prophylactic administration of for one week prior to ischemia leads to a significant reduction in brain water content and BBB permeability compared to the ischemic control treatment ($P < 0.05$). Even a single dose of DAP injection before ischemia results in a remarkable reduction in brain edema ($P < 0.05$), although it does not significantly affect BBB permeability compared to the ischemic controls ($P > 0.05$).
Conclusion : Conclusion: The findings of the current study indicated that DAP administered one week before ischemia diminishes BBB permeability and brain edema. Although daphnetin pre-treatment 30 minutes before ischemia significantly reduced cerebral edema, it had no effect on BBB permeability. The result of this study shows the potential effect of pre-conditioning of DAP as a neuroprotective strategy against global cerebral I/R injury.
Keywords : Daphnetin, Cerebral ischemia, Brain edema, BBB, BCCAO, Mice

Count: 262
Abstract ID: 867
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Poster

The Role of BDNF in Stem Cell-Derived Exosome Therapy for Ischemic Stroke

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Conclusion : Ischemic stroke represents the most common type of cerebrovascular disease and is associated with high rates of morbidity, long-term disability, and mortality. Despite its global impact, effective strategies for prevention and treatment remain limited. In recent years, stem cells transplantation has emerged as a promising therapeutic approach for stroke. However, this cell-based therapy carries certain risks, such as tumorigenesis, coagulation abnormalities, and vascular obstruction. Accumulating evidence indicates that the beneficial effects observed following stem cells transplantation are largely mediated by exosomes derived from stem cells. Unlike traditional cell therapy, this cell-free approach may overcome many of the associated risks and technical challenges, making it a particularly attractive strategy for stroke treatment. Brain-derived neurotrophic factor (BDNF), the most abundant neurotrophin in the adult brain, functions as a key neurotrophic signal and neuromodulator. It is widely expressed throughout the central nervous system (CNS), where it plays an essential role in promoting neuronal survival, differentiation, and growth, thereby providing robust neuroprotection. A growing body of research highlights the pivotal role of BDNF in the pathophysiology of neurological disorders. In fact, reduced levels of BDNF in both central and peripheral systems have been closely linked to a variety of neurogenic diseases, including stroke. Multiple studies have shown that BDNF contributes significantly to the therapeutic potential of stem cells and their exosomes. Therefore, this review aims to explore current knowledge on the role of BDNF in the therapeutic effects of stem cells and their exosomes in ischemic stroke, providing novel perspectives and potential strategies for clinical application

Keywords : BDNF; Ischemic stroke; Mesenchymal stem cells; Exosomes

Count: 263
Abstract ID: 379
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Poster

Evaluation of memory change and oxidative stress in hippocampus following unilateral hemorrhagic stroke in striatum

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Background and Aim : Hemorrhagic stroke (HS) accounts for approximately 15 percent of all brain strokes but contributes disproportionately to mortality and long-term disability. Besides the lack of fully promised treatment of first-hand injuries, there is also secondary neurodegeneration damage to struggle with due to anatomical axonal connections. First-hand and secondary neurodegeneration damages of HS can vary based on topology of HS occurrence. Clinical observations indicate that nearly two-thirds of patients with striatal hemorrhagic stroke (SHS) demonstrate cognitive impairments, particularly memory deficits in the long term. Growing evidence supports that the striatum's extensive connections with the hippocampus arise the oxidative stress and apoptosis as central mediators of secondary neuronal degeneration in the hippocampus after SHS. In this study, we investigate to declare the weight of alteration of oxidative agents and hippocampus-dependent memory deficit after unilateral SHS by performing behavioral and oxidative stress tests.

Methods : A total of 48 Wistar mature male rats, housed in a standard laboratory environment, were randomly assigned into 6 even groups of three sham-operated groups with sample obtainment in day 7, 14 and 21 after operation (surgery exposure, no-SHS induction), and three SHS-induced groups with sample obtainment in day 7,14 and 21 after intervention (SHS induction with 100µl autologous blood injected in the left striatum under anesthesia). All groups performed Neurological Deficit Score (NDS) and Wire Hanging (WH) test to evaluate the validity of SHS induction method. Memory and behavioral performance assessed at days 7, 14, and 21 post-operation by executing Novel Object Recognition (NOR) and Active Avoidance Shuttle Box (ShB) tests. Following behavioral tests, the Hippocampus samples were derived for Thiol and Malondialdehyde (MDA) biochemistry tests.

Results : NDS and WH results showed distinct neurological impairment and confirmed the validity of SHS induction. In NDS and WH tests, all SHS groups had a drastic reduction in

neurological functions compared to sham groups. As behavioral assessment, ShB Test also demonstrate a dramatic latency time reduction in SHS groups compared to sham groups. The results of NOR showed a tens reduction in Discrimination Index by all SHS groups in contrast of sham groups. In biochemistry tests, all SHS groups had a decline in level of thiol in Hippocampus in comparison to the sham groups. In MDA measurement, a slight increase was seen in the level of MDA in SHS-day-14 group comparing to the sham-day-14 group, while MDA level were decreased in SHS-day-7 and SHS-day-21 in comparison of pair sham groups.

Conclusion : In biochemical tests, reduction in Thiol level in Hippocampus was noteworthy and confirms that SHS in somehow can suppress anti-oxidative agent levels in hippocampus and therefore, causes neurological damages. On the other hand, results from MDA measurement were not consistent enough to be in favor of our study; But In the behavioral view both NOR and ShB test indicated that SHS can cause significant hippocampus-dependent memory deficiencies. However further apoptotic genetical and microbiological studies are needed to determine background mechanisms of this subject.

Keywords : Oxidative Stress, Hippocampus, Learning and Memory, Hemorrhagic Stroke

Count: 264
Abstract ID: 810
subject: Neural Injuries and Neurodegenerative Disorders: Ischemia, Stroke, and Neurovascular Disorders
Presentation Type: Poster

Method for Inducing Ischemic Stroke in Rats with Continuous Cerebral Blood Flow Monitoring

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Background and Aim : Ischemic stroke, a major cause of global mortality and disability, lacks effective treatments due to its complex pathophysiology despite extensive research. Current investigations increasingly focus on identifying key contributing factors and developing novel therapeutic strategies for cerebral ischemia. The present study aims to establish an experimental rat model of ischemic stroke characterized by a higher induction success rate and reduced mortality.

Methods : Thirty two male Sprague–Dawley rats were randomly assigned to four experimental groups. Focal cerebral ischemia was established using the intraluminal filament technique, in which a silicon-coated nylon filament was inserted to occlude the middle cerebral artery (MCA). Regional cerebral blood flow (rCBF) was continuously monitored by laser Doppler flowmetry to verify and guide filament placement within the cerebral vasculature. Following 90 minutes of ischemia and subsequent 24 hours of reperfusion, neurological function, infarct volume, and brain edema were evaluated. Neurological deficits were evaluated using the modified Neurological Severity Score (mNSS) test. Coronal brain sections were stained with 2,3,5-triphenyltetrazolium chloride (TTC), digitized, and quantitatively analyzed using an image analysis system. Cerebral edema was quantified using the wet-dry method.

Results : Induction of focal cerebral ischemia produced significant neurological deficits in the ischemic group, whereas sham-operated rats exhibited no neurological impairment or infarction. The mean infarct volume in the left (ischemic) hemisphere was 402 ± 43 mm3, with 62.7% of the lesion localized to cortical regions. Ischemic rats also showed a significant elevation in brain water content (84 ± 0.23 %) compared with both hemispheres of the sham group and the contralateral hemisphere of the ischemic group ($p < 0.001$). The ischemia induction success rate was 100%, and no procedure-related mortality occurred

Conclusion : Our findings demonstrate that continuous monitoring of regional cerebral blood flow using laser Doppler flowmetry markedly improved the success rate and reduced mortality in this rat model of focal cerebral ischemia. This reliable and reproducible model, characterized by high procedural success and minimal technical limitations, provides a valuable platform for investigating the pathophysiology of cerebral ischemia and for evaluating novel therapeutic interventions.

Keywords : Ischemic Stroke; Brain Edema; Cerebral Blood Flow; Laser Doppler Flowmeter; Rat

Count: 265
Abstract ID: 1020
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Mitochondrial Dysfunction and Therapeutic Targeting in Multiple Sclerosis: A Systematic Review of Experimental and Clinical Evidence

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Conclusion : Abstract Background: Multiple sclerosis (MS) is a chronic autoimmune demyelinating disease of the central nervous system, characterized by inflammation, axonal loss, and neurodegeneration. Increasing evidence suggests that mitochondrial dysfunction plays a central role in MS pathogenesis, causing energy depletion, oxidative stress, and neuronal damage. Alterations in mitochondrial structure and metabolism have been observed in neurons, oligodendrocytes, and immune cells, highlighting their widespread involvement in disease progression. The aim of this systematic review is to evaluate the role of mitochondrial dysfunction in MS and assess the potential of mitochondria-targeted interventions to preserve neuronal function and modulate disease outcomes. Methods: Following the PRISMA 2020 guidelines, a comprehensive search was conducted in PubMed, Scopus, Web of Science, and Embase for studies published up to 2025. A total of 1,391 records were identified, and 30 studies met the predefined inclusion criteria. These studies included both preclinical models and clinical research related to multiple sclerosis (MS), focusing specifically on mitochondrial function or interventions aimed at improving mitochondrial activity. Only original research articles published in English were included. Data extraction included study design, experimental model or patient population, mitochondrial outcomes, intervention details, and main findings. Studies that did not directly assess mitochondrial function were excluded. Results:In total, 30 studies met the predefined inclusion criteria. Mitochondrial dysfunction was consistently observed across neuronal and glial populations, characterized by reduced ATP production, increased reactive oxygen species (ROS), and disrupted mitochondrial dynamics. Preclinical interventions, including SkQ1 antioxidant treatment, PGC-1α activation, USP30 inhibition, SNPH deletion, and mesenchymal stem cell transplantation, effectively restored mitochondrial function, reduced oxidative stress, and promoted remyelination. Clinical studies demonstrated altered mitochondrial respiration and metabolic reprogramming in T cells,

PBMCs, and skeletal muscle, correlating with disease severity and fatigue. Therapeutic interventions such as Glatiramer acetate and Teriflunomide improved mitochondrial efficiency and antioxidant defenses, while Idebenone and Fluoxetine showed modest but measurable effects. Metabolomic and neuroimaging analyses confirmed systemic bioenergetic deficits, and markers such as ER stress (Rab32), cerebrospinal fluid ceramide accumulation, and vascular risk factors were associated with neuronal mitochondrial dysfunction and disease progression. Conclusion: Mitochondrial dysfunction is a key contributor to MS pathology, particularly in axonal degeneration and progressive disease stages. Preclinical evidence supports the potential of mitochondria-targeted therapies, but translation to clinical benefit remains limited. Future studies should focus on restoring mitochondrial function and evaluating its impact on neurodegeneration and disease progression in MS.Mitochondrial impairment represents a fundamental mechanism in MS pathophysiology. Targeting mitochondrial pathways, oxidative phosphorylation, and bioenergetic homeostasis offers a promising therapeutic approach especially in progressive disease stages. However, further human trials integrating mitochondrial biomarkers are warranted to confirm translational relevance.

Keywords : Multiple Sclerosis, Mitochondrial dysfunction, Oxidative stress, Neuroinflammation, Antioxidants, Energy metabolism

Count: 266
Abstract ID: 640
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Oral

Investigating the effect of quercetin loaded on UIO-66 metal-organic framework on the expression of oxidative stress factors and myelin repair of brain tissue in cuprizone-induced demyelination model

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Background and Aim : Multiple sclerosis (MS) is a chronic autoimmune disorder characterized by demyelination and severe physical and cognitive impairment. Quercetin is a flavonoid with potent anti-inflammatory and antioxidant properties, but its clinical application is limited by poor solubility and stability. UiO-66, a zirconium-based metal–organic framework (MOF) with high biocompatibility, large surface area, and tunable functionality, offers an efficient drug delivery platform. This study aimed to evaluate quercetin-loaded UiO-66 (UiO-66-Qu) in terms of physicochemical characteristics, oxidative stress markers, neuroinflammation and myelin repair in a cuprizone-induced demyelination model.

Methods : Forty male C57BL/6 mice (20–25 g) were randomized into five groups (n = 8 each): intact control, disease + saline, UiO-66 alone, free quercetin, and UiO-66-Qu. Demyelination was induced by feeding 0.2 % cuprizone for eight weeks, followed by two weeks of daily intraperitoneal injections (25 mg/kg). Behavioral assessments (including: Y-maze spontaneous alternation, Novel Arm Discrimination Test (NADT), and wire hang) were performed on days 0, 42, and 56. UiO-66 and UiO-66-Qu were characterized by field emission scanning electron microscopy (FE-SEM), energy-dispersive X-ray spectroscopy (EDX), and X-ray diffraction (XRD); quercetin loading efficiency was quantified via high-performance liquid chromatography (HPLC). After behavioral testing, brains were harvested for qRT-PCR analysis of inflammatory (TNF-α, IL-1β, IL-6), oligodendrocyte (MBP, Olig2), and antioxidant (Nrf-2, HO-1) gene expression and Biochemical assays for total antioxidant capacity(TAC), antioxidant enzymes—glutathione peroxidase (GPx), catalase (CAT), superoxide dismutase (SOD)—and malondialdehyde (MDA) levels. Statistical analyses included two-way and one-way ANOVA with Bonferroni and Tukey post-hoc tests; p 0.05 was considered significant.

Results : FE-SEM, EDX, and XRD confirmed the expected crystalline structure and uniform morphology of UiO-66 and UiO-66-Qu, while HPLC analysis demonstrated high quercetin loading efficiency. Quercetin-loaded UiO-66 significantly improved working memory, evidenced by higher spontaneous alternation rates in the Y-maze ($p < 0.001$) and increased time spent in the novel arm during NADT ($p < 0.0001$), compared with disease + saline . Motor coordination was also enhanced, as indicated by longer hanging times in the wire hang test ($p < 0.001$). qRT-PCR showed substantial upregulation of MBP, Olig2, Nrf-2, and HO-1 ($p < 0.0001$), alongside pronounced downregulation of pro-inflammatory cytokines NLRP-3, TNF-α, IL-1β, and IL-18 ($p < 0.001$). Moreover, GPx, CAT, and SOD activities and TAC were markedly elevated, whereas MDA levels were significantly reduced in the UiO-66-Qu group ($p < 0.0001$).

Conclusion : Quercetin-loaded UiO-66 effectively attenuates oxidative stress, suppresses neuroinflammation, and promotes remyelination and functional recovery in cuprizone-induced demyelination. These findings emphasize the potential of MOF-based delivery systems to enhance the bioavailability and therapeutic efficacy of quercetin in neurodegenerative diseases, presenting a promising, safe, and cost-effective strategy for MS treatment.

Keywords : Drug Delivery, Nano carrier, Metal Organic Framework, Multiple sclerosis, Quercetin

Count: 267
Abstract ID: 674
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Machine Learning Models for Predicting Treatment Response During Multiple Sclerosis Flares: A Comparative Analysis with Traditional Statistical Approaches

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Background and Aim : Multiple sclerosis (MS) is a chronic, immune-mediated disorder characterized by recurrent demyelinating flares with heterogeneous responses to corticosteroid and disease-modifying therapies. Predicting treatment response remains challenging due to multifactorial disease behavior and inter-patient variability. This study aimed to develop and evaluate machine learning (ML) models for short-term treatment response during MS flares, compared with conventional statistical methods.

Methods : Clinical, imaging, and biochemical data from 136 patients with relapsing-remitting MS (RRMS) experiencing acute flares were retrospectively analyzed. Demographic, clinical, longitudinal (EDSS change, relapse frequency, DMT duration), and MRI-derived quantitative features (T2 lesion volume, T1 black hole volume, brain parenchymal fraction) were included. Serum biomarkers (NfL, GFAP, CHI3L1) were available for 88 patients. Treatment response was defined as ≥1.0 EDSS improvement or full remission within 30 days post-corticosteroids. Data were split (70/30) into training and validation cohorts. Logistic regression was compared with random forest, support vector machine, and gradient boosting models using nested 5-fold cross-validation. Feature scaling, imputation, and SMOTE oversampling were applied. Model accuracy, sensitivity, specificity, AUC (95% CI), and calibration were assessed.

Results : ML models outperformed logistic regression. Gradient boosting achieved 82% accuracy (95% CI: 78–86) and AUC 0.88 (95% CI: 0.83–0.92) vs. 70% and 0.75 (95% CI: 0.69–0.81) for logistic regression ($p<0.01$). Key predictors included MRI lesion burden, NfL, relapse duration, DMT exposure, and prior EDSS change. Adding longitudinal and biochemical features improved AUC by 0.07. Calibration was strong (Brier=0.14; slope=1.03).

Conclusion : ML models significantly improved prediction of treatment response during MS flares compared with conventional methods. These tools may support individualized treatment planning; multicenter prospective validation is warranted.

Keywords : Multiple sclerosis; machine learning; treatment response; prediction

Count: 268
Abstract ID: 475
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

The impacts of acetyl-l-carnitine on functional recovery and oxidative stress level in a demyelinated mouse model

Submission Author: Pegah Hazrati

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Background and Aim : Multiple sclerosis (MS) is a heterogeneous neuroinflammatory disease that damages the central nervous system. Inflammatory events and oxidative stress are usually interconnected components in MS. This study investigates the effects of antioxidant acetyl-L-carnitine (ALC), the most predominant acylated ester of amino acid carnitine, on motor function recovery and oxidative stress levels in a cuprizone-induced demyelination mouse model.

Methods : Male C57BL/6 mice were divided into three groups: The control group which received a standard diet; the cuprizone (CPZ) group which was fed a diet containing 0.2% CPZ for 12 weeks to induce demyelination; the CPZ+ALC group that administered ALC at 300 mg/kg/day from weeks 10 to 12 in addition to the cuprizone diet. Motor coordination and balance were assessed using the beam walking test (BWT). Furthermore, the corpus callosum was analyzed biochemically to evaluate the therapeutic effects of ALC on redux homeostasis status.

Results : The ALC treatment led to significant improvements in motor coordination and balance, as evidenced by enhanced performance in the BWT. Biochemical analysis revealed an increase in myelin sheath thickness in the corpus callosum. Biochemical assays indicated a reduction in malondialdehyde (MDA) levels and an increase in antioxidant levels including glutathione (GSH) and catalase activity (CAT) following ALC co-administration.

Conclusion : ALC-coadministration was able to improve motor functions in the demyelinated mouse model. These neuroprotective effects of ALC may be exerted through the maintenance of redox hemostasis balance in the corpus callosum as a susceptible area of demyelination in cuprizone intoxication.

Keywords : Multiple sclerosis; cuprizone; acetyl-L-carnitine; functional recovery; oxidative stress

Count: 269
Abstract ID: 405
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Effectiveness of Auditory Evoked Potentials in Evaluating Patients with Multiple Sclerosis

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Conclusion : Objective: Multiple sclerosis (MS) is an inflammatory and demyelinating disease that can affect several areas of the central nervous system. In MS, appearance of sclerotic plaques in the auditory pathways can cause a significant loss of neural integrity. Given that conventional audiological assessments do not consider many changes that occur along the central auditory pathway; therefore, examining the auditory pathway, from the brainstem to the cerebral cortex, can help assess the integrity of this pathway in MS patients. Method and materials: We performed a literature review with the following keywords: Multiple Sclerosis, Evoked Potentials and P300. Five different databases (PubMed, Scopus, Web of science, Science Direct and Google Scholar) were independently screened. Publications in English were reviewed from 2000 to 2025. Results: Statistical evaluations showed that there is significant differences between the normal and MS groups regarding the results of the auditory brainstem response and the latency of the P300 waves. These differences were observed between the control and study groups for the latency of waves III and V and between the peaks I-III and I-V of auditory brainstem response and in the latency of P300, the values of which were higher for the group with MS. Conclusion: Patients with MS represent abnormalities in auditory evoked potentials, which indicate dysfunction of different areas of the central auditory nervous system in this disease. In addition, researchers have emphasized the use of P300 as a clinical differentiator in patients with MS, because P300 is effective in diagnosing cognitive disorders and therefore increases the possibility of neuropsychological intervention with an emphasis on cognitive rehabilitation.

Keywords : Multiple Sclerosis, Evoked Potentials; P300

Count: 270
Abstract ID: 737
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Oral

Berberine Protects Against Cuprizone Demyelination In C57BL/6 Mice

Submission Author: Mohaddeseh sadat Alavi

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Background and Aim : Multiple Sclerosis (MS) is a neurological disorder characterized by demyelination and neuroinflammation. Berberine, a plant alkaloid found in Berberi's species, possesses various pharmacological properties, notably anti-inflammatory and antioxidant capabilities. This research aimed to assess the protective potential of berberine in C57BL/6 mice exposed to cuprizone.

Methods : The mice were fed with a daily diet intoxicated with cuprizone (0.2% w/w) and given 5 or 10 mg/kg of berberine for five weeks. Subsequently, changes in weight gain, balance, motor behavior, oxidative stress, and histopathology were evaluated.

Results : Berberine-treated animals demonstrated enhanced exploratory behavior and increased locomotor activity compared to cuprizone-fed mice. Additionally, assessments of oxidative stress showed that berberine reduced MDA level and increased GSH content, significantly. Furthermore, histological evaluations using Luxol Fast Blue staining indicated preservation of myelin sheaths, prevention of myelin breakdown in neurons, and reduction of demyelination-induced injuries in the berberine-treated groups.

Conclusion : The findings suggested that berberine has protective effects against cuprizone-mediated demyelination in C57BL/6 mice. The protective action of berberine was shown to be exerted via inhibition of oxidative stress.

Keywords : Multiple sclerosis, Demyelination, Cuprizone, Berberine, Oxidative stress

Count: 271
Abstract ID: 864
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Oral

Effect of Image Normalization on Detecting Active Multiple Sclerosis Lesions Using Deep Learning on Non-Contrast MRI

Submission Author: AmirAbbas Amini

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Background and Aim : Contrast-enhanced MRI is currently used to detect active lesions in multiple sclerosis (MS), but it carries potential side effects. Deep learning offers a promising alternative approach. While some studies incorporate MRI image normalization as a preprocessing step, its actual impact on MS lesion detection, given the unique nature of these lesions, remains unclear. This study investigates the effect of normalization on diagnostic accuracy.

Methods : Data were collected from 130 relapsing-remitting MS patients across four imaging centers in Isfahan, Iran (Ethics Code: IR.MUI.MED.REC.1400.474). Radiologists identified and classified lesions as active or inactive, totaling 9097 lesions. First, the lesions are masked, and then each mask is dilated. Next, each lesion is extracted as a region of interest (ROI) from FLAIR-sequence MRI scans to serve as input for the deep learning model. The images are then normalized using the z-score method. A convolutional neural network (CNN) is designed and trained to distinguish active from inactive lesions. The network's performance is evaluated and compared between normalized and non-normalized images using 5-fold cross-validation.

Results : For non-normalized images, accuracy, specificity, sensitivity, and AUC were 85±0.44, 95±2.79, 75±2.49, and 0.90±0.01, respectively. Normalized images yielded 84±0.38, 94±2.34, 74±2.42, and 0.90±0.02.

Conclusion : Our results demonstrate that deep learning models can effectively identify active MS lesions with high accuracy. However, image normalization did not significantly improve performance

Keywords : Multiple sclerosis (MS); Deep Learning; Image Normalization; Non-Contrast MRI

Count: 272
Abstract ID: 858
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Investigation of Fisetin Effects on Serum Immunoglobulin G Levels and Maintenance of Locomotor and Balance Posture in Mice, a Toxic Model of Myelin Tissue Destruction

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Background and Aim : Common molecular mechanisms are involved in the pathogenesis of neurodegenerative diseases. Compounds that can simultaneously target multiple molecular mechanisms are typically used to treat these diseases. In the present study, the effects of fisetin on serum immunoglobulin G levels and the maintenance of motor coordination and balance in mice were investigated.

Methods : In this study, 20 C57BL/6 mice were randomly divided into 5 groups including control, sham, cuprizone, fisetin, and cuprizone/fisetin. Motor coordination and balance impairment was induced using cuprizone. In the groups receiving fisetin, this compound was injected intraperitoneally at a dose of 20 mg/kg daily. Serum immunoglobulin G levels were evaluated using the ELISA method, and motor coordination and balance were assessed using the hanging wire test. Finally, data were analyzed using One Way ANOVA.

Results : The mean expression level of the IgG factor in the fisetin-treated groups showed a significant decrease compared to the cuprizone group ($P \leq 0.01$). Also, the mean score in the hanging wire test was significantly increased in the fisetin-treated groups compared to the cuprizone group ($P \leq 0.05$).

Conclusion : Exposure to toxic compounds such as cuprizone can lead to increased IgG expression and cause balance motor problems. Therefore, the use of flavonoids such as fisetin, whose immunomodulatory, antioxidant, and neuroprotective effects have been proven, can play an important role in reducing these complications.

Keywords : Fisetin Immunoglobulin G Cuprizone

Count: 273
Abstract ID: 769
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

The Effect of Combination of Hydro Alcoholic Extracts of Nigella Sativa and Linum Usitatissimum on Fatigue and Its Contributing Factors in Relapsing-Remitting Multiple Sclerosis; an RCT

Submission Author: Mohammad Abbasalizadeh

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Background and Aim : Fatigue affects up to 90% of multiple sclerosis (MS) patients with limited effective treatments. This study evaluated the effects of combined hydroalcoholic extracts of Nigella sativa and Linum usitatissimum on fatigue and contributing factors in relapsing-remitting MS (RRMS).

Methods : A parallel-group, randomized, double-blind, placebo-controlled clinical trial was conducted with RRMS patients (EDSS<4.5) experiencing fatigue. Participants received either herbal supplement capsules (0.75 mL each extract; n=17) or placebo (n=18) twice daily for three months. Primary outcomes were assessed using the Comprehensive Fatigue Assessment Battery for Multiple Sclerosis (CFAB-MS) and Visual Analogue Scale (VAS). Statistical analysis employed SPSS version 23 with appropriate parametric and non-parametric tests.

Results : Thirty-five patients completed the trial (mean age 34.11±9.10 years, 85.7% female). The intervention group showed statistically significant improvement in nutritional status VAS scores (p=0.009) compared to placebo. No significant differences were observed between groups for physical, mental, and social fatigue indices, pain, sleep disturbances, stress, mood, anxiety, mobility, environmental factors, or fatigue management (all p>0.05).

Conclusion : Three months of N. sativa and L. usitatissimum supplementation did not significantly improve fatigue in RRMS patients. While demonstrating acceptable safety profile,

this combination lacks efficacy as a standalone fatigue treatment, though potential nutritional benefits warrant further investigation in larger trials.

Keywords : Multiple sclerosis, Fatigue, Nigella sativa, Randomized controlled trial, Herbal medicine

Count: 274
Abstract ID: 477
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Oral

Effects of acetyl-l-carnitine on histopathology and myelin-related gene expression of corpus callosum in the demyelinated mouse model

Submission Author: SeyedMohammadhadi Mirab

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Background and Aim : Multiple sclerosis (MS) is a chronic demyelination disorder that affects the myelin sheath. Current treatments primarily aim to modulate the immune response, but there is an ongoing need for therapies that promote remyelination and neuroprotection. This study aims to evaluate the neuroprotective effects of acetyl-L-carnitine (ALC) on cuprizone-induced demyelination.

Methods : Adult C57BL/6 mice were randomly assigned to three groups. The control group received a standard diet throughout the study (CONT). The Cuprizone (CPZ) group was fed a diet containing 0.2% cuprizone for 12 weeks to induce consistent demyelination. The CPZ+ALC group received the same CPZ diet for the first 9 weeks, followed by ALC co-administration from weeks 10 to 12. At the end of the study, the mice were euthanized, and their brains were subjected to histopathological and molecular evaluations in the corpus callosum.

Results : Histopathological analysis revealed substantial remyelination in the corpus callosum of ALC-treated mice. On a molecular level, the expression of key genes associated with oligodendrocyte function and myelin production namely oligodendrocyte lineage transcription factor 2 (Olig-2) and proteolipid protein (Plp) was significantly upregulated in the CPZ + ALC Group. Olig-2 is essential for oligodendrocyte precursor cell differentiation into mature oligodendrocytes, while PLP is a major component of the myelin sheath.

Conclusion : This study demonstrates that ALC has a considerable role in the remyelination process in the cuprizone intoxicant mice. These effects may be exerted throughout the upregulation of Olig-2 and Plp genes indicating that ALC not only aids in the protection of existing myelin but also promotes the generation of new myelin.

Keywords : Multiple sclerosis, cuprizone, remyelination, acetyl-L-carnitine, myelin-related gene expression.

Count: 275
Abstract ID: 798
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Identifying a panel of objective biomarker profiles is crucial before using mesenchymal stem cells for multiple sclerosis: a systematic review

Submission Author: Shahrzad Najafi

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Conclusion : Abstract Background: Multiple sclerosis (MS) is a chronic autoimmune disorder of the central nervous system (CNS), characterized by axonal degeneration, demyelination, and inflammation, which collectively contribute to progressive neurological disability. Although disease-modifying therapies (DMTs) have improved outcomes in relapsing forms, progressive phenotypes—secondary progressive MS (SPMS) and primary progressive MS (PPMS)—remain challenging to manage. Mesenchymal stem cell (MSC) therapy has emerged as a promising approach due to its immunomodulatory and neuroprotective properties. This systematic review aimed to identify objective biomarkers associated with clinical outcomes and evaluate the therapeutic potential of MSC therapy in MS patients. Methods: Following the PRISMA 2020 guidelines and the Patient–Intervention–Comparison–Outcome (PICO) framework, a systematic search was conducted across PubMed, Scopus, Web of Science, and Embase databases. Thirty-four eligible clinical studies evaluating MSC therapy in MS were included. Extracted data encompassed MSC source, dosage, administration route, biomarker alterations, and clinical outcomes. Study quality was appraised using Joanna Briggs Institute

(JBI) tools. Results: Clinical indices such as the Expanded Disability Status Scale (EDSS), 25-Foot Walk Test (25-FWT), and 9-Hole Peg Test (9-HPT) frequently showed stability or improvement. MRI findings revealed reduced or stable gadolinium-enhancing and T2 lesion volumes. Immunological biomarkers, including increased $FOXP3^{+}$ regulatory T cells and decreased pro-inflammatory cytokines (TNF-α, IL-6, IL-17), indicated immune modulation, while neurodegenerative markers such as neurofilament light (NFL) were largely reduced or stabilized. Umbilical cord-derived MSCs and repeated dosing regimens were associated with superior outcomes. MSC therapy was generally well tolerated, with no serious adverse events reported. Conclusion: MSC therapy demonstrates substantial promise in modulating immune responses and mitigating neurodegeneration in MS, particularly in progressive subtypes. However, a standardized, quantitative biomarker with high diagnostic accuracy, sensitivity, and specificity is still lacking. Establishing unified protocols and integrating sensitive biomarkers with clinical measures are essential steps toward validating MSC therapy. Further large-scale, well-designed studies are required to develop a reliable biomarker panel for assessing MSC efficacy in MS.

Keywords : Multiple sclerosis; Mesenchymal stem cells; Biomarkers; Neurofilament light; Immunological markers; Progressive MS; Regenerative medicine.

Count: 276
Abstract ID: 619
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Interferon-β and Glatiramer Acetate Differentially Remodel the Gut-Immune Axis in Multiple Sclerosis

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Background and Aim : While disease-modifying therapies like Interferon-β (IFN-β) and Glatiramer Acetate (GA) are cornerstones of multiple sclerosis (MS) management, their impact on the gut microbiome and its interplay with the host immune system is not fully understood. It remains unknown whether these agents promote a common pathway towards immune homeostasis or induce distinct, treatment-specific gut-immune signatures. This study aims to dissect and compare the molecular remodeling induced by IFN-β and GA across the gut-immune axis.

Methods : We conducted an integrative re-analysis of public multi-omics data, including 16S rRNA gut microbiome sequencing (BioProject PRJNA321051) and peripheral T-cell/monocyte transcriptomics (GEO GSE81279). Cohorts were stratified into Untreated MS, IFN-β treated, and GA treated. Using the Galaxy platform, we performed differential abundance analysis (LEfSe) to identify treatment-specific microbial biomarkers and differential gene expression analysis (DESeq2) to define transcriptional reprogramming in immune cells. Finally, we constructed correlation networks to link the drug-induced microbial shifts with host immune responses.

Results : Our comparative analysis revealed a stark mechanistic divergence between the two therapies. Venn diagrams showed minimal overlap in the microbial taxa and host genes modulated by each drug. Specifically, IFN-β treatment was associated with a significant enrichment of the mucin-degrading bacterium Akkermansia muciniphila. This microbial shift was strongly correlated with a transcriptional signature in monocytes characterized by the upregulation of canonical Interferon-Stimulated Genes (ISGs), such as STAT1 and OAS1. In stark contrast, GA administration promoted the expansion of butyrate-producing Clostridia, including Faecalibacterium and Roseburia. This alteration was significantly correlated with a distinct transcriptional program in T-cells, marked by an increased expression of the master regulatory T-cell transcription factor, FOXP3, and its downstream targets, suggesting a promotion of peripheral tolerance.

Conclusion : Our findings demonstrate that IFN-β and GA do not converge on a single mechanism of action. Instead, they drive divergent, non-overlapping pathways of gut-immune remodeling. IFN-β appears to leverage the gut microbiome to potentiate innate antiviral signaling in monocytes, whereas GA utilizes it to foster an adaptive regulatory T-cell response. This mechanistic insight challenges the notion of a simple "correction" of dysbiosis and provides a framework for personalizing therapies and predicting treatment response based on a patient's baseline microbial and immune profile.

Keywords : Multiple Sclerosis, Immunomodulation, Gut-Immune Axis, Treatment Response

Count: 277
Abstract ID: 654
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Identifying a Core Microbial Dysbiosis Signature in Multiple Sclerosis Independent of Treatment Modality

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Background and Aim : Gut dysbiosis is a recognized feature of multiple sclerosis (MS), but the extent to which this signature is modified by disease-modifying therapies (DMTs) remains unclear. It is plausible that certain microbial alterations represent a persistent, “core” pathological feature of MS that is not fully rectified by current treatments. This study aimed to identify such a treatment-independent microbial signature by comparing a combined cohort of untreated and treated MS patients against healthy controls (HC).

Methods : We conducted a meta-analysis of public 16S rRNA gene sequencing data (SRA accession: SRP329371), which includes samples from HC, untreated MS patients (N=4), and patients treated with either dimethyl fumarate (DMF, N=9) or fingolimod (FTY720, N=3). To identify a core MS signature, we grouped all MS patients (N=16) into a single “MS cohort” and compared it against the HC group. The analysis, performed in Galaxy, involved alpha/beta diversity assessment and differential abundance analysis using LEfSe to detect consistent taxonomic differences. Functional potential was inferred using PICRUSt2 to identify conserved metabolic pathway alterations.

Results : Despite the distinct ecological shifts induced by DMF and FTY720, a significant core dysbiosis signature was identified in the combined MS cohort compared to HC. Beta diversity analysis confirmed a clear separation between the HC and the MS cohort ($p<0.01$, PERMANOVA). The core MS signature was characterized by a consistent depletion of key butyrate-producing families, including Ruminococcaceae and Lachnospiraceae, and a lower abundance of the mucin-degrading bacterium Akkermansia muciniphila. Conversely, the MS cohort showed a robust enrichment of several genera within the Proteobacteria phylum, known for their pro-inflammatory potential. Functionally, this taxonomic signature corresponded to a predicted decrease in microbial pathways for short-chain fatty acid (SCFA) biosynthesis and an increase in pathways related to lipopolysaccharide (LPS) production and bacterial motility. These features persisted across the MS spectrum, irrespective of treatment status.

Conclusion : Our analysis reveals a persistent microbial dysbiosis signature in MS that transcends the effects of immunomodulatory treatments. This “pathobiome” core,

characterized by a reduced capacity for producing anti-inflammatory SCFAs and an elevated pro-inflammatory potential, may represent a fundamental aspect of the disease's gut-immune axis disruption. These findings suggest that while DMTs effectively modulate systemic immunity, they may not fully resolve the underlying gut dysbiosis. Targeting this core microbial signature with complementary strategies, such as tailored probiotics or dietary interventions, could offer a novel therapeutic avenue to more comprehensively manage MS.

Keywords : multiple sclerosis; Gut Microbiome; Microbial Dysbiosis; Data Analysis; Genetics

Count: 278
Abstract ID: 662
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Divergent Remodeling of the Gut Microbiome by Dimethyl Fumarate and Fingolimod in Multiple Sclerosis

Submission Author: Ghazaleh Shahan behbahani

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Background and Aim : Dimethyl fumarate (DMF, Tecfidera) and fingolimod (FTY720, Gilenya) are effective oral therapies for multiple sclerosis (MS) but operate via distinct mechanisms—DMF primarily through the Nrf2 antioxidant pathway and fingolimod via lymphocyte sequestration. Their differential impact on the gut microbiome, a key modulator of neuro-inflammation, remains poorly defined. This study performs a comparative bioinformatic analysis to delineate the specific signatures of microbiome alteration induced by these two treatments.

Methods : We analyzed public 16S rRNA gene sequencing data (SRA accession: SRP329371) from untreated MS patients (N=4), DMF-treated patients (N=9), and a cohort of fingolimod-treated patients (N=3). The analysis pipeline, implemented in Galaxy, included alpha and beta diversity assessment, differential abundance analysis (LEfSe), and prediction of metagenomic functional potential (PICRUSt2) to compare the DMF and fingolimod groups against the untreated cohort.

Results : Both treatments induced significant, yet distinct, shifts in the gut microbiome structure. DMF treated patients exhibited a marked enrichment of butyrate-producing taxa, particularly Faecalibacterium, and a corresponding depletion of pro-inflammatory Proteobacteria. Functional profiling predicted a significant upregulation of microbial pathways for butyrate synthesis, suggesting an enhanced potential to produce metabolites that activate the host Nrf2 pathway. In striking contrast, the fingolimod-treated cohort, while also distinct from untreated patients, did not show enrichment in butyrate producers. Instead, preliminary analysis pointed towards alterations in taxa associated with the metabolism of lipids and amino acids, potentially reflecting secondary metabolic consequences of lymphocyte sequestration in gut-associated lymphoid tissues (GALT). The overall microbial diversity(alpha diversity) was notably lower in the fingolimod group compared to both DMF-treated and untreated groups.

Conclusion : DMF and fingolimod drive divergent ecological shifts in the MS gut microbiome. DMF appears to foster a functionally beneficial community, enhancing the microbiome's potential to produce anti-inflammatory metabolites like butyrate, which may synergize with the drug's direct Nrf2-activating effects. Conversely, fingolimod induces a different dysbiotic state, potentially linked to its profound impact on immune cell trafficking in the gut. These findings underscore that different MS therapies exert unique effects on the gut-immune axis and suggest that microbiome profiling could be instrumental in personalizing treatment strategies and understanding their holistic biological impact.

Keywords : Multiple Sclerosis; Gut Microbiome; Gut-Immune Axis; fingolimod; Dimethyl fumarate

Count: 279
Abstract ID: 601
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Evaluation of salivary nitric oxide levels and anxiety in multiple sclerosis patients, with and without Xerostomia: correlation with clinical variables

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Background and Aim : Xerostomia is a prevalent but often overlooked condition in multiple sclerosis (MS) patients, significantly impacting their quality of life and oral health. This cross-sectional observational study investigates the role of nitric oxide (NO) in the pathophysiology of multiple sclerosis (MS) and explores its association with xerostomia in MS patients. The primary objective was to compare salivary NO concentrations and stress levels between MS patients with and without xerostomia.

Methods : MS patients diagnosed by neurologists and MRI were categorized into two groups: those with xerostomia and those without. Unstimulated whole saliva samples were collected using the spitting method, and salivary NO levels were quantified using an enzyme-linked immunosorbent assay (ELISA) kit based on the Griess reaction. Stress levels were assessed using the Beck Anxiety Inventory (BAI) questionnaire. The presence of xerostomia was evaluated through the Xerostomia Inventory (XI) and clinical examinations.

Results : Salivary NO levels were significantly higher in MS patients without xerostomia (227.47 ng/mL) compared to those with xerostomia (102.37 ng/mL, $p < 0.001$). Stress levels were also notably higher in MS patients with xerostomia (17.23) versus those without (11.77, $p = 0.03$). A moderate negative correlation was observed between salivary NO levels and

xerostomia ($r = 0.44$, $p < 0.001$), indicating that lower NO levels were associated with a higher likelihood of xerostomia. The correlation between stress levels and xerostomia was weaker but still significant ($r = 0.28$, $p = 0.03$). Multivariate binary logistic regression analysis identified salivary NO, stress levels, and age as significant predictors of xerostomia in MS patients. The logistic regression model achieved an 80% accuracy in predicting xerostomia based on salivary NO levels and stress

Conclusion : This study highlights a significant negative correlation between salivary NO levels and xerostomia, suggesting that decreased salivary NO concentrations are associated with an increased risk of xerostomia in MS patients. Additionally, stress levels were positively correlated with xerostomia, indicating a potential link between higher stress and the likelihood of xerostomia in MS patients.

Keywords : Multiple sclerosis, Nitric oxide, Xerostomia, Saliva, Stress, Anxiety, Oral health

Count: 280
Abstract ID: 632
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Poster

Bioinformatic Dissection of EBV Latency in MS: B-Cell Transcriptomic Reprogramming Drives CNS Autoimmunity

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Background and Aim : Epstein-Barr Virus (EBV) is increasingly recognized as a pivotal environmental trigger in Multiple Sclerosis (MS), reprogramming B cells through latency genes to drive autoimmunity. This study investigates how EBV latency genes, such as EBNA1 and EBNA2, alter B-cell transcriptomes, promoting inflammation, migration, and antigen presentation relevant to MS pathogenesis. Using RNA-sequencing data from GEO datasets GSE244314 (MS patient-derived B cells with unstable EBV latency) and GSE221624 (dysregulated EBV control in pro-inflammatory B cells), we performed differential expression analysis to identify EBV-induced perturbations. Key findings reveal upregulated pathways in NF-κB signaling, cytokine production (IL-6, IL-23), and adhesion/migration (CXCR4, SPP1), linking viral latency to CNS barrier breakdown and T-cell activation. These insights underscore EBV's role in exploiting MS genetic risk loci, offering targets for B-cell therapies and vaccines.

Methods : To evaluate EBV's transcriptomic impact on B cells in MS, we analyzed public RNA-seq datasets GSE244314 and GSE221624. GSE244314 includes EBV-infected memory B cells from MS patients, highlighting unstable latency and inflammation. GSE221624 focuses on intrinsic B-cell dysregulation of EBV expression, driving pathogenic phenotypes. Raw FASTQ files underwent quality control (FastQC), adapter trimming (Trimmomatic), and alignment to GRCh38 (STAR). Counts were generated (HTSeq), followed by DESeq2 differential expression in R, comparing infected vs. uninfected B cells. We prioritized genes in B-cell activation, cytokine signaling (IL-6/IL-23), antigen presentation, and migration (CXCR4/SPP1). GSEA assessed MS pathways (NF-κB, MAPK, apoptosis). Batch effects were corrected (ComBat-seq). Consensus genes/pathways were integrated across datasets for robustness.

Results : Analysis yielded 1,247 differentially expressed genes (DEGs) in GSE244314 (FDR < 0.05, |log2FC| > 1), with 892 upregulated in EBV-infected B cells, including EBNA2 targets like BATF and IRF5, aligning with MS risk alleles. In GSE221624, 1,056 DEGs emerged, with

overlap of 312 consensus genes, notably pro-inflammatory markers. Upregulated cytokines IL6 (log2FC=2.3) and IL23A (log2FC=1.8) suggest enhanced Th17 support, while SPP1 (osteopontin, log2FC=3.1) and CXCR4 (log2FC=2.0) indicate CNS homing. GSEA confirmed NF-?B (NES=2.4, p<0.001) and MAPK (NES=2.1) enrichment, driven by LMP1/LMP2A mimics of BCR signaling, alongside apoptosis resistance via BCL2L1 (log2FC=1.5). Antigen presentation genes (HLA-DRA, CD70) were elevated, fostering B-T interactions. In MS contexts, these mirror atypical B-cell signatures with PD-L1 expression on infected cells, promoting T-cell exhaustion. Integrated results show EBV latency reprograms ~15% of MS GWAS loci, validating viral exploitation of host genetics for B-cell survival and inflammation.

Conclusion : EBV latency genes fundamentally reprogram B cells, transforming them into pathogenic drivers of MS by upregulating survival, migration, and pro-inflammatory pathways. Consensus DEGs and enriched signaling cascades from GSE244314 and GSE221624 affirm EBV's orchestration of immune dysregulation, from barrier infiltration to tertiary lymphoid structure formation. These findings extend the abstract's framework, positioning EBNA1/2 as molecular mimics and regulators exploiting genetic vulnerabilities. Therapeutically, targeting EBV reservoirs via adoptive T-cells or vaccines could complement anti-CD20 depletion, disrupting B-T collaboration. Future prodromal studies on reactivation and myeloid involvement are essential to halt progression in susceptible individuals. Ultimately, EBV emerges not as a bystander but a master instigator of CNS autoimmunity, demanding integrated antiviral-immunomodulatory strategies.

Keywords : Epstein-Barr Virus (EBV), Multiple Sclerosis (MS), B-cell Reprogramming, Latency Genes, NF-κB Signaling

Count: 281
Abstract ID: 655
subject: Neural Injuries and Neurodegenerative Disorders: Demyelinating Disorders
Presentation Type: Oral

Pegylated Chitosan Nanoparticles of Fluoxetine Enhance Cognitive Performance and Hippocampal Brain Derived Neurotrophic Factor Levels in a Rat Model of Local Demyelination

Submission Author: Salva Afshari

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Background and Aim : Destruction of myelin sheaths can lead to disruptions in information transmission, a crucial factor in various neurodegenerative diseases such as Multiple Sclerosis (MS). This study investigates the potential of enhancing the positive effects of fluoxetine, a neuroprotective agent known for its ability to increase neurotrophin levels, on remyelination by utilizing pegylated chitosan nanoparticles loaded with fluoxetine.

Methods : Local demyelination was induced in the CA1 region of the hippocampus using a lysolecithin injection via a stereotaxic procedure, and anxiety-like behavior was assessed through open field maze (OFM) and elevated plus maze (EPM) tests. The novel object recognition memory test (NORMT) was employed to evaluate recognition memory. ELISA was used to measure insulin-like growth factor 1 (IGF-1) and brain-derived neurotrophic factor (BDNF) levels in the hippocampus, while Luxol fast blue staining was utilized to quantify the extent of remyelination.

Results : The nanoparticle (NP)-treated groups exhibited reduced anxiety-like behavior in the OFM and EPM tests. NORMT results showed a better performance in NP-treated groups. Moreover, the NP-treated groups demonstrated significantly higher levels of BDNF ($P < 0.01$), no significant change in IGF-1 levels ($P > 0.05$), along with a reduced extent of demyelination ($P < 0.001$)

Conclusion : Treatment with nanoparticles of fluoxetine resulted in better cognitive performance, enhanced levels of BDNF, and decreased demyelination lesion extent compared to fluoxetine. These findings support the argument for further research into innovative drug delivery systems for repurposed drugs, as they have the potential to yield improved and sometimes unexpected outcomes.

Keywords : Demyelination; Fluoxetine; Cognitive performance; Nanoparticle drug delivery; Neurodegenerative diseases

Count: 282
Abstract ID: 1036
subject: Development: Neuronal Cell Death
Presentation Type: Poster

Investigating the Effects of Curcumin on Cigarette Smoke-Induced Neuronal Damage in PC12 Cells

Submission Author: Mona Borughani

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Background and Aim : Cigarette smoke exposure induces severe oxidative stress, a key process that contributes to the pathophysiology of several neurodegenerative disorders via the mediation of neuronal damage. This study investigates the neurotoxic impact of cigarette smoke extract (CSE) on PC12 cells, a validated neuronal model, and the potential protective effect of curcumin, the active component in turmeric, as a known natural antioxidant agent.

Methods : To prepare the cigarette smoke extract (CSE), the smoke produced from a single cigarette was collected and dissolved in 10 mL of PBS. PC12 cell line were exposed to the cigarette extract in 10 different concentrations ranging from 1.5µM to 30µM (also 3, 6,9, 12, 15, 18, 22.5 and 27µM), also co-treated with curcumin solution in two concentrations, 2.5µM and 5µM at 37 °C and 5% CO2. After 24 h, the cell viability was tested by MTT assay in PC12 cells.

Results : The MTT assay revealed a concentration-dependent decrease in PC12 cell viability in response to cigarette smoke extract (CSE), with viability dropping to as low as 26% at the highest concentration tested (30 µM). A significant reduction was observed at concentrations of 22.5 µM and higher but not in a monotonic pattern. Interestingly, a low concentration of CSE (3 µM) alone, slightly increased viability compared to the control. Moreover, curcumin pretreatment demonstrated a protective effect against CSE-induced toxicity at 2.5 µM. However, this protection was not observed at 5 µM curcumin concentration, which instead further reduced cell viability compared to CSE alone. This protective effect attenuated in higher than 22.5 µM concentrations of CSE.

Conclusion : The cigarette smoke extract activated cell death especially in higher concentrations. Lower concentrations of curcumin showed protective effects to some extent, but higher concentrations act the opposite, highlighting the dual effects of curcumin on PC12 cells. Further investigation on potential effects of curcumin, notably in lower than 2.5µM concentrations, and genetic tests are needed.

Keywords : pc12 cells; curcumin; neuroprotective; Tobacco Products

Count: 283
Abstract ID: 557
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Oral

From Words to Connection: AI-Guided Empathy Training to Enhance Verbal Social Communication in High-Functioning Autistic Male Adolescents

Submission Author: Forough Kasiri

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Background and Aim : High-functioning autistic individuals often face significant challenges in understanding communicative intent and recognizing affective nuances in social interactions. These difficulties hinder their ability to generate appropriate verbal empathic responses, which are essential for initiating and maintaining social relationships. While AI-assisted interventions show promise in supporting socio-emotional learning in autistic children, few studies have moved beyond basic emotion recognition to foster verbal empathy in autistic adolescents within conversational settings. To address this gap, we investigated whether real-time feedback from a Large Language Model (GPT-4), delivered under therapist guidance, could improve verbal empathic responses in autistic adolescents.

Methods : Thirty diagnosed high-functioning autistic male adolescents, aged 12 to 15, participated in this study. All were capable of engaging in 20-minute social interactions using grammatically correct sentences but demonstrated less than 60% accuracy in producing appropriate empathic responses during pre-intervention conversations. Inclusion criteria allowed co-occurring diagnoses such as ADHD, OCD, and social anxiety, reflecting their complex clinical profiles. Following baseline assessment, participants were randomly assigned to either the experimental or control group (15 each). The intervention employed the web-based GPT-4 platform provided by OpenAI. Therapists orally presented leading statements—four positive, two neutral, and four negative—in random order and simultaneously submitted these statements to the AI system. Participants first identified the emotional tone of each statement, then generated verbal empathic responses. The AI instantly evaluated responses as empathetic or unempathetic and provided tailored feedback explaining its judgment. When responses required improvement, the AI supplemented feedback with examples of verified empathic replies. Therapists closely monitored sessions to prevent AI hallucinations or inaccuracies, ensuring clinical validity. Feedback focused on empathy level and suggested ways to enhance

conversational quality. Experimental participants completed at least ten trials per day, twice weekly for five weeks (approximately 100 trials). Additionally, all treatment sessions were conducted individually (one-on-one) rather than in group settings, allowing for personalized therapist supervision and tailored AI feedback. The control group lacked personalized AI feedback and merely rehearsed standardized empathic responses provided by the therapist. Pre- and post-intervention assessments involved verbal responses to ten emotional statements, with similar but distinct probes used at each time point. All responses were recorded and rated by two independent autism experts using a five-point Likert scale ranging from unacceptable (1) to highly acceptable empathic responses (5). Inter-rater reliability was high.

Results : The results of running MANCOVA revealed significant improvement in affective emotion recognition and empathic responding of the experimental group compared to the control one ($p = 0.001$). Specifically, the experimental group showed an average 21.4% increase in empathic response accuracy, while the control group demonstrated a 13% improvement. Additionally, during interviews, parents reported that these gains modestly extended beyond the intervention and were observed in the participants' natural conversations.

Conclusion : These findings underscore the benefits of integrating AI-driven feedback with traditional empathy training to enhance verbal empathy and affective recognition in autistic adolescents. Using such an approach can help bridge the gap between computer science and psychology, fostering the development of more effective AI-assisted interventions.

Keywords : AI-assisted Intervention; Autism; Empathy; Adolescents; Therapy

Count: 284
Abstract ID: 187
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Oral

Effectiveness of storytelling program on self-concept of children with physical and motor disabilities

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Background and Aim : The present study aimed to investigate the effectiveness of storytelling on the self-concept and of children with physical and motor disabilities.

Methods : The research method was a semi-experimental pre-test post-test design with a control group. The statistical population of this study was all elementary school students in schools for children with physical and motor disabilities in Tehran who were studying in the academic year 1402-1403. A total of 30 people were selected as a sample using a purposive non-random sampling method and Pierce and Harris' (1969) children's self-concept questionnaire was used. Then the samples were randomly divided into two groups of 15 people, experimental and control. The experimental group participated in storytelling sessions and the control group waited in the waiting list to receive the intervention after the end of the original intervention. At the end, the children responded to the post-test. The collected data were analyzed using SPSS statistical software.

Results : According to the statistical findings, it was shown that there was significant difference between the self-consept in the experiment and the control group in the pre and post test stages.

Conclusion : It can be concluded that storytelling has an effect on the self-concept of children with physical-motor disabilities, and after storytelling in the experimental group, self-concept. As a result, storytelling can be a suitable treatment for improving self-concept and body image in children with physical-motor disabilities.

Keywords : storytelling, self-concept, body image, physical-motor disability.

Count: 285
Abstract ID: 188
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

The effectiveness of cognitive-visual-motor- electronic training package, “Neuro-light”, on working memory of students with specific learning disability Abstract

Submission Author: Hanie Salehi

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Background and Aim : The aim of this study was to evaluate the effect of cognitive-visual-motor- electronic training package on working memory in students with specific learning disability.

Methods : The research method is a semi-experimental type and a pre-test and post-test design was used along with the control group. The statistical population of the present study was all students with special learning disability aged 6-12 years in Karaj city, 1403, and 22 of these people were selected using a targeted sampling method. Then the Digit span sub-test of Wechsler intelligence scale (1986) was used to measure the memory of the participants. The students were randomly divided into two experimental and control groups. After that, each of the people of the experimental group was individually trained for 8 sessions during 45 minutes with face-to-face and cognitive-visual-motor-electronic training package, which is called “Neuro-light” and was designed, developed and manufactured by the Pars-cognition Company, with collaboration with the computer science department of University of Tehran and used for this group of exceptional children for the first, during the training sessions, while the control group did not receive any intervention. After the completion of the training sessions, from the experimental and control group, Digit Span sub-test of Wechsler intelligence scale were performed as a post-test, and the data obtained were analyzed in SPSS software using covariance analysis.

Results : The findings of the research show the significant difference between experiment and control group in the working memory and indicated the effectiveness of the cognitive-visual-

motor- electronic training package of working memory for students with specific learning of students with specific learning disability.

Conclusion : As the result, it can be concluded that this cognitive-visual-motor-electronic training package increased the working memory performance of the participating students and it can be used along with other rehabilitation intervention for improving the working memory performance of student with specific learning disability.

Keywords : working memory, specific learning disorder, Neuro-light cognitive-visual-motor-electronic training package,

Count: 286
Abstract ID: 598
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

Oxytocin nano-formulation reduces the effective therapeutic dose by half in mitigating autism symptoms in maternal separated rats

Submission Author: SeyedehMaryam AlaviMoghaddam

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Background and Aim : Oxytocin (OXT) is a neuropeptide that plays a pivotal role in regulating social cognition and behavior. Dysregulation of the oxytocin system, driven by genetic, environmental, and epigenetic factors, has been implicated in the pathophysiology of autism spectrum disorder (ASD). However, the clinical application of oxytocin remains limited due to its poor permeability across the blood–brain barrier (BBB). To overcome this challenge, nanotechnology-based drug delivery strategies have emerged as promising approaches for enhancing oxytocin's therapeutic potential. This study aimed to assess the effectiveness of nano-capsulated oxytocin with Noisome at a dose of 0.5 mg/kg (half of the reported effective dose of oxytocin (1mg/kg) in animal models of autism) on autism-related behavior in maternal separated (MS) male rats.

Methods : To induce autism-like behaviors, three groups of male rats were subjected to maternal separation (MS) from postnatal day (PND) 1–14, being separated from their dams for 3 h daily (09:00–12:00) and housed individually; one group served as the control (n = 10 per group). After weaning (PND 21), rats received daily intraperitoneal injections from PND 22–30 of niosomal oxytocin, conventional oxytocin, or saline (0.5 mg/kg). Autism-related behaviors were assessed in adolescence (PND 42–50) using the three-chamber, marble-burying, and open-field tests to evaluate sociability, repetitive, and anxiety-like behaviors.

Results : The MS model induced autistic-like behaviors, including social deficits, increased repetitive behaviors, and heightened anxiety. Niosomal oxytocin (0.5 mg/kg) improved sociability ($p \leq 0.05$) and reduced repetitive ($p \leq 0.001$) and anxiety-like behaviors, whereas conventional oxytocin at the same dose had no effect.

Conclusion : This study showed that niosomal oxytocin had greater therapeutic effects on autistic-like behaviors in maternal-separated male rats than conventional oxytocin. Nanoencapsulation halved the effective dose (from 1 mg/kg) and improved blood–brain barrier penetration, reducing the need for high systemic dosing.

Keywords : Animal models; Autism treatment; Maternal separation; Nanoencapsulation; Oxytocin dose.

Count: 287
Abstract ID: 589
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

Brain Organoids as Precision Medicine Platforms for Autism Spectrum Disorder: Bridging Developmental Neurobiology and Targeted Therapies

Submission Author: Mozhan Parsa

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Background and Aim : Autism Spectrum Disorder (ASD) is an extremely heterogeneous neurodevelopmental disorder that is often qualified on behavioral criteria, frequently after important developmental pathways have been completed. Two major impediments to the development of successful pharmacotherapy are the lack of accessible biomarkers and the inability to track disease progression at the cellular and molecular levels. The conventional use of postmortem human brain tissue has been inadequate, as it cannot capture the rapidly changing developmental processes and is subject to tissue degradation. Animal models, while informative, do not recapitulate human-specific features, including cortical gyrification and subclasses of neurons, and are unable to fully model aspects of human social cognition. Recent advances in induced pluripotent stem cells (iPSCs) and organoid technology offer promising new approaches to deal with these challenges in applied frameworks in precision medicine.

Methods : This research project was based on a narrative review of the current body of literature. We used thorough searches of key scientific literature databases, including PubMed, Scopus, and Web of Science, up to 2025, to find appropriate articles. The search terms included combinations of: Autism Spectrum Disorder; Brain Organoids; Precision Medicine; Biomarkers; iPSCs; and Neurodevelopment. Additional references were discovered by exploring the reference lists of articles identified through the previously detailed approaches. Each article was screened for relevance, and selected articles for analysis focused on applications of iPSCs and cerebral organoids for biomarker discovery, disease models, and therapeutic testing in ASD.

Results : Cerebral organoids faithfully recapitulated processes of early brain development and showed deficits in higher-order cognitive regions implicated in ASD previously. Organoid models derived from patients showed: • Altered proliferation of neuronal progenitor cells

consistent with macrocephaly in sub-groups of ASD patients. • Disruption of the excitatory/inhibitory balance and synaptic development. • Disrupted pathways were identified (FOXG1-dependent overproduction of inhibitory neurons). These findings enabled the identification of candidate biomarkers using an omics-based profiling approach from specific developmental time points that could not be examined in vivo. In addition, drug-testing using organoids may show translational potential, and we were able to restore synaptic and signaling deficits in patient-specific syndromic and idiopathic ASD models with pathway-specific compounds (IGF-1, rapamycin, and lithium) and treatment paradigms.

Conclusion : Brain organoids offer a robust, human-limited, and developmentally relevant platform for precision medicine in autism spectrum disorder (ASD). These organoids can be utilized for the detection of biomarkers, provide mechanistic insights into disrupted neurodevelopmental trajectories, and perform individualized therapeutic screening. Brain organoids would overcome the limitations of animal models and postmortem tissues, thereby providing a potential bridge from basic neuroscience to clinical implementation. Certain challenges currently exist in the implementation of cerebral organoids, namely variability and lack of vascularization, but new solutions, ranging from CRISPR-engineered isogenic controls, new bioreactors, and including heterogeneous non-neuronal cell types, facilitate the rapid trajectory of this research toward translation. Introducing research with brain organoids into ASD studies offers an exciting future for the modification and development of diagnostic strategies and implementing targeted, patient-specific interventions.

Keywords : Autism Spectrum Disorder; Brain Organoids; Precision Medicine; Biomarkers; Neurodevelopment; iPSCs

Count: 288
Abstract ID: 608
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Oral

Oxytocin Restores Social Behavior and Cortical Plasticity Impairments Induced by Early-Life Sensory Overstimulation: Implications for Autism Spectrum Disorder

Submission Author: Mozhan Parsa

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Background and Aim : Overexposure to digital screens in early life has been linked to behavioral and neurodevelopmental deficits similar to autism spectrum disorder (ASD). Previously, we demonstrated that excessive audio-visual stimulation (EAVS) administered during postnatal days (PNDs) 12-35 produces social deficits and structural changes in the amygdala of rats. In this present work, we aimed to extend previous observations, and to do so, we examined whether intranasal oxytocin (OXT), a neuropeptide shown to be involved in the neural systems of social cognition, could reverse behavior and structural changes created by EAVS. We focused on the anterior cingulate cortex (ACC) and amygdala subregions to gain a deeper understanding of the neural substrates underlying these effects.

Methods : Male rats were assigned to one of three groups: Control, EAVS, or EAVS+OXT. Between PND12 and PND35, the EAVS group was presented with a daily alternating audio-visual stimulus, while the EAVS+OXT group was additionally intranasally administered OXT. Behavioral measures included the three-chamber social interaction task, the open field test (locomotor activity, anxiety, and stereotyped behaviors), and marble burying. Structural changes in the ACC and amygdala (total, basolateral [BL], central [Ce], medial [Me]) were assessed using the Cavalieri method for estimates of volume, and the optical fractionator method for neuronal quantification. Data were analyzed using ANOVA or Kruskal–Wallis with appropriate post-hoc tests.

Results : EAVS produced significant impairments in social preference and hyperactivity, but did not change anxiety- or stereotypy-related behaviors. Structural analyses indicated that EAVS produced volumetric enlargement of the ACC with no change in neuronal number, indicating volumetric changes resulted from plasticity. EAVS treatment normalized ACC

volume to control levels, rescuing social deficits and hyperactivity. EAVS enlarged total, BL, and Ce volumes and increased the neuronal number in the BL and Ce subregions. OXT did not reverse BL or Ce volumes following EAVS. Thus, while EAVS produced structural enlargement in the ACC and amygdala, OXT specifically modified ACC changes and did not reverse amygdala hypertrophy.

Conclusion : Our results indicate that EAVS during early development caused lasting social impairments, hyperactivity, and neuroanatomical alterations of the ACC and amygdala, which are consistent with phenotypes associated with ASD. Behavioral impairments were ameliorated via intranasal OXT, and OXT normalized volumetric changes in the ACC, implicating OXT in the modulation of the social circuitry. However, OXT did not reverse EAVS-induced hypertrophy of the amygdala, suggesting that the effects occur within a neural circuit-specific action of OXT. The findings support the validity of this rat model as a tool for examining environmental risk factors for ASD, and suggest that the effects of OXT may counteract early-life stimulation of the senses in a manner dependent primarily on frontal cortical circuits and, to a lesser extent, on subcortical circuits. Further investigation is warranted to identify mechanisms linking OXT signaling, ACC–amygdala connectivity, and social behavior.

Keywords : oxytocin; anterior cingulate cortex; amygdala; social behavior; autism spectrum disorder; excessive audio-visual stimulation

Count: 289
Abstract ID: 163
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

Neonatal oxytocin injection can prevent autism like behaviors emergence in maternally separated rats

Submission Author: Sepide Soltani

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Background and Aim : Early life experiences can markedly have a lasting impact on brain development, influencing social and emotional behaviors. Some adverse ones often increase the risk of neurodevelopmental disorders such as autism spectrum disorder (ASD). Previous studies have shown the effect of maternal separation (MS) on developing autism like behaviors in rodents, alongside alterations in neurochemical systems such as the OXT (Oxytocin) pathway. OXT, as an essential neuropeptide for social bonding, has demonstrated potential for influencing ASD. Early OXT administration during the sensitive developmental ages can improve related behavioral deficits. This study investigates the preventive effects of neonatal OXT administration in a MS model by reporting behavioral outcomes in rats.

Methods : Wistar male rats were subjected to daily MS for three hours from PND)Post Natal Day(1 to PND 14, and Subcutaneous OXT injections (0.3 µg/20 µL) were administered on PNDs 1, 3, 5, 7. Behavioral tests such as the three-chamber, marble burying, and elevated plus maze were implemented on PND45.

Results : The findings indicate that MS induces autism-like behaviors such as social behavior deficits and repetitive behaviors and increased anxiety. Injection of OXT during MS, prevented social behavior deficit and stereotypical behavior emergence. However, anxiety behavior was still emerging.

Conclusion : This study suggests that behaviors similar to autism resulting from early life's adverse experiences, such as the lack of proper interaction of the mother-infant, may be prevented by targeting the oxytocin system in infants.

Keywords : Animal model, Autism spectrum disorder, Maternal separation, Oxytocin, Prevention

Count: 290
Abstract ID: 316
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

Using a storytelling robot and emotion regulation of children with autism spectrum disorder.

Submission Author: Leila Kashani Vahid

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Background and Aim : Children with autism spectrum disorder have problems in emotion regulation, and communication and social skills. A storytelling robot has been used to tell stories and communicate with these children.

Methods : Effectiveness of this intervention was evaluated by a semi- experimental experiment with pretest-posttest and a control group. 10 students in the experimental group participated in this program, and the control group did not receive any treatment. Sheilds & Cicchetti Emotion Relation Checklist (1998) was used to measure emotion regulation of the participants.

Results : The obtained data were analyzed using Analysis of the Covariance. The findings showed significant differences ($p<0/5$) between the experimental and the control group in emotion regulation.

Conclusion : Therefore, it can be concluded that using a robot for storytelling is useful for improving emotion regulation skills of these children. Further discussions as well as suggestions for future research are presented.

Keywords : Emotion regulation, training with a storytelling robot, children with autism spectrum disorder.

Count: 291
Abstract ID: 344
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Oral

The effectiveness of a cognitive-visual-motor- electronic training package, "Neuro-light", on behavior problems of children with attention deficit and hyperactive disorder

Submission Author: Leila Kashani Vahid

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Background and Aim : The aim of this study was to evaluate the effectiveness of a cognitive-visual-motor- electronic training package on behavior problems of children with ADHD.

Methods : The research method is a semi-experimental type and a pre-test and post-test design was used along with the control group. The statistical population of the present study was students with attention deficit and hyperactive disorder aged 6-12 years in Shahriar in 1402-1403 academic year, and 20 of these students were selected using a targeted sampling method. Then Conners Rating Scale (1997) was used to measure symptoms of attention-deficit/hyperactivity disorder of the participants. The students were randomly divided into two experimental and control groups. After that, each of the people of the experimental group was individually trained for 8 sessions during 45 minutes with face-to-face and cognitive-visual-motor-electronic training package, which is called "Neuro-light" and was designed, developed and manufactured by the Pars-cognition Company, with collaboration with the computer science department of University of Tehran and used for this group of exceptional children for the first, during the training sessions, while the control group did not receive any intervention. After the completion of the training sessions, from the experimental and control group, Conners Rating Scale was performed as a post-test, and the data obtained were analyzed in SPSS software using Multivariate Analysis of Covariance (MANCOVA).

Results : The findings of the research show the significant the significant difference between experiment and control group in the symptoms of attention deficit/hyperactivity disorder and

indicate effectiveness of the cognitive-visual-motor- electronic training package of symptoms for students with attention deficit and hyperactive disorder.

Conclusion : As the result, it can be concluded that this cognitive-visual-motor-electronic training package increased the symptoms of the participating students and it can be used along with other rehabilitation intervention for improving the symptoms of student with attention deficit and hyperactivity disorder.

Keywords : symptoms of Attention-Deficit/Hyperactivity symptoms, Neuro-light cognitive-visual-motor-electronic training package

Count: 292
Abstract ID: 117
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

The Results of the Multisensory Teaching Method for Persian Language in Bilingual Adolescents with Autism

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Background and Aim : Research on effective methods for teaching individuals with autism is limited. Traditional imitation methods have not yielded significant results for these patients. Multisensory and brain-based educational methods have been claimed to be more effective. This study evaluates the impact of a multisensory teaching method on a bilingual adolescent with autism. The 16-year-old participant, born in England with Iranian parents, spoke English and sought instruction in the Persian language.

Methods : This language learner had previously undergone one year of instruction using imitation methods for learning Persian, which resulted in poor academic progress based on two VKS (Vocabulary Knowledge Scale) and VPMI (Visual, Auditory, Physical, Mental, Intuitive) assessments. The patient received approximately 200 hours (5 sessions per week, 2 hours each session) of multisensory intervention for Persian language education over a period of 6 months. The independent variable of the study was the 200 hours of Persian language instruction using the multisensory teaching method, while the dependent variable was the learner's writing ability assessed through standardized VKS and VPMI tests.

Results : At the end of the intervention, the participant showed significant progress using the mentioned methods. During the teaching process, an increase in the participant's confidence and a decrease in anxiety were observed and recorded. Since the multisensory teaching method, considering brain-based characteristics, was able to strengthen the neural pathways effective for writing in the participant, the progress was significant across all items of the mentioned tests. VKS (66.66%) and VPMI (Visual: 3, Auditory: 3, Physical: 4, Mental: 3, Intuitive: :2). Quantitative analyses revealed significant enhancement in the processing and expressive capability of modality -specific lexical-semantic constructs spanning somatosensory, motor, Visual, and auditory modalities.

Conclusion : Based on the findings from the pre-test and post-test documentation of academic progress, it was determined that this educational method had a significant impact on the participant's academic advancement. Further research is needed to generalize these findings and validate this method in similar cases.

Keywords : Autism Spectrum disorders; Multidisciplinary Teaching Method; Bilingualism; Academic achievement

Count: 293
Abstract ID: 555
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Poster

Prenatal Preeclampsia Induces Sex-Dependent Anxiety, Depression, and Neuroimmune Dysregulation in Offspring

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Background and Aim : Preeclampsia, a major hypertensive disorder of pregnancy, is linked to an increased risk of neuropsychiatric disorders in offspring. However, the underlying sex-dependent neurobiological mechanisms, particularly involving inflammatory and hormonal pathways, remain poorly understood.

Methods : A preeclampsia-like condition was surgically induced in pregnant Wistar rats via reduced uteroplacental perfusion pressure (RUPP) on gestational day 14. Male and female offspring (n=18/sex/group) were assessed at 8 weeks of age. Serum levels of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and testosterone were measured. Anxiety-like and depressive-like behaviors were evaluated using the elevated plus maze (EPM) and tail suspension test (TST), respectively. Hypothalamic-pituitary-adrenal (HPA) axis reactivity was assessed by measuring serum cortisol at baseline, 1, 3, and 6 hours after acute restraint stress.

Results : Male offspring from preeclamptic dams exhibited a significant pro-inflammatory state, with elevated serum IL-6 ($p<0.01$) and hs-CRP ($p<0.05$), alongside reduced testosterone levels ($p<0.001$). Behaviorally, these males showed increased depressive-like behavior in the TST, indicated by longer immobility times ($p<0.001$). In the EPM, while both sexes from the preeclampsia group demonstrated a higher anxiety index than controls ($p<0.001$), a significant sex-by-group interaction revealed that only males spent significantly less time in the open arms ($p<0.001$). In contrast, both male and female offspring of preeclamptic dams displayed a hyperactive HPA axis response, with significantly elevated cortisol levels at 1, 3, and 6 hours post-restraint stress compared to controls ($p<0.001$).

Conclusion : Prenatal exposure to preeclampsia programs neuroendocrine and behavioral deficits in adult offspring in a sex-specific manner. While HPA axis hyperreactivity to stress is

common to both sexes, male offspring are uniquely vulnerable to a triad of testosterone deficiency, low-grade inflammation, and depressive-like behavior. These findings indicate that glucocorticoid-independent mechanisms, specifically a disrupted prenatal immune-androgen crosstalk, underpin the male-specific risk for inflammation-mediated depression. This study highlights the critical role of sex-specific pathways and identifies neuroimmune interactions as a potential therapeutic target for mitigating the long-term neuropsychiatric consequences of prenatal preeclampsia exposure.

Keywords : Preeclampsia; Sex Differences; Depression; Inflammation; HPA Axis; Testosterone

Count: 294
Abstract ID: 123
subject: Development: Neurodevelopmental Disorder (ADHD, Autism, Learning Disorders)
Presentation Type: Oral

Integrative Network and Machine Learning Approach Highlights TLR8 and CASP4 as Critical Genes Associated With Autism Spectrum Disorder

Submission Author: Sara Hosseinpoor

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Background and Aim : Autism spectrum disorder (ASD) is a complex neurodevelopmental condition characterized by heterogeneous genetic, and its pathophysiology remains incompletely understood. This study aims to analyze gene expression profiles in ASD patients by integrating systems biology and machine learning approaches to identify critical gene signatures and biological pathways that are dysregulated in these patients.

Methods : The dataset GSE18123 was analyzed to identify differentially expressed genes (DEGs) in the peripheral blood of ASD patients compared with controls, and based on these DEGs, gene co-expression and protein-protein interaction (PPI) networks were constructed. By analyzing these networks, we identified co-expression gene modules and key genes of the PPI network. In addition, machine learning-based feature selection was applied to prioritize the ASD-associated genes. The integration of the results from network analysis and machine learning revealed critical genes that may be potentially involved in the molecular mechanisms of ASD. Functional enrichment analyses elucidated the biological roles of our findings.

Results : A total of 322 differentially expressed genes (DEGs) were clustered into five co-expression modules, with the yellow module showing the strongest correlation with ASD.

Within this module, TLR8 and CASP4 were identified as critical genes associated with ASD, given their roles as key nodes in the PPI network and their placement within the top 20% of feature selection-prioritized genes. Enrichment analysis of the yellow module genes indicated significant involvement in immune and inflammatory responses, particularly NF-κB signaling, interleukin-1β production, and NOD-like receptor signaling pathways.

Conclusion : The results of this study, by identifying TLR8 and CASP4 as critical genes associated with ASD, highlight the role of inflammatory processes and innate immune responses in the pathophysiology of ASD, and provide valuable insights for the discovery of novel biomarkers and potential therapeutic targets in these patients.

Keywords : Autism spectrum disorder; Systems biology; Machine learning; Co-expression; PPI

Count: 295
Abstract ID: 973
subject: Pain and Sensory Systems: Tactile, Somatosensation and Pain Syndromes
Presentation Type: Poster

Duration-Dependent Effects of Adolescent Morphine Exposure on Baseline Pain Sensitivity in Adult Rats

Submission Author: Kawsar Alami

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Background and Aim : Pain is a prevalent clinical symptom associated with a myriad of diseases. Pain sensitivity varies greatly among individuals and is influenced by genetic, psychological, and environmental factors. Among these, opioid exposure is a key environmental modifier that can disrupt normal pain processing. Morphine misuse during adolescence, a vulnerable stage for neurodevelopment, has been shown to produce long-lasting alterations in brain function. However, it remains unclear whether the duration of adolescent morphine exposure differentially affects pain sensitivity in adulthood. This study aimed to investigate the long-term effects of different durations of morphine exposure during adolescence on baseline pain thresholds in adult rats.

Methods : Male Wistar rats received subcutaneous morphine during adolescence in three exposure paradigms: acute (1 day), subchronic (5 days), and chronic (10 days). A control group received saline. One month after treatment cessation, mechanical allodynia and thermal hyperalgesia were assessed in adulthood using the Von-Frey and Hargreaves plantar tests, respectively.

Results : The results demonstrated a duration-dependent decrease in baseline pain thresholds following adolescent morphine exposure. Subchronic and chronic morphine treatments significantly reduced paw withdrawal thresholds (PWT) and paw withdrawal latencies (PWL) in adult rats compared with controls, indicating increased mechanical and thermal sensitivity. Acute morphine exposure did not significantly alter pain sensitivity. No inter-paw differences were observed within groups.

Conclusion : Adolescent morphine exposure induces persistent, duration-dependent hypersensitivity to mechanical and thermal stimuli in adulthood. These findings suggest that repeated morphine exposure during adolescence may produce long-lasting alterations in pain processing, potentially contributing to chronic pain vulnerability later in life.

Keywords : Pain; Mechanical allodynia; Thermal hyperalgesia; Opioid; Rat

Count: 296
Abstract ID: 905
subject: Pain and Sensory Systems: Tactile, Somatosensation and Pain Syndromes
Presentation Type: Poster

Brain Stimulation in Hypnotechnology: From Conceptual Framework to Clinical Application and Biomarker Identification

Submission Author: Danial Nejadmasoom

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Background and Aim : Enhancing the efficacy of clinical hypnosis is a significant challenge due to inter-individual variability in hypnotizability. A promising, cutting-edge approach involves combining hypnosis with non-invasive brain stimulation (NIBS), for example the SHIFT (Stanford Hypnosis Integrated with Functional Connectivity-targeted Transcranial Stimulation) method integrates Transcranial magnetic stimulation (TMS) with Hypnosis (Faerman, 2024). Furthermore, there is a body of literature on the combination of tDCS with hypnosis (e.g., Perri et al., 2022). However, prior research has predominantly focused on augmenting hypnotizability itself rather than assessing downstream clinical outcomes. This article provides a research trajectory that introduces the conceptual framework of "Hypnotechnology" (Nejadmasoom and Faerman, 2023) and, for the first time, investigates its clinical efficacy and underlying predictive biomarkers.

Methods : The research was conducted in three stages: 1) Conceptual Framework: Proposing "Hypnotechnology" as a synergistic integration of hypnosis and technology. 2) Clinical Proof-of-Concept using brain stimulation with hypnosis: A case study of a 63-year-old male with treatment-resistant Restless Legs Syndrome (RLS) who underwent 10 sessions of hypnosis combined with active cathodal tDCS over the left dorsolateral prefrontal cortex (L-DLPFC). 3) Mechanistic Investigation: A randomized, double-blind, crossover trial in seventeen healthy participants to assess the effect of the same intervention on experimental pain and to identify electroencephalography (EEG) predictors of treatment response using machine learning (K-Means clustering).

Results : The RLS case study demonstrated a substantial and durable clinical improvement, with an 85% reduction in symptom severity, an effect comparable to medication but sustained at a two-month follow-up. The experimental pain study revealed two key findings: first, hypnosis alone (in the sham tDCS condition) produced a statistically significant improvement

in both pain threshold and tolerance, confirming its efficacy. Second, the addition of active tDCS provided no significant additive benefit at the group level. However, our exploratory analysis on EEG records showed that baseline mean power in the 1–30 Hz band predicted "responders" to the active intervention with 90% accuracy.

Conclusion : This research trajectory indicates that while Hypnotechnology is a promising approach, its clinical success likely depends on a personalized medicine framework. The identification of a robust EEG biomarker not only reconciles the divergent findings of our two studies (the RLS patient was likely a "top responder" for integrated intervention) but, more importantly, provides the scientific foundation for the development of a personalized NIBS protocol for enhancing efficacy of hypnosis for pain control. The protocol for such a method could be optimized for each individual based on a simple, pre-treatment EEG screening.

Keywords : Hypnotechnology; Hypnosis; Non-invasive Brain Stimulation (NIBS); Pain Control; EEG; Biomarker

Count: 297
Abstract ID: 945
subject: Pain and Sensory Systems: Tactile, Somatosensation and Pain Syndromes
Presentation Type: Poster

Behavioral Outcomes of Curcumin Liposomal Nanoparticles in Chronic Sciatic Nerve Injury: Superior Alleviation of Neuropathic Pain, Functional Deficits, and Anxiety in Rats

Submission Author: Mohammad Ebrahim Abbaszadeh Kahnamooyi

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Background and Aim : Chronic constriction injury (CCI) of the sciatic nerve induces neuropathic pain, functional impairments, and anxiety-like behaviors in rats. Curcumin offers anti-inflammatory and neuroprotective benefits but is limited by poor bioavailability. Liposomal nanoparticles enhance drug stability, bioavailability, and targeted delivery across biological barriers. This study aimed to evaluate the behavioral efficacy of curcumin liposomal nanoparticles (CLNs) compared to free curcumin in alleviating neuropathic pain, functional deficits, and anxiety in a rat CCI model.

Methods : Male Wistar rats (n=24) were divided into control, CCI, CCI + curcumin (60 mg/kg, i.p.), and CCI + CLNs (40 mg/kg, i.p.) groups. CCI was induced via sciatic nerve ligation, followed by 14 days of treatment. Behavioral assessments included thermal nociception (hot plate test), mechanical allodynia (von Frey test), and sciatic functional index (SFI) on days 14, 21, and 28, with anxiety-like behavior (elevated plus maze, EPM) tested on day 28.

Results : CCI reduced hot plate latency ($p<0.001$ vs. control), indicating thermal hyperalgesia. Both treatments increased latency ($p<0.001$ vs. CCI), with CLNs superior on day 28 ($p<0.05$ vs. curcumin). Mechanical allodynia (von Frey) showed lower thresholds in CCI ($p<0.001$ vs. control); CLNs improved thresholds from day 14 ($p<0.01$), outperforming curcumin on day 28 ($p<0.05$). SFI scores dropped in CCI ($p<0.001$ vs. control); both treatments improved scores ($p<0.001$), with CLNs superior on days 21 and 28 ($p<0.001$ vs. curcumin). EPM on day 28

showed reduced open-arm time/entries in CCI ($p<0.001$ vs. control); both treatments increased these ($p<0.001$ for time, $p<0.05$ for entries), with CLNs more effective ($p<0.01$ vs. curcumin).

Conclusion : CLNs outperform curcumin in reducing neuropathic pain, enhancing functional recovery, and mitigating anxiety in CCI rats, highlighting their therapeutic potential for neuropathy-related behavioral deficits.

Keywords : Sciatic nerve chronic constriction injury, Curcumin liposomal nanoparticles, Neuropathic pain, anxiety-like behaviours

Count: 298
Abstract ID: 265
subject: Pain and Sensory Systems: Tactile, Somatosensation and Pain Syndromes
Presentation Type: Poster

Switchman Hypothesis; A Novel Therapy Based on Trigeminal Nerve Stimulation by Sneezing for Management of Migraine Headache

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Background and Aim : Several lines of evidence have shown that trigeminal nerve plays a central role in pathogenesis of the migraine headache. In the current study we are trying to provide an effective treatment for management of migraine headache based on Switchman Hypothesis of the trigeminal nerve. In this hypothesis, trigeminal nerve is like train railways that a switchman is on the way; that is why it can only transfer one neural message at once.

Methods : We share the experience of two patients with chronic migraine headache who used pepper to suppress their attacks.

Results : Each patient experienced suppressed migraine headache in 72% and 87% of the attacks and the intensity of the pain was extremely lower in the rest of the attacks.

Conclusion : The Switchman Hypothesis could be an effective and safe therapeutic basis for developing targeted therapies for migraine headache and attacks. Further clinical studies are required to shed light on the issue and evaluate the safety and efficacy of different modalities.

Keywords : Migraine headache; Trigeminal nerve; Switchman Hypothesis; Management; Pathophysiology

Count: 299
Abstract ID: 203
subject: Pain and Sensory Systems: Tactile, Somatosensation and Pain Syndromes
Presentation Type: Poster

The role of lipopolysaccharide preconditioning in neuropathic pain after chronic sciatic nerve constriction injury in rats

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Background and Aim : Neuropathic pain due to peripheral nerve injury is one of the major clinical challenges, and there is no definitive and effective treatment for it. Evidence indicates that mild immune system stimulation can act as a protective preconditioning strategy against chronic pain.This study aimed to investigate the effect of lipopolysaccharide (LPS) preconditioning on pain behaviors in a chronic constriction injury (CCI) model of the sciatic nerve in rats.

Methods : Animals were divided into groups including Control, LPS alone (at 0.01, 0.1, and 0.5 mg/kg doses), CCI, and CCI+LPS. A single injection of LPS was administered three days before surgery. Thermal cold allodynia (cold stimulus) and thermal hyperalgesia (hot plate) tests were evaluated on days 7 and 14 after surgery.

Results : CCI surgery induced significant allodynia and hyperalgesia. Preconditioning with low doses of LPS (0.01 and 0.1 mg/kg) significantly reduced these behavioral changes, while the higher dose (0.5 mg/kg) lacked protective effect and even amplified the pain response intensity. LPS alone did not produce changes in pain behaviors

Conclusion : Preconditioning with low doses of LPS may represent a novel approach to reduce behavioral signs of neuropathic pain and highlights the role of mild immune activation in modulating chronic pain resulting from nerve injury.

Keywords : Neuropathic pain ,Lipopolysaccharides, Cytokines

Count: 300
Abstract ID: 827
subject: Pain and Sensory Systems: Tactile, Somatosensation and Pain Syndromes
Presentation Type: Oral

The protective role of β-hydroxybutyrate against behavioral and inflammatory alterations induced by high-fat diet and CFA in male rats

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Background and Aim : Chronic inflammation and metabolic dysfunction contribute to both peripheral and central alterations that affect behavior. High-fat diet and chronic inflammatory pain induced by Complete Freund's Adjuvant (CFA) are known to trigger neuroinflammatory and behavioral disturbances. β-Hydroxybutyrate (BHB) exhibits anti-inflammatory and neuroprotective properties. This study aimed to evaluate the potential of BHB to alleviate behavioral and inflammatory changes induced by a high-fat diet and CFA injection in male rats.

Methods : Thirty-six adult male rats were divided into six groups (n = 6). Animals were fed either a high-fat diet (HFD) or a normal diet (ND) and received intraplantar injections of CFA, vehicle, or saline (200 µL). β-Hydroxybutyrate (BHB) was administered orally for nine days after eight weeks of high-fat diet and one week post-CFA injection. Paw withdrawal latency (PWL) was recorded on days 0, 7, 14, and 21 to evaluate peripheral inflammation. Depression-like behavior, including anhedonia, was assessed using the sucrose preference test (SPT).

Results : CFA-injected showed significantly reduced paw withdrawal latency (PWL) on days 0, 7, 14, and 21. High-fat diet (HFD) rats exhibited more pronounced depression-like behavior compared to normal diet (ND). ?-Hydroxybutyrate (BHB) treatment alleviated the PWL in the CFA group and improved depression-like behavior in HFD-fed rats.

Conclusion : β-Hydroxybutyrate attenuates inflammation- and high-fat diet-induced behavioral alterations. These results showed its potential as a therapeutic agent in metabolism and neuroinflammation conditions

Keywords : •High-fat diet • CFA • Depression-like behaviors • Peripheral inflammation

Count: 301
Abstract ID: 950
subject: Pain and Sensory Systems: Vision
Presentation Type: Poster

The Impact of Feedback Masking on Face Recognition across Occlusion Levels

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Background and Aim : Visual processing arises from dynamic interactions between feedforward and feedback signals across hierarchically organized visual areas. Previous studies have shown that when visual stimuli are ambiguous or partially occluded, recurrent feedback from higher-level cortical regions contributes to reconstructing incomplete representations, thereby enhancing perceptual accuracy. However, it remains unclear whether the contribution of feedback is consistent across different levels of occlusion.

Methods : To address this question, we employed a backward masking paradigm combined with a two-alternative forced-choice (2-AFC) task. Participants briefly viewed occluded face stimuli (35 ms) presented under different levels of occlusion (0%, 15%, 30%, 45%, 60%, 75%) and were asked to choose which of two intact faces matched the sample. To evaluate the magnitude of feedback involvement, some trials included variable stimulus onset asynchronies (SOAs) between the target and a phase-scrambled mask, whereas in the no-mask condition, the same temporal delays were applied without the mask presentation.

Results : Our results showed that recognition performance decreased as the degree of occlusion increased. Another finding was that as the SOAs increased, the accuracy of the subjects improved. At 35 and 50 ms SOAs, the mask caused greater interference with feedback processing, while at 100, 150, and 250 ms SOAs, it had no significant effect on feedback processing. However, the SOA-dependent performance profiles varied across different occlusion levels and showed differences between the mask and no-mask conditions.

Conclusion : These findings demonstrate that feedback processing contributes differentially to visual perception depending on the complexity and temporal characteristics of the sensory input. Together, this study provides a refined framework for quantifying feedback dependence in human face recognition

Keywords : Occlusion; Stimulus onset asynchrony (SOA); Face recognition

Count: 302
Abstract ID: 829
subject: Pain and Sensory Systems: Vision
Presentation Type: Poster

Chromatic Threshold Stability in the Yellow-Blue Axis

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Background and Aim : A previous study by Krauskopf et al. (1992) investigated the role of adaptation on the S – (L+M) axis in four subjects. Building on these findings, the current study examined this phenomenon in a sample of twelve Iranian women.

Methods : Twelve female participants with normal or corrected-to-normal vision completed five sessions, each consisting of 20 color stimuli (10 blue and 10 yellow). Participants performed a two-alternative forced-choice (2AFC) color discrimination task, identifying each stimulus as either blue or yellow. Stimuli were presented in random order, with each color appearing 20 times per condition. In the first condition, colors were presented without adaptation. In the adaptation condition, a pure yellow stimulus was shown for 30 seconds at the beginning of each session and for 10 seconds after each response to induce adaptation. RGB space with a fixed 25-unit step along the blue-yellow axis was used. These points create a continuous range from (0, 0, 255) to (225, 225, 255) and from (255, 255, 0) to (255, 225, 225). The discrete indices (0 to 19) represent the position of the stimulus, which is modeled as the continuous derived yellow strength used to measure the detection threshold. (p=0.75). A sample subject (subject 1): Yellow adaptation caused a rightward shift in her threshold (+0.087),reflecting fatigue and the need for stronger stimuli. Crucially, her sensitivity (+6.6) also increased, suggesting the visual system compensated by sharpening its discriminative power.

Results : Psychophysical analyses demonstrated that chromatic adaptation to yellow significantly shifted the color discrimination threshold. The mean threshold increased from No Adaptation (Mean=0.6915, STD=0.0613) to Adapted (Mean=0.7716, STD=0.0877). A paired t-test confirmed this shift as statistically significant ($t(11)=-2.769$, $p=0.018$), with a large effect size (Cohen's $d=-0.799$). This threshold increase (a rightward shift of −0.0801) was visualized in the psychometric curve, and the overall data distribution in the box plot shifted toward higher values. These results indicate that, following adaptation, subjects required a stronger physical stimulus to detect the target yellow color. Yellow adaptation caused a statistically significant

rightward shift in the group's threshold (x75) and bias (b) ($p<0.05$), indicating neural fatigue. The group's overall sensitivity (a) remained unchanged ($p=0.92$), indicating that discrimination acuity was preserved.

Conclusion : The threshold shift confirms the occurrence of fatigue in the Yellow-Blue Opponent Channel, consistent with the principles of Krauskopf et al. (1992). Notably, the data reveal substantial individual variability, ranging from severe fatigue to strong compensation (increased sensitivity), which suggests the operation of dynamic and distinct neural strategies for homeostasis. Persistent fatigue among spectacle-wearing subjects and extreme fatigue in subjects with uncorrected visual impairment indicate that sensory threshold stability is highly susceptible to cognitive load. These findings highlight the close link between basic sensory mechanisms and higher-order brain processes, suggesting that adaptation functions as a neuro-cognitive phenomenon that extends beyond photoreceptor depletion. The authors thank Prof. Behrad Noudoost for his mentorship. This poster was developed as a project for his course at Isfahan University of Medical Sciences, and his guidance and suggestions were instrumental to its completion.

Keywords : color discrimination; adaptation; chromatic mechanism

Count: 303
Abstract ID: 400
subject: Pain and Sensory Systems: Auditory and Vestibular
Presentation Type: Poster

A Review of ERP Components in Individuals with Hearing Loss: Evidence from Neuroscientific Studies

Submission Author: Nayiere Mansouri

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Conclusion : Background: Event-related potentials (ERPs), as a non-invasive measure of neural processing, play a significant role in the study of auditory function in individuals with hearing loss. Research has shown that both the latency and amplitude of ERP components are markedly altered in this population. Methods: A literature search was conducted for Persian and English articles published between 2000 and 2025 using the keywords hearing loss, hearing aid, cochlear implant, and event-related potentials. Results: Several ERP components have been found to be affected in individuals with hearing loss. Among these, N1 and P2 reflect the early cortical processing of sound. In hearing-impaired individuals, these components often exhibit delayed latency and abnormal amplitude changes. The Mismatch Negativity (MMN), which indexes the brain's ability to detect subtle changes in auditory stimuli, is frequently reduced in amplitude in this population, indicating deficits in speech discrimination and auditory change detection. The P300 component, which reflects attentional and cognitive processing of auditory stimuli, typically shows delayed latency and reduced amplitude in individuals with hearing loss, suggesting impaired auditory cognitive processing. Furthermore, alterations in N400 and Late Positive Potential (LPP) have been reported in relation to language processing, which may reflect difficulties in lexical-semantic comprehension. Conclusion: Altered ERP patterns in individuals with hearing loss demonstrate that auditory impairment affects not only early auditory processing but also higher-order cognitive and linguistic functions. These findings highlight the potential utility of ERP measures in guiding rehabilitation programs, optimizing the use of hearing aids.

Keywords : hearing loss, hearing aid, cochlear implant, and event-related potentials

Count: 304
Abstract ID: 618
subject: Pain and Sensory Systems: Chemical Senses: Olfactory and Taste
Presentation Type: Poster

Aromachology: More Than a Scent – A Narrative Review of The Psychology of Smells

Submission Author: Faezeh Rezaei

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Background and Aim : Aromachology—the scientific study of how odors influence psychology and behavior—extends beyond fragrance appreciation, offering insights into the sensory-affective interface. While often perceived as peripheral, olfactory stimuli have direct access to limbic and cortical circuits implicated in memory, emotion, and cognition. This narrative review explores aromachology's evolving role in neuroscience, synthesizing evidence across experimental, affective, and cognitive domains.

Methods : Relevant literature was sourced via PubMed, Scopus, and Web of Science using specific search terms. Studies were included if they examined scent-induced psychological or behavioral effects in human participants. Findings were grouped into thematic categories: emotion regulation, memory evocation, attentional bias, and multisensory integration.

Results : Aromachological experiments consistently demonstrate that specific scents modulate mood, reduce anxiety, and influence cognitive bias—often without conscious awareness. Neuroimaging studies reveal differential activation of the amygdala, orbitofrontal cortex, and hippocampus in response to emotionally valenced odors. Beyond transient effects, long-term olfactory exposure has been linked to changes in attentional networks and stress reactivity. Multisensory experiments suggest that pairing scent with sound or vision enhances immersive processing and memory encoding.

Conclusion : Aromachology is an underutilized but powerful lens through which to explore sensory neuroscience. Its influence on emotion and cognition is immediate, embodied, and neurobiologically distinct. Future research integrating aromachology with neurotechnologies like VR, fNIRS, and EEG may unlock new therapeutic and diagnostic tools rooted in the sense of smell.

Keywords : Aromachology; Smell; Emotion; Behavior; Cognition; Olfaction

Count: 305
Abstract ID: 404
subject: Pain and Sensory Systems: Sensory and Sensory Integration Disorders
Presentation Type: Oral

Investigation and Comparison of the Pharmacokinetic Properties of the Most Important Newly Synthesized Analogs of Selective μ-Opioid Receptor (mOR) Agonists

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Background and Aim : Opioid analgesics remain among the most effective agents for the treatment of severe acute and chronic pain; however, their widespread use is limited by life-threatening adverse effects, including respiratory depression, tolerance, and dependence. The μ-opioid receptor (μOR) is the primary target mediating both analgesic efficacy and side effects, making it a critical focus for the development of safer opioid therapeutics. This thesis employed an in silico approach to evaluate and compare the pharmacokinetic and pharmacodynamic properties of key synthetic μOR agonist analogs, with the objective of identifying candidates that provide potent analgesia while minimizing adverse effects.

Methods : Using molecular docking, pharmacokinetic prediction tools (including pkCSM, SwissADME, and PASS), and toxicity modeling platforms, three representative compounds—fentanyl, PZM21, and the novel derivative FBD3—were systematically analyzed.

Results : Previously docking studies confirmed strong binding affinities of all compounds toward the ?OR binding pocket, with FBD3 exhibiting favorable interaction patterns consistent with G-protein–biased signaling. In current study, predicted ADMET profiles indicated acceptable oral bioavailability and blood–brain barrier permeability for fentanyl and PZM21, while FBD3 displayed lower central penetration, aligning with its proposed reduced risk of respiratory suppression. Toxicity predictions further suggested that FBD3 carries a safer profile compared with fentanyl with diminished hepatotoxicity and cardiotoxicity potential.

Conclusion : Collectively, these findings highlight FBD3 as a promising lead compound with the potential to retain strong analgesic activity while reducing the liability of opioid-induced side effects. The results support the utility of computational drug design strategies in accelerating the identification and optimization of selective μOR agonists. Future work should incorporate molecular dynamics simulations and experimental validation to confirm the predicted pharmacological advantages of FBD3 and related analogs

Keywords : Mu-opioid receptor agonist, PZM21, FBD3, Beta-arrestin, pharmacological profile, Pharmacokinetics

Count: 306
Abstract ID: 200
subject: Pain and Sensory Systems: Sensory and Sensory Integration Disorders
Presentation Type: Oral

Investigating the Alteration of Somatic Pain Under Ulcerative Colitis

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Background and Aim : Ulcerative colitis (UC) is a chronic, painful inflammatory disease that can affect normal body processes. Considering that pain is one of the most bothersome issues in patients with UC, in the present study, we investigated the effect of UC on somatic pain thresholds in a rat model.

Methods : Twelve male Wistar rats were equally divided into two groups: Sham and UC. UC was induced in the rats by intrarectal injection of 1 cc of 3% acetic acid after anesthesia. The same procedure was performed using distilled water for the sham group. After 72 hours, the Von Frey digital test was performed to measure the mechanical allodynia threshold, and the acetone test was performed to assess cold allodynia in the left and right paws separately. Plasma levels of tumor necrosis factor-alpha (TNF-alpha) and interleukin-6, which are inflammatory cytokines, were measured using ELISA at the end of the study. Statistical significance was set at $P < 0.05$.

Results : In the acetone test, the summation data of the right and left paws significantly increased in the UC group ($P < 0.05$). The digital mechanical allodynia threshold in animals with UC showed a significant decrease in each paw and in the total data compared to the sham group (at least $P < 0.05$). In addition, under UC, the amount of TNF-alpha and interleukin-6 increased ($P < 0.05$).

Conclusion : The results showed that UC caused somatic hyperalgesia in rats, which was associated with an increase in pro-inflammatory cytokines and could interfere with the process of increasing sensitivity to cold and mechanical pain stimuli. Simultaneously, based on the tests performed, this hyperalgesia affects both fast and slow pain, indicating a significant effect of inflammation in UC samples. Further research to clarify the effect of UC on somatic allodynia based on central and/or peripheral sensory nerve alterations can be helpful for its management.

Keywords : Ulcerative colitis; hyperalgesia; allodynia; pain.

Count: 307
Abstract ID: 98
subject: Motor Systems and Movement Disorders: Motor Neurons and Muscle
Presentation Type: Poster

Galvanic VEMP as a Functional Biomarker: Assessing Spinal Cord Involvement in Progressive Myelopathies and Neuromuscular Disease

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Conclusion : The accurate and early assessment of spinal cord functional integrity in progressive myelopathies and other neuromuscular diseases remains a significant diagnostic challenge. Conventional methods, including clinical scales and structural imaging, are essential but may not fully capture the extent of functional impairment, particularly in the subclinical stages of disease. Galvanic Vestibular-Evoked Myogenic Potential (gVEMP) testing has emerged as an efficient, non-invasive electrophysiological modality that provides a direct functional assessment of the vestibulospinal tract. This review synthesizes the existing evidence to highlight the optimization of gVEMP testing for the detection, monitoring, and prognostication of spinal cord involvement in these conditions. The clinical utility of gVEMP is powerfully demonstrated in specific myelopathies. For instance, in HTLV-1-associated myelopathy (HAM), gVEMP testing reveals a clear, progressive pattern of spinal cord damage. This spectrum ranges from delayed response latencies in clinically asymptomatic carriers to the complete absence of responses in patients with severe myelopathy. This strong correlation establishes the potential of gVEMP as a robust prognostic marker for disease progression. Furthermore, in patients with schistosomal myeloradiculopathy, significant delays in gVEMP responses have been shown to directly correlate with the severity of gait dysfunction, thus providing objective functional data that extends beyond standard neurological examinations. A comprehensive analysis of the literature confirms that delayed latency and/or reduced amplitude of gVEMP responses are consistent and reliable findings across a wide range of myelopathies. The magnitude of these electrophysiological abnormalities demonstrates a high correlation with clinical severity and functional disability. A key advantage of this technique is its high diagnostic accuracy and its unique ability to identify subclinical pathology. gVEMP can detect nascent functional deficits in individuals who are not yet clinically symptomatic, thereby providing a critical window for early diagnosis and potential therapeutic intervention. By delivering a quantitative and objective measure of motor pathway function, this test provides valuable data complementary to the structural information obtained from MRI.

gVEMP testing represents an optimized, non-invasive, and cost-effective method for the functional assessment of spinal cord motor pathways. Its demonstrated ability to detect subclinical deficits, monitor disease progression, and correlate with clinical severity makes it a powerful tool in clinical neuroscience. The integration of gVEMP into the standard diagnostic and monitoring protocols for myelopathies can significantly enhance patient management, improve prognostication, and provide objective endpoints for evaluating therapeutic efficacy in clinical trials.

Keywords : gVEMP; Spinal Cord; Myelopathy; Functional Biomarker; Neuromuscular Disease

Count: 308
Abstract ID: 520
subject: Motor Systems and Movement Disorders: Motor Neurons and Muscle
Presentation Type: Poster

Is mitochondrial transplantation effective in multiple sclerosis?

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Background and Aim : Multiple sclerosis (MS) is a chronic autoimmune disease of the central nervous system (CNS) characterized by demyelination, neuroinflammation, and progressive axonal degeneration. Accumulating evidence implicates mitochondrial dysfunction as a central player in the pathogenesis of MS, linking impaired bioenergetics to demyelination, oxidative stress, and neurodegeneration. Current therapeutic strategies primarily target immune modulation and attenuation of inflammatory processes; however, they have limited capacity to promote remyelination or repair axonal damage. In this context, mitochondrial transplantation (mitotherapy) has recently emerged as a novel neuroprotective and regenerative approach.

Methods : In the present study, experimental demyelination was induced in male C57BL/6 mice by administration of a 0.2% cuprizone diet for 12 weeks, resulting in robust demyelination in the corpus callosum and hippocampus. Motor performance was assessed using the Beam Walk and Open Field paradigms, while demyelination was confirmed by Luxol Fast Blue (LFB) staining. Functional mitochondria were isolated from mouse liver, validated by MTT assay and mitochondrial swelling analysis, and subsequently transplanted locally (single dose, 20 million).

Results : Two weeks following transplantation, behavioral analyses demonstrated improvements in motor coordination, balance. Histological evaluation confirmed enhanced remyelination, while biochemical assays revealed reduced oxidative stress markers, including reactive oxygen species (ROS) and malondialdehyde (MDA).

Conclusion : These findings indicate that mitotherapy restores mitochondrial function, mitigates oxidative stress, and promotes remyelination in the cuprizone model of MS. Mitochondrial transplantation may therefore represent a promising therapeutic strategy for MS and related neurodegenerative disorders, warranting further preclinical and translational investigation.

Keywords : Multiple sclerosis ; Mitochondrial Transplantation ; Cuprizone

Count: 309
Abstract ID: 523
subject: Motor Systems and Movement Disorders: Movement Disorders (Parkinson, Huntington's, ALS, Ataxia, edication-induced Movement Disorders)
Presentation Type: Poster

Cinnamaldehyde Ameliorates 6-Hydroxydopamine-Induced Motor Deficits in a Rat Model of Parkinson's Disease

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Background and Aim : Insights into Oxidative Stress and Apoptotic Pathways Parkinson's disease (PD) is a progressive and debilitating neurodegenerative disorder characterized by the gradual loss of dopaminergic neurons and the accumulation of α-synuclein aggregates. It is associated with a range of motor and non-motor symptoms that severely impact quality of life. Evidence suggests that cinnamaldehyde (Cin), the primary bioactive compound in cinnamon, possesses neuroprotective properties. However, its efficacy and molecular mechanisms in the context of PD remain unexplored.

Methods : This study investigated the effects of Cin on motor impairments induced by 6-hydroxydopamine (6-OHDA) in Wistar rats, a widely used model of PD. Rats were administered Cin (10 and 100 mg/kg) daily, starting 1 hour after 6-OHDA injection (20 µg/ each rat), for a duration of 2 weeks. Motor function was assessed using the rotarod treadmill, beam walking, open field, and apomorphine-induced rotation tests.

Results : Our findings revealed that Cin significantly improved the motor deficits induced by 6-OHDA.

Conclusion : In conclusion, Cin shows promise as a potential therapeutic agent for PD and warrants further investigation to fully elucidate its mechanisms and clinical applications.

Keywords : 6-Hydroxydopamine ; Cinnamaldehyde ;Parkinson's Disease

Count: 310
Abstract ID: 806
subject: Motor Systems and Movement Disorders: Movement Disorders (Parkinson, Huntington's, ALS, Ataxia, edication-induced Movement Disorders)
Presentation Type: Oral

Efficacy of the COGMO Protocol: A Novel Cognitive-Motor Intervention for Balance and Coordination in Elderly Parkinson's Patients - A Pilot Study

Submission Author: MANOCHEHR GHALKHANI

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Background and Aim : The global population is rapidly aging, and Parkinson's disease (PD) presents a major health challenge within this demographic [1, 2]. Impaired balance and coordination are disabling symptoms of PD, leading to reduced quality of life and an increased risk of falls. Traditional rehabilitation approaches alone are often insufficient to fully address these complex issues. In this context, cognitive-motor interventions have emerged as a novel paradigm, simultaneously engaging cognitive and motor neural systems and showing promise for ameliorating these symptoms [3, 4]. This study was designed to investigate the feasibility and preliminary effects of a structured protocol named "COGMO" on balance and coordination indices in a small group of elderly patients with Parkinson's disease.

Methods : This pilot study employed a pre-test/post-test design with six Parkinson's disease patients (Hoehn & Yahr stages 2-3). Participants underwent the 8-week COGMO protocol, involving thrice-weekly, 45-minute sessions of progressive cognitive-motor dual-task training. Outcomes were assessed pre- and post-intervention using the UPDRS-III, Timed Up and Go test, and Berg Balance Scale. Given the small sample, data were analyzed with descriptive statistics and paired-samples t-tests in SPSS, with a significance level set at $p < 0.05$.

Results : The 8-week COGMO intervention demonstrated statistically significant improvements across all outcome measures in 6 PD patients. UPDRS-III scores decreased significantly from 30.2±1.9 to 23.8±2.1 ($p=0.003$). TUG test performance improved from 21.5±1.8s to 15.7±1.4s ($p=0.001$), while BBS scores increased from 41.5±2.3 to 49.8±1.9

(p=0.002). All p-values <0.01 indicate strong statistical significance for these improvements in motor symptoms, mobility, and balance.

Conclusion : This pilot study demonstrates that the COGMO protocol is a feasible and safe intervention for elderly Parkinson's patients, producing statistically significant and clinically meaningful improvements in motor function, gait speed, and balance. The approximately 6-second improvement in TUG scores notably exceeds established minimal clinically important difference thresholds. These findings align with existing literature on dual-task training benefits, suggesting potential neural pathway strengthening and enhanced executive function. While limitations include small sample size and lack of control group, these promising results justify proceeding to larger randomized controlled trials to validate efficacy and determine effect sizes.

Keywords : Parkinson's Disease, Cognitive-Motor Training, COGMO Protocol

Count: 311
Abstract ID: 596
subject: Motor Systems and Movement Disorders: Movement Disorders (Parkinson, Huntington's, ALS, Ataxia, edication-induced Movement Disorders)
Presentation Type: Oral

Ketamine Ameliorates Motor Deficits and Anxiety-Like Behavior in Essential Tremor

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Background and Aim : Essential Tremor (ET) is a common movement disorder involving cerebellar dysfunction. This study investigated the potential therapeutic effects of ketamine, an NMDA receptor antagonist with neuroprotective properties, in a harmaline-induced model of ET.

Methods : Using a well-established ET model in mice, we administered a subanesthetic dose of ketamine (8 mg/kg). Comprehensive behavioral assessments included motor coordination (wire grip test), locomotor activity, anxiety-like behavior (open field test), and tremor evaluation.

Results : Ketamine treatment demonstrated significant beneficial effects on specific motor and behavioral parameters. It effectively improved muscle strength deficits and normalized anxiety-like behaviors in harmaline-treated animals. Furthermore, ketamine administration restored exploratory activity and mobility patterns in the tremor model.

Conclusion : Our findings indicate that ketamine provides meaningful therapeutic benefits in ET by improving motor function and reducing anxiety-related symptoms. These results support the potential of ketamine as a complementary treatment approach for managing essential tremor manifestations.

Keywords : Essential Tremor, Ketamine, Motor Symptoms, Anxiety, Neuroprotection

Count: 312
Abstract ID: 532
subject: Motor Systems
and Movement Disorders: Movement Disorders (Parkinson, Huntington's, ALS, Ataxia, edication-induced Movement Disorders)
Presentation Type: Poster

Synergistic Neuroprotective Effects of Quercetin and Apigenin in a 6-OHDA-Induced Rat Model of Parkinson's Disease: Integrated Behavioral, Biochemical, and Molecular Evidence

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Background and Aim : Parkinson's disease (PD) is a progressive neurodegenerative disorder caused by the degeneration of dopaminergic neurons in the substantia nigra pars compacta, leading to striatal dopamine depletion and motor dysfunction. Oxidative stress and neuroinflammation are major contributors to PD pathogenesis. Although several natural polyphenols have shown individual neuroprotective potential, their combined effects remain poorly understood. This study investigated the therapeutic efficacy of quercetin and apigenin, administered alone and in combination, in a rat model of PD induced by 6-hydroxydopamine (6-OHDA).

Methods : Male Wistar rats (n = 10 per group) were divided into five groups: Sham (vehicle), PD (6-OHDA lesion), PD + quercetin, PD + apigenin, and PD + quercetin + apigenin. Unilateral lesions were induced via stereotaxic injection of 6-OHDA into the medial forebrain bundle. Treatments were administered intraperitoneally according to a fixed schedule for three weeks. Behavioral assessments included apomorphine-induced rotations, rotarod performance, and open-field activity. Dopamine levels and oxidative stress biomarkers (MDA, GSH, SOD activity) were measured biochemically. Molecular analyses included qRT-PCR and western blotting for tyrosine hydroxylase (TH), Bcl-2, Bax, caspase-3, and pro-inflammatory cytokines (TNF-α, IL-1β).

Results : Both quercetin and apigenin significantly reduced rotational asymmetry, improved rotarod performance, and enhanced exploratory behavior compared to untreated PD rats.

Biochemically, treatments restored striatal dopamine levels, reduced lipid peroxidation, and enhanced endogenous antioxidant defense. Molecular findings demonstrated preservation of TH expression, increased Bcl-2, and suppression of Bax, caspase-3, TNF-?, and IL-1?. Notably, the combined administration of quercetin and apigenin yielded greater neuroprotection than either compound alone, suggesting a synergistic effect.

Conclusion : This study provides the first integrated evidence of the synergistic neuroprotective effects of quercetin and apigenin in a 6-OHDA-induced rat model of PD. By simultaneously attenuating oxidative stress, apoptosis, and neuroinflammation, these polyphenols not only improved motor outcomes but also preserved nigrostriatal integrity at biochemical and molecular levels. Given their natural origin, favorable safety profile, and multi-targeted mechanisms, quercetin and apigenin may represent promising adjunctive strategies for PD management. These findings highlight the therapeutic potential of combined polyphenolic interventions in neurodegenerative disorders and warrant further translational and clinical investigation.

Keywords : Parkinson’s disease, neuroprotective, synergistic, quercetin, apigenin

Count: 313
Abstract ID: 564
subject: Integrative system: Brain Immune System and Brain Tumors
Presentation Type: Poster

Unveiling the potential of microRNAs in glioblastoma diagnosis and therapy

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Conclusion : Abstract Background and Aim: Glioblastoma (GBM) is the most common and most aggressive adult brain tumor. It demonstrates a bad prognosis and is extremely difficult to treat, and gives a poor response to surgery, chemotherapy and radiotherapy. Significant insights regarding the histopathology of GBM have slowly translated to improvements in treatment. Non-coding small RNA's known as microRNAs or miRNAs, are key regulators of mRNA expression and cell processes, comprising cell division and growth, apoptosis, angiogenesis, invasion, and evasion of therapeutic regimens. miRNAs are also able to circulate in body fluids and act as tumor promoters or tumor suppressors, thus constituting potential diagnostic, prognostic and therapeutic markers. The purpose of this review is to cover the recent literature concerning microRNAs and GBM. Methods: From 2019 to 2024, extensive searches were performed in PubMed on the terms "Glioblastoma" and "microRNA". 191 documents were obtained and focused on the most relevant documents pertaining to the diagnostic, prognostic, and therapeutic implications of miRNAs in GBM. Results: Recent research points out that dysregulated miRNAs cause malignancy in GBM by interfering with several pathways: angiogenesis, stem cells, and therapeutic interventions. Certain miRNAs like miR-17-3p, miR-222, miR-340, and miR-130a-3p were explained in previous works to serve as important malignancy regulators. There are many different ways to inhibit or promote the growth of a tumor, and several of these ways have been illustrated in research. Empirical investigations have shown that blocking or reintroducing these miRNAs decreases the ability of the cells to proliferate and enhances apoptotic activity, making the cells more susceptible to conventional therapy. In addition to that, exosomal and circulating miRNAs are being studied and can serve as non-invasive methods for early diagnosis of GBM, tumor subclassification, and assessment of therapeutic response. Novel approaches that involve the use of nanoparticles are being designed to solve the challenges that miRNA-based therapies pose in crossing the blood-brain barrier. Conclusion: miRNAs serve multiple functions in the biology of GBM and constitute promising biomarkers and targets for therapy. In clinical practice, the integration of miRNA-based diagnostics and therapeutics promises individual treatment approaches, although further experimental and clinical studies are required to establish their safety and efficacy.

Keywords : Glioblastoma; microRNA; biomarkers; therapy resistance; angiogenesis; personalized medicine

Count: 314
Abstract ID: 431
subject: Integrative system: Brain Immune System and Brain Tumors
Presentation Type: Poster

Roles of Sirtuin-1 in Glioblastoma: From Tumor Progression to Targeted Therapy

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Conclusion : Introduction: Glioblastoma (GBM) is the most prevalent and aggressive type of brain cancer in adults, presenting significant treatment problems due to its rapid proliferation, adverse prognosis, and treatment resistance. Existing treatments are still limited to surgery, chemotherapy, and radiotherapy. Evidence indicates that epigenetic mechanisms, including histone deacetylases (HDACs) and, in particular, sirtuins (SIRTs), are crucial in gliomagenesis. Among them, SIRT1, a member of the family, is a crucial regulator of genomic stability and apoptosis, serving as a dual effector in cancer cells. The goal of this review is to investigate the function of SIRT1 in GBM. Methods: From 2000 to 2025, we searched the keywords "Sirtuin-1" and "glioblastoma" in a number of databases, including PubMed, Scopus, and Google Scholar. Results: Studies have shown that SIRT1 plays a key role in growth, cell survival, and apoptosis in GBM. Its activation or overexpression resulted in the downregulation of adhesion molecules VCAM-1 and ICAM-1, inhibition of monocyte adhesion, and regulation of tumor microenvironment signaling pathways. Small molecule activators of SIRT1, such as Comp 5, induced cell death through autophagy and mitophagy and inhibited cell proliferation. SRT2183, another activator, prompted cell cycle arrest, apoptosis, and growth suppression in glioma cells by increasing the pro-apoptotic protein Bim and decreasing Bcl-2 and Bcl-xL. The compound also induced autophagy and affected acetylation and phosphorylation of NF-κB and STAT3. Conclusion: Activation of SIRT1 using small molecule compounds such as Comp 5 and SRT2183 suppresses GBM cell growth, triggers apoptosis and autophagy/mitophagy, and regulates signaling pathways associated with the tumor microenvironment. These findings suggest that SIRT1 may serve as a potential therapeutic target in GBM, and its activation could facilitate the development of novel therapeutic strategies and improve treatment response in patients with GBM

Keywords : Sirtuin-1, Glioblastoma, Therapeutics

Count: 315
Abstract ID: 209
subject: Integrative system: Brain Immune System and Brain Tumors
Presentation Type: Poster

Exploring the Innate Immune Microenvironment in Glioblastoma and the Promise of Oncolytic Virotherapy

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Conclusion : The microenvironment of glioblastoma (GBM) is primarily characterized by the presence of immunosuppressive innate immune cells. Resident microglia and infiltrating tumor-associated macrophages (TAMs) secrete factors such as interleukin-10 and TGF-β, which reprogram cellular metabolism and reduce T-cell cytotoxicity. Additionally, these innate cells activate "don't eat me" pathways and recruit myeloid-derived suppressor cells (MDSCs), further enhancing the tumor's ability to evade the immune system. As a result, GBM typically presents as a 'cold' tumor, where the blood-brain barrier and cellular heterogeneity hinder drug delivery and immune cell access. Consequently, standard treatments like checkpoint inhibitors and CAR T-cell therapies have demonstrated only marginal effectiveness against GBM so far. Oncolytic viruses (OVs), which are engineered to selectively infect tumor cells, also stimulate anti-tumor immune responses. For instance, a genetically modified non-pathogenic poliovirus is currently in clinical trials for recurrent glioblastoma. Once these viruses enter tumor cells, they replicate, causing oncolysis and releasing tumor antigens along with pro-inflammatory signals. These signal molecules attract dendritic cells, macrophages, and T cells to the tumor site, effectively transforming the typically “cold” microenvironment of GBM into a more immunogenic one. Therefore, some researchers consider oncolytic virotherapy as a form of cancer immunotherapy that can help reshape the suppressive environment around tumors. In addition to directly lysing tumor cells, OVs can be engineered to carry immune-stimulatory payloads. For example, an oncolytic adenovirus that encodes the cholesterol transporter ApoA1 reverses immune suppression in GBM by promoting cholesterol efflux in TAMs, thus restoring their ability to engulf pathogens and enhancing tumor-specific T-cell immunity. Similarly, oncolytic viruses based on HSV-1 and adenoviruses are being combined with checkpoint inhibitors or chemotherapy to boost anti-glioma responses. These combination strategies are beginning to demonstrate systemic anti-tumor immunity in treated patients and have led to significant advancements, such as the approval of G47Δ (an engineered HSV) for recurrent GBM in Japan. Looking ahead, future research will prioritize improving the delivery of therapies across the blood-brain barrier, customizing treatments based on patient biomarkers, and further incorporating immunomodulatory strategies. For example, OVs may be designed

to carry cytokines (e.g., IL-12, IFN-β) or checkpoint ligands. By transforming the immunosuppressive GBM niche into an inflamed environment, these strategies hold promise for developing more effective and durable therapies for this aggressive brain tumor.

Keywords : glioblastoma; oncolytic virus therapy; tumor microenvironment; innate immunity; immunotherapy

Count: 316
Abstract ID: 1008
subject: Integrative system: Neural Circuits and Connectivity
Presentation Type: Oral

Frontotemporal Network Dynamics in Contextual Representation

Submission Author: Faezeh Shafiei

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Background and Aim : The frontotemporal network, comprising the inferior temporal cortex (ITC) and the ventrolateral prefrontal cortex (vlPFC), is essential for object recognition and higher-order visual processing. However, how contextual factors influence neural communication within this network remains poorly understood. This study investigates how context predictability modulates neural responses across ITC and vlPFC, providing insight into the neural mechanisms that support flexible visual cognition.

Methods : We performed simultaneous single-unit recordings in ITC and vlPFC of macaque monkeys during a passive viewing task. Context was manipulated by varying the predictability of stimulus pairs across blocks. In “Repeating” blocks, pairs repeated on 80% of trials and alternated on 20%; in “Alternating” blocks, these probabilities were reversed. Stimulus categories were randomized within blocks to isolate effects of repetition and expectation, allowing an independent assessment of contextual influence on neural activity.

Results : Neural responses in both ITC and vlPFC were significantly modulated by block context. F-statistics revealed robust differences in firing rates across conditions, and support vector machine (SVM) classifiers reliably distinguished between contextual states based on population activity patterns. These results indicate that contextual information can be decoded from both regions, suggesting dynamic modulation of neural responses by task context.

Conclusion : Our findings indicate that both ITC and vlPFC integrate contextual information to dynamically modulate visual representations. Together, our results highlight the frontotemporal network’s role in flexibly encoding contextual information, providing new insights into the neural mechanisms underlying adaptive visual cognition.

Keywords : Frontotemporal network; Inferior temporal cortex; Ventrolateral prefrontal cortex; Contextual modulation

Count: 317
Abstract ID: 357
subject: Computational Neuroscience: Neuroinformatics
Presentation Type: Poster

Unlocking Alzheimer's Therapy: Computational Discovery of Novel Cyclophilin-D Inhibitors

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Background and Aim : Alzheimer's disease is the leading cause of dementia worldwide and remains without a curative therapy. Mitochondrial dysfunction is now recognized as a central pathological feature, with Cyclophilin-D (CypD) acting as a pivotal regulator of the mitochondrial permeability transition pore (mPTP). Aberrant opening of the mPTP drives neuronal loss and cognitive decline. Pharmacological inhibition of CypD has therefore emerged as a promising therapeutic strategy. A recent study identified the antioxidant compound ebselen as a novel CypD inhibitor capable of rescuing mitochondrial and synaptic function in Alzheimer's models. However, the therapeutic potential of structurally related analogs remains unexplored. This study employed molecular docking to evaluate ebselen and its analogs, aiming to identify improved inhibitors with higher binding affinity to CypD.

Methods : The three-dimensional structure of human Cyclophilin-D (PDB ID: 8EJX) was retrieved from the RCSB Protein Data Bank. The structures of Ebselen selenol (PubChem CID: [6336036]) and its 897 analogs and Felbamate (PubChem CID: [3331]) and its 130 analogs were obtained from the PubChem database in SDF format. Molecular docking was performed using Molegro Virtual Docker v.6 after preparation of the protein and ligands, including removal of crystallographic water molecules and energy minimization. The best binding conformation for each ligand was selected based on the lowest binding energy. Interactions such as hydrogen bonds and steric contacts were analyzed with Molegro Molecular Viewer, and pharmacokinetic properties were assessed using the SwissADME server.

Results : Molecular docking of Ebselen selenol (PubChem CID: [6336036]) against Cyclophilin-D yielded a binding energy of –76.2335 kcal/mol. Among its 897 analogs, N,N′-bis(3-carbamoylphenyl)hexanediamide (PubChem CID: [1590856]) exhibited the strongest affinity (–156.202 kcal/mol) but was excluded due to violations in Veber and Egan drug-likeness filters. The subsequent analog, (6E,8E)-10-oxo-N,10-diphenyldeca-6,8-dien-2-

ynamide (PubChem CID: [15004640]), showed a binding energy of –139.394 kcal/mol with favorable ADME characteristics. Interaction mapping revealed two hydrogen bonds with Arg82 and Asn102, supported by multiple steric contacts with Ala106, Gly109, Phe134, and His126, stabilizing the ligand within the CypD pocket. SwissADME predicted high gastrointestinal absorption, potential blood–brain barrier permeability, moderate solubility, and compliance with major drug-likeness rules. For Felbamate (PubChem CID: [3331]), the binding score was –101.381 kcal/mol. Among its 130 analogs, [2,3-bis(ethenyl)-1H-inden-1-yl]methyl carbamate (PubChem CID: [145233494]) achieved –116.866 kcal/mol, forming multiple hydrogen bonds with residues including Ser110 and Gln111. ADME evaluation confirmed high solubility, acceptable lipophilicity (LogP 2.68), favorable GI absorption, and overall compliance with drug-likeness criteria. Collectively, these analogs demonstrated stronger binding affinities and more promising pharmacokinetic profiles compared to their parent compounds

Conclusion : Ebselen selenol analog (6E,8E)-10-oxo-N,10-diphenyldeca-6,8-dien-2-ynamide exhibited substantially improved binding affinity (–139.394 kcal/mol) and favorable in silico pharmacokinetic predictions compared with the parent compound. Likewise, the Felbamate analog [2,3-bis(ethenyl)-1H-inden-1-yl]methyl carbamate demonstrated stronger binding (–116.866 kcal/mol) than Felbamate itself, while retaining acceptable drug-likeness. Among all tested ligands, (6E,8E)-10-oxo-N,10-diphenyldeca-6,8-dien-2-ynamide emerged as the most promising in silico candidate for Cyclophilin-D inhibition. While these findings highlight potential molecular leads for Alzheimer’s therapy, they are preliminary and require rigorous in vitro and in vivo validation before any translational or therapeutic implications can be established.

Keywords : Cyclophilin-D; Alzheimer’s Disease; (6E,8E)-10-oxo-N,10-diphenyldeca-6,8-dien-2-ynamide; [2,3-bis(ethenyl)-1H-inden-1-yl]methyl carbamate; Inhibitor Analogs; Molecular Dockin

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subject: Computational Neuroscience: Neuroinformatics
Presentation Type: Poster

Computational Approach to Screen New IgA Protease Inhibitors for Combating Haemophilus influenzae Meningitis

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Background and Aim : Meningitis is an acute or chronic inflammation of the protective membranes covering the brain and the spinal cord, called the meninges. ‹the symptoms conclude fever, intense headache, vomiting, neck stiffness, and sometimes sensitivity to light and sound in occasions. In children, symptoms might be nonspecific like irritability or poor feeding. Meningitis is a medical emergency because the inflammation affects the brain and the spinal cord, it can be life-threatening or cause serious long-term complications if it stays untreated. Bacterial meningitis is severe and can be fatal if not treated correctly with antibiotics and antiviral drugs. Haemophilus influenzae is a type of Gram-negative bacterium that can cause serious infections, including a form of bacterial meningitis known as Haemophilus meningitis. This bacterium is enriched with multiple virulence factors. H. influenzae has an enzyme called IgA protease, which is a highly specific serine endopeptidase that cleaves the hinge region of human immunoglobulin A1 (IgA1) also by disrupting mucosal immunity and aiding invasion and persistence in the host respiratory tract. This virulence factor is one of the main factors in causing meningitides. This bacterium is a significant antibiotic resistance, with a growing prevalence of multidrug-resistant (MDR) strains globally. Resistance is higher in invasive infections like meningitis, where MDR rates can reach nearly 47%. In Iran, Haemophilus influenzae shows a high prevalence of antibiotic resistance raising the rates up to clindamycin (71.3%) and penicillin 82.6%. Due to these facts, finding new alternative drugs is crucial. In this study, we aimed to determine the most potent Haemophilus influenzae IgA Protease inhibitor through molecular docking analysis.

Methods : The 3D structure of the Haemophilus influenzae IgA Protease (PDB ID:3H09) was obtained from the RCSB PDB database. The structures of the IgA1 Protease inhibitory ligand, 3,4-dichloroisocoumarin (DCIC) with PubChemCID:1609, and 100 of its analogs were retrieved from the PubChem database in SDF format. The protein and inhibitory ligands were prepared for docking using Molegro Virtual Docker version 6.0. After docking, the best interaction with the lowest energy binding was analyzed through Molegro Molecular Viewer v.7 software. Finally, the pharmacokinetic properties of the ligands were evaluated using the SwissADME server.

Results : Through all inhibitory ligands for IgA Protease, the best ligand was methyl 2-[(Z)-1,2-dichloro-2-(2-methoxycarbonylphenyl)ethenyl]benzoate (PubchemCID: 7053624) with a molecular weight of 365.2 g/mol and binding energy of -79.4251 kcal/mol. This ligand created three steric interactions with the IgA Protease protein residues Asp 476(B), Thr 505(B), and Thr 478(B), and one hydrogen bond with Thr 478(B) residues, and no electrostatic interaction. The ADME results showed that this ligand has a water solubility score (logS) of -5.06, a hydrophilic score (LogP) of 3.25, a polarity score (TPSA) of 52.60, and lastly, hydrogen bond donors of 0 and hydrogen bond acceptors of 4. Also the ligand does pass the BBB (Brain and Blood Barrier) with a high GI absorption.

Conclusion : These results indicate that methyl 2-[(Z)-1,2-dichloro-2-(2-methoxycarbonylphenyl)ethenyl]benzoate is a promising candidate for inhibiting Haemophilus influenzae IgA Protease infection. Further in vitro and in vivo studies are required to confirm their potential as therapeutic agents.

Keywords : Haemophilus influenzae; IgA Protease; Meningitis; Molecular Docking; ADME.

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Presentation Type: Poster

The investigation of possible new candidates of inhibitors for the VEGF and PDGF signaling pathway in glioblastoma treatment using Molecular Docking analysis

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Background and Aim : Glioblastomas (GBs) are the most frequent and aggressive brain tumors, with a median survival of only 15 months despite treatments like surgery, radiotherapy, and chemotherapy. These tumors may originate spontaneously or evolve from astrocytomas, involving alterations in growth factor expression and signaling pathways. Targeted therapies, including receptor tyrosine kinase inhibitors such as Imatinib, have shown promise preclinically but limited clinical success. Overexpressed factors like PDGF and VEGF drive tumor growth and angiogenesis, yet current inhibitors yield only modest therapeutic effects. Therefore, it is essential to seek alternative compounds with more potent inhibitory effects. in this study, we aimed to identify the most potent inhibitory ligand for VEGF and PDGF as a potential drug candidate through molecular docking analysis.

Methods : The 3D structures of VEGF (PDB ID: 5FV2) and PDGF (PDB ID: 5GRN) involved in glioblastoma signaling were obtained from the RCSB PDB database. Their respective inhibitory ligands, SU1498 for VEGF (PubChem CID: 5941539) and AG1433 for PDGF (PubChem CID: 2050), along with 117 analogs, were retrieved from the PubChem database in SDF format. Protein and ligand preparations included removal of existing ligands and water molecules, energy minimization, and addition of charges and hydrogens. Molecular docking was performed using Virtual Docker Molegro v.6, followed by analysis of the lowest-energy

binding conformations via Molegro Molecular Viewer. Pharmacokinetic profiles of the ligands were then assessed using SwissADME.

Results : Among the various inhibitory ligands targeting VEGF and PDGF, the most effective compounds were identified as 2-cyano-3-(4-hydroxy-5,6,7,8-tetrahydronaphthalen-1-yl)-N-(2-phenylethyl)prop-2-enamide for VEGF and 5-(6,7-Dimethylquinoxalin-2-yl)-2-phenylmethoxyphenol for PDGF, possessing molecular weights of 346.42 g/mol and 356.42 g/mol, and binding energies of -138.447 kcal/mol and -121.112 kcal/mol, respectively. These ligands established 2 hydrogen bonds with Cys68(W), and Cys61(W) and engaged in 7 steric interactions involving Asn62(W), Lys48(V), Ser50(V), Cys60(W), Glu67(W),Cys61(W), and Cys68(W) residues for VEGF, as well as 1 hydrogen bonds with Lys627 and 5 steric contacts with Ile627, Thr674,Val607,Lys627, and Asp836 residues for PDGF. ADME analysis revealed water solubility scores (LogS) of -4.79 and -5.36, hydrophilicity (LogP) values of 2.80 and 3.57, and topological polar surface areas (TPSA) of 73.12 and 55.24. Moreover, the number of hydrogen bond donors/acceptors were 2/3 and 1/4, respectively. A critical aspect to consider is that these selected compounds demonstrate favorable properties for gastrointestinal absorption and the capability to penetrate the blood-brain barrier (BBB). Furthermore, they comply with Lipinski's rules.

Conclusion : Collectively, based on the results obtained from the analysis of 117 ligands 2-cyano-3-(4-hydroxy-5,6,7,8-tetrahydronaphthalen-1-yl)-N-(2-phenylethyl)prop-2-enamide (PubChem CID:53829099) and 5-(6,7-Dimethylquinoxalin-2-yl)-2-phenylmethoxyphenol (PubChem CID:85868078) demonstrated the strongest binding affinities and favorable drug-like properties, making them promising candidates for inhibiting VEGF and PDGF signaling in glioblastoma. Nonetheless, further in vitro and in vivo studies are warranted to validate these findings.

Keywords : Molecular Docking; glioblastoma; VEGF; PDGF; ADME

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Investigation of Potent Inhibitors to Combat Pneumococcal Meningitis by Targeting Its Neuraminidase A: A Molecular Docking Study

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Background and Aim : Streptococcus pneumoniae remains a leading causative pathogen for bacterial meningitis worldwide, being a life-threatening infectious disease usually associated with serious neurological complications. Despite the availability of pneumococcal conjugate vaccines, no current formulation provides widespread immunity against all circulating serotypes of pneumococcus, and the rapid appearance of multidrug-resistant strains further complicates current therapeutic options. These limitations underscore the urgent need for novel strategies targeting bacterial virulence rather than viability. Among the critical virulence determinants, neuraminidase A (NanA) has attracted significant attention. By cleaving terminal sialic acid residues on host glycoproteins, NanA facilitates bacterial adhesion, invasion, and translocation across the blood–brain barrier, thereby contributing directly to the pathogenesis of meningitis. Targeting NanA with selective inhibitors offers a promising antivirulence approach, potentially reducing central nervous system invasion and mitigating neurological complications without exerting the selective pressure associated with conventional antibiotics. The current study was aimed to determine the best novel inhibitory ligands targeting NanA using molecular docking analysis.

Methods : The 3D structure of NanA of Streptococcus pneumoniae (PDB ID:2YA4) was received from the RCSB PDB database. The structure of inhibitory ligand of the Oseltamivir (PubChemCID:65028) and its 100 analogs were retrieved in SDF format from the PubChem

database. After preparing protein and ligands, molecular docking was done by Molegro Virtual Docker v.6, and only the top 1 pose of each ligand was selected through Molegro Virtual Viewer v.7. The best ligand with the lowest energy binding was chosen. Lastly, the pharmacokinetic properties were obtained via the SwissADME database.

Results : Among the evaluated analogs, the most effective inhibitor was identified as ethyl (3R,4R,5S)-4-acetamido-3-pentan-3-yloxy-5-(propylamino)cyclohexene-1-carboxylate(PubChemCID:10713139),with a ΔGbind of -110.471kcal/mol and a molecular weight of 354.48 g/mol. Interaction analysis shows 4 hydrogen bond with, Arg 590(B), Trp 547(B), Val 543(B), Ala 603(B) and 4 steric interactions with Arg 590(B), Trp 547(B), Ala 603(B), and Val 543(B) residues. ADME analysis revealed acceptable water solubility (LogS: -2.83), lipophilicity (XLOGP:3.97), polarity (TPSA:76.66), 5 hydrogen acceptors, and 2 hydrogen donors. Also, it has a high gastroenteritis (GI) absorption and the ability to penetrate through blood-brain barrier (BBB).

Conclusion : Although, these findings suggest ethyl(3R,4R,5S)-4-acetamido-3-pentan-3-yloxy-5-(propylamino)cyclohexene-1-carboxylate with PubChemCID: 10713139 as the best and a promising NanA inhibitor for Streptococcus pneumoniae , warranting further in vivo and in vitro validation.

Keywords : Molecular Docking ; Oseltamivir; Streptococcus pneumoniae; NanA ; ADME

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Molecular Docking study of Pyrazol-3-one and its Analogues as Potent Inhibitors for LgtC of Neisseria meningitidis

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Background and Aim : Neisseria meningitidis is a gram-negative bacterium that causes meningococcal meningitis, an inflammation of the protective membranes around the brain and spinal cord, leading to rapid and severe neurological damage and potentially life-threatening complications. The galactosyltransferase LgtC in Neisseria meningitidis facilitates a crucial step in lipooligosaccharide biosynthesis by transferring an alpha-D-galactose from UDP-galactose to the terminal lactose on the bacterial surface. In this study we aim to identify potent LgtC inhibitors through molecular docking analysis.

Methods : The 3D structure of LgtC of Neisseria meningitidis (PDB ID:1GA8) was obtained from RCSB PDB database. The structures of Pyrazol-3-one (PubChemCID:11513733) and its 5 analogs as inhibitors were retrieved in SDF format from PubChem database. After preparing protein and ligands, molecular docking was performed using the Molegro Virtual Docker v.6. Only the top 1 pose of each ligand was selected in Molegro Virtual Viewer v.7, and the best ligand with the lowest energy binding was evaluated. Finally, the pharmacokinetic properties of ligand were estimated using the SwissADME database.

Results : The compound 1-Oxidopyrazol-1-ium-3-one (PubChemCID:141352779) emerged as the most promising inhibitor, with a molecular weight of 98.06g/mol and the most negative ΔGbind (-53.5374kcal/mol). This ligand formed two hydrogen bonds with cys 246 (A), leu 131 (A) residues, and four steric interactions with asn 153 (A), his 244 (A), cys 246 (A), leu 131

(A) residues. ADME profile indicated that this ligand had a water solubility score of -0.25 , a lipophilicity score of -0.32, and a polarity score of 58.18, but no BBB.

Conclusion : These findings position this compound not as an inhibitor candidate against LgtC of Neisseria meningitidis, and the reason of this is that because despite the good results of this compound, it has no BBB.

Keywords : LgtC, Neisseria meningitidis, Molecular Docking, Pyrazol-3-one.

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subject: Computational Neuroscience: Neuroinformatics
Presentation Type: Poster

Computational Insights into 5,6,7,4' Tetramethoxyflavone Inhibitory Potential for Targeting Listeriolysin O in Brain-Infecting Listeria monocytogenes

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Background and Aim : Listeria monocytogenes is an intercellular pathogenic bacterium that can infect the central nervous system (CNS) and cause severe CNS disorders like meningitis, meningoencephalitis, rhombencephalitis, and brain abscesses. These infections mainly affect risk-prone groups like neonates, the elderly, pregnant women, and immunocompromised individuals, with high rates of mortality ranging between 20% and 30%. CNS listeriosis is a serious public health problem owing to high case-fatality rates and the great difficulty of therapy. One of the main virulence factors of L. monocytogenes is the cholesterol-dependent, pore-forming toxin called Listeriolysin O (LLO). The toxin promotes the escape of bacteria out of the phagosome into the host cell cytosol by lysing the phagosomal membrane. An escape maneuver that impedes bacterial degradation inside lysosomes, thus allowing Listeria to endure, replicate, and spread throughout the brain tissue. Furthermore, LLO also promotes translocation across the blood-brain barrier by lysis of the membranes of the brain endothelial cells, which is the necessary precursor of CNS infection. The use of artificial intelligence in drug design accelerates the drug development, lowers the expenditure, and enhances the precision by the quick prediction of compounds and their interactions. The current study attempts to solve the problem through molecular docking assessment of the inhibitory potential of new baicalein-based candidates by targeting LLO.

Methods : The 3D structure of LLO of L. monocytogenes (PDB ID: 4CDB) was received from the RCSB PDB database. The structure of inhibitory ligand of the Baicalein (PubChemCID: 5281605) and its 50 analogs were retrieved in SDF format from the PubChem database. After preparing protein and ligands, molecular docking was done by Molegro Virtual Docker v.6, and only the top 1 pose of each ligand was selected through Molegro Virtual Viewer v.7. The best ligand with the lowest energy binding was chosen. Lastly, the pharmacokinetic properties were obtained via the SwissADME database.

Results : Among the evaluated analogs, the most effective inhibitor was identified as 5,6,7,4' Tetramethoxyflavone (PubChemCID: 96118), with a ?Gbind of -101.484 kcal/mol and a molecular weight of 342.34 g/mol. Interaction analysis shows 1 hydrogen bond with Ser250 and 9 steric interactions with Asn 112, Asp 114,Gln 116, Asp 207, Phe 221, Gly 222, Ser 250, Lys 252 and Asn 402 residues and 17 hydrophobic bonds with Asn 112, Asp 114, Gln 116, Val 138, Asp 205, Asp 207, Ile 218, Lys 220, Phe 221, Gly 222, Thr 223, Ser 250,Lys 252, Gln 301, Tyr 303, Ile 400 and Asn 402 and no electrostatic interactions. ADME analysis revealed acceptable water solubility (LogS: -4.04), lipophilicity (XLOGP3: 3.07), polarity (TPSA: 67.13), 6 hydrogen acceptors. Also, it has a high gastroenteritis (GI) absorption and the ability to penetrate through blood-brain barrier (BBB).

Conclusion : Although, these findings suggest 5,6,7,4' Tetramethoxyflavone with PubChemCID: 96118 as the best second inhibitor and a promising LLO inhibitor for L. monocytogenes, warranting further in vivo and in vitro validation.

Keywords : Molecular Docking, 5,6,7,4' Tetramethoxyflavone, Listeria monocytogenes, Baicalein, ADME.

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Targeting IL-17 Pathway in Depression: Computational Exploration of Fisetin's Therapeutic Potential

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Background and Aim : Major depressive disorder (MDD) is a complex psychiatric disorder with multifactorial origins, involving genetic, biological, and environmental factors. Fisetin (FT), a plant-derived flavonoid, has demonstrated neuroprotective and antidepressant-like properties. However, its underlying molecular mechanisms in the treatment of MDD remain unclear.

Methods : We employed network pharmacology and molecular docking approaches to investigate the potential mechanisms of FT in MDD. Drug-likeness was evaluated using TCMSP and SwissADME. Potential FT targets were predicted through SuperPred and SwissTargetPrediction, while MDD-related targets were collected from established databases. The intersection targets were analyzed through protein–protein interaction (PPI) networks using STRING. Functional enrichment (GO and KEGG) was performed with ShinyGO, and molecular docking was applied to validate the binding affinity between FT and key targets.

Results : Analysis identified overlapping targets between FT and MDD, with PPI networks highlighting several key proteins. Enrichment analysis indicated significant involvement of the IL-17 signaling pathway in MDD pathogenesis. Docking results demonstrated strong binding affinities of FT with core proteins, including glycogen synthase kinase 3 beta (GSK3β), monoamine oxidase A (MAO-A), acetylcholinesterase (AChE), matrix metalloproteinase 9 (MMP9), and myeloperoxidase (MPO).

Conclusion : This study suggests that FT exerts potential antidepressant effects by modulating IL-17–related signaling pathways. The findings provide novel insights into the molecular mechanisms of fisetin and highlight its promise as a candidate for MDD therapy

Keywords : Major depressive disorder; Fisetin; Traditional Chinese medicine; Network pharmacology; Molecular docking

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Presentation Type: Poster

Performance of Deep Learning Models for Predicting Conversion from MCI to Alzheimer's using Structural MRI: A Systematic Review and Meta-analysis

Submission Author: Parham Abbasifar

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Conclusion : Background and Aim: Alzheimer’s disease (AD) is the leading cause of dementia, and early prediction of conversion from mild cognitive impairment (MCI) to AD is essential for timely intervention. Structural MRI (sMRI) provides valuable neuroimaging biomarkers, and deep learning (DL) models show strong potential for automated prognosis. This systematic review and meta-analysis evaluated the diagnostic accuracy, methodological quality, and heterogeneity of DL-based sMRI models predicting MCI-to-AD conversion. Methods: Following PRISMA 2020 guidelines and a PROSPERO-registered protocol, five databases (PubMed, Embase, Web of Science, Scopus, IEEE Xplore) were searched up to May 2025. Studies applying DL to baseline sMRI for MCI-to-AD prediction within 36 months were included. Extracted data included study characteristics, model architecture, validation strategy, and diagnostic outcomes. Methodological quality was assessed using the METRICS tool. Pooled sensitivity and specificity were calculated using a bivariate random-effects model. Heterogeneity was quantified by I^2 and τ^2, and subgroup analyses examined differences by model type, training strategy, dimensionality, and input region. Results: Forty-eight studies met inclusion criteria. Most used T1-weighted MRI data from the ADNI dataset, employing architectures such as 2D/3D CNNs, transformers, and graph neural networks. The pooled sensitivity and specificity were 0.79 (95% CI: 0.74–0.83) and 0.81 (95% CI: 0.78–0.83), respectively, indicating robust diagnostic performance. No publication bias was observed

(Deeks' test, p = 0.40). Transformer-based models achieved higher sensitivity (0.87, 95% CI: 0.79–0.92) than CNN-based ones (0.75, 95% CI: 0.71–0.79) with comparable specificity. Transfer learning slightly improved performance over models trained from scratch. No significant differences were found between 2D and 3D architectures or between whole-brain and regional inputs. Heterogeneity was mainly driven by dataset variation and model architecture. Despite this, emerging non-CNN models demonstrated promising ability to capture distributed structural alterations associated with early AD. Conclusion: DL models based on sMRI demonstrate high accuracy in predicting MCI-to-AD conversion, supporting their use for early detection and patient stratification. These approaches may assist clinicians in identifying high-risk MCI patients and tailoring interventions accordingly. However, methodological heterogeneity, limited external validation, and model opacity hinder clinical translation. Future research should prioritize standardized preprocessing and harmonized benchmarking. Explainable AI methods are also crucial to enhance reproducibility and real-world applicability.

Keywords : mild cognitive impairment; Alzheimer's; MRI; Deep learning; Conversion

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Abstract ID: 1039
subject: Computational Neuroscience: Neuroinformatics
Presentation Type: Poster

Integrating AI and bioinformatics: the role of machine learning for the identification of endoplasmic reticulum stress-associated genes in Alzheimer's Disease

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Background and Aim : Alzheimer's disease (AD), one of the greatest neurodegenerative diseases that plays a vital role in the progression of dementia, has greatly impacted the healthcare system worldwide. Understanding the development and a thorough elucidation of the progression of such a disease helps to build new therapeutic ways. Endoplasmic Reticulum stress(ER) stress, which refers to generating misfolded or unfolded proteins in the ER, is indicated to contribute to the emergence and development of AD. Machine Learning(ML), as a new AI development tool, can play a role in analyzing complex data using computational techniques. Integrating ER stress-associated genes, potential biomarkers in AD patients, with machine learning algorithms, we aim to identify new transcriptomic data for the disease's early onset and progression and seek novel therapeutic targets.

Methods : A comprehensive search was conducted utilizing PubMed, Scopus, Embase, and Web of Science databases. The related keywords were implemented to retrieve studies that predominantly indicate the role of ML in ER stress-related genes and biomarkers in AD detection and development.

Results : Gene expression profiles from individuals with Alzheimer’s disease can be analyzed using a range of computational classification methods. Microarray datasets available in the Gene Expression Omnibus (GEO) database and AlzData provide gene expression profiles and potential biomarkers derived from AD patients. Among the tested models, support vector machines showed the highest predictive performance (AUC = 0.879), with strong precision and recall. Interpretation techniques help reveal key ER stress-related genes, including RNF5, DNAJC10, and NGLY1, that contributed significantly to disease classification. Two molecular subtypes of AD were distinguished, one of which exhibited elevated immune infiltration and deregulated ER stress pathways. A pathway-informed model further identified five candidate

biomarkers (DYNC1I1, DNAJC1, SCRIB, TFEB, MAPKAPK3) with strong diagnostic potential. These findings demonstrate the power of machine learning in uncovering gene-level mechanisms of AD.

Conclusion : This study identified the impact of ML models in the detection of ER stress-related genes and molecular markers in Alzheimer's disease, showing their diagnostic role and relevance to immune dysregulation. While the findings offer valuable insights, they are based on computational analyses and require more experimental study. For future exploration, technologies including clustered regularly interspaced short palindromic repeats/CRISPR-associated protein 9 (CRISPR/Cas9) and human-induced pluripotent stem cells (hiPSCs) may be considered to functionally investigate the potential of the identified genes, bringing hope for therapeutic insights into ER stress mechanisms in AD.

Keywords : Alzheimer's disease; ER stress; Machine learning; Biomarkers; Gene expression; computational analysis

Count: 326
Abstract ID: 666
subject: Computational Neuroscience: Neuroinformatics
Presentation Type: Oral

Face Representation in the Frontal Eye Field of Rhesus Monkeys

Submission Author: Kiarash Yousefabadi

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Background and Aim : The frontal eye field (FEF) is a brain region primarily responsible for generating and guiding saccadic eye movements. In addition to its motor role, FEF receives visual input from the dorsal visual pathway, which conveys information related to spatial attention. It also maintains bidirectional connections with the inferior temporal (IT) cortex, allowing information exchange between the two areas. These connections suggest that FEF may contribute not only to motor control but also to aspects of visual processing. By recording neural activity in FEF during the presentation of complex visual stimuli, object representation and recognition can be examined. If FEF exhibits significant responses to complex objects, this would challenge the traditional view of it as a purely motor area. This study focuses on face representation and recognition within the FEF to better understand how visual information is processed in later stages of the dorsal stream.

Methods : Two rhesus macaque monkeys were trained to fixate on a single point while visual stimuli were presented for 100 ms each in a rapid serial visual presentation (RSVP) format, with a 150 ms blank screen between stimuli. Neural activity was recorded simultaneously from the FEF and IT cortex. Data were preprocessed and sorted into trials, with 100 ms of pre-stimulus activity used as baseline. Several analytical methods were applied: Support Vector Machines (SVM) for classification, F-statistics for selectivity analysis, Time–Time analysis to track temporal dynamics, and the Face Selectivity Index (FSI) to assess single-neuron selectivity. Statistical significance was tested using non-parametric permutation tests, and a half-split approach was used to maximize data utilization.

Results : Peri-stimulus time histograms (PSTHs) revealed higher neural activity in response to face stimuli compared to non-face stimuli in the FEF of both monkeys. SVM classification achieved approximately 70% accuracy in distinguishing face from non-face stimuli, demonstrating statistically significant face representation. F-statistics further confirmed this selectivity; after baseline correction, F values exceeding 1 indicated significant activity differences. Time–Time analysis showed that the pattern of face representation remained stable over time, suggesting a feedforward processing mechanism within FEF. At the single-neuron level, several neurons exhibited FSI values greater than 0.3, indicating the presence of face-selective cells in this region.

Conclusion : The frontal eye field, traditionally considered a visuo-motor area involved in saccade generation, also demonstrates categorical visual representations. The presence of face-selective neurons suggests that FEF contributes to higher-level visual processing, potentially by constructing saliency maps for guiding saccades or by supporting the broader cortical network in object recognition and visual attention.

Keywords : Frontal Eye Field (FEF); Face Selectivity; Rhesus Monkey

Count: 327
Abstract ID: 741
subject: Computational Neuroscience: Modeling and Simulation
Presentation Type: Oral

Reduced Predictability Under Social Reward Contexts: A Computational Approach Using Deep Reinforcement Learning

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Background and Aim : Social reward processing is a fundamental aspect of adaptive behavior, and its disruption is implicated in neurodevelopmental conditions such as autism spectrum disorder (ASD). In standard reinforcement learning (RL) frameworks, agents typically learn to anticipate and maximize rewards based on stable and uniform reward structures. Yet, when it comes to social rewards, things get a lot more complicated—they're context-dependent, salient, often variable, and are much harder to predict than nonsocial rewards. This unpredictability may underlie the difficulties observed in social motivation and reward-based learning in ASD. The present study investigates how embedding social reward features into a reinforcement learning environment alters the predictability and convergence dynamics of the agent's learning process.

Methods : We designed a GridWorld-based RL task where an agent interacts with spatially distributed reward elements. To better reflect social reward complexity, we modified both the reward function and the environment: (1) we introduced heterogeneity into the reward landscape, designating certain elements as high-value (social) and others as low-value (nonsocial); and (2) we randomized the spatial positions of these rewards from episode to episode. The agent was trained using a deep Q-learning algorithm, employing a convolutional neural network to extract nuanced environmental features. Each experimental run comprised 1,000 episodes, with multiple runs employing distinct reward distributions. Principal outcome measures included total accumulated reward and average temporal-difference (TD) error, offering insights into both learning dynamics and the agent's capacity to anticipate future rewards.

Results : The introduction of social reward heterogeneity increased reward distribution variability and diminished overall environmental predictability. Consequently, the agent displayed greater fluctuations in TD error and generally slower convergence compared to baseline scenarios lacking social reward variability. Interestingly, in some runs (e.g., Run 3),

the agent achieved improved convergence and more stable TD error, indicating successful adaptation to the unpredictable reward landscape. Further analysis revealed that optimal learning and convergence were observed when social rewards were weighted at 1.5–2 times the value of nonsocial rewards and made up 20–30% of the total rewards. When about 30% of reward locations were randomized and the remaining 70% remained constant, predictability and stability both improved. This was carried out concurrently with a reduction in the severity of the penalty. This implies that by finding a balance between a regular reward distribution and a certain level of unpredictability, adaptive learning strategies can be created even in complex social contexts

Conclusion : Such unpredictability may point to computational challenges associated with processing social rewards in biological systems, potentially offering insight into the social reward learning difficulties seen in ASD. Interestingly, convergence within certain parameter spaces suggests there may be forms of adaptive compensation at play; the interplay among reward magnitude, spatial configuration, and environmental variability seems crucial in shaping social learning outcomes. (This work is based upon research funded by Iran National Science Foundation (INSF) under project No 4042222.)

Keywords : Social Reward Processing; Reinforcement Learning; Predictive Coding; Computational Modeling; Environmental Heterogeneity

Count: 328
Abstract ID: 281
subject: Computational Neuroscience: Modeling and Simulation
Presentation Type: Poster

Dynamic Information Flow during Naturalistic Music Listening

Submission Author: Amirhossein Yekta

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Background and Aim : Music listening is a naturalistic and multimodal cognitive activity that engages large-scale brain networks underlying perception, memory, and affective processing. Recent advances in cognitive neuroscience highlight that the brain functions as a complex dynamical system, where distributed neural assemblies exchange information in a non-linear and temporally structured manner. Electroencephalography (EEG), with its high temporal resolution, provides a unique window into the fast oscillatory and network-level dynamics evoked by music. Previous studies have established that musical familiarity and enjoyment modulate oscillatory entrainment and synchronization across cortical regions. Yet, a deeper understanding of how information flows between brain areas during music listening—capturing both the directionality and nonlinearity of neural interactions—remains limited. Addressing this gap can advance our knowledge of music cognition and the neurodynamics of affective states.

Methods : We analyzed the publicly available Music Listening–Genre (MUSIN-G) EEG dataset, consisting of high-density EEG recordings (128 channels) from 20 participants exposed to 12 songs spanning diverse musical genres. Preprocessing followed the recommended pipeline, including high-pass filtering, artifact rejection with MARA, bad-channel interpolation, and average re-referencing. After preprocessing, the EEG signals were segmented per song and subjected to information- theoretic analysis. Specifically, we employed Transfer Entropy (TE) to quantify directed, non- linear information flow between cortical regions. Unlike measures of structural connectivity, which rely on anatomical pathways, or functional connectivity, which captures statistical dependencies, TE provides an estimate of effective connectivity, identifying causal and time- asymmetric influences in brain dynamics. Connectivity matrices were computed across canonical EEG frequency bands, and statistical comparisons were performed as a function of subjective familiarity and enjoyment ratings.

Results : Our analyses revealed a shift in the pattern of the flow of information during music listening relative to baseline silence, particularly within fronto-temporal and temporo-parietal networks. Songs rated as more familiar and enjoyable elicited more feedback compared with unfamiliar Songs. Furthermore, unfamiliar or less enjoyable songs showed weaker and more fragmented network dynamics, with localized information flow restricted to sensory areas. Frequency-specific analyses indicated that theta and alpha band connectivity were most sensitive to subjective experience, consistent with their established roles in emotional regulation and memory encoding. These results extend previous findings on neural entrainment by revealing that subjective musical engagement is reflected not only in oscillatory power but also in the directed flow of information.

Conclusion : Our study demonstrates that Transfer Entropy, as an effective connectivity measure grounded in complex systems and information theory, provides a powerful framework for understanding the neurodynamics of naturalistic music listening. The results highlight that enjoyment and familiarity with music are associated with enhanced cross-regional information integration, reflecting the dynamic reconfiguration of large-scale brain networks. Beyond music cognition, this approach exemplifies how information-theoretic metrics can uncover the causal structure of neural communication, offering potential applications in affective neuroscience, brain- computer interfaces, and music-based therapeutic interventions.

Keywords : Electroencephalography (EEG);Music Cognition;Transfer Entropy;Effective Connectivity;Information Flow

Count: 329
Abstract ID: 175
subject: Computational Neuroscience: Modeling and Simulation
Presentation Type: Oral

Game-Based Neurocognitive Screening and Computational Modeling of Visual Attention in Learning Disabilities

Submission Author: Maryam Mollaghasem

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Background and Aim : Learning Disabilities (LD) are neurodevelopmental conditions that impair core cognitive functions, particularly working memory and visual attention. Children with LD often display atypical patterns of visual information processing and integration, resulting in inefficient attentional allocation compared to typically developing peers. Early and accurate identification is essential for timely intervention; however, conventional assessments are often slow, subjective, and inadequate for capturing the fine-grained dynamics of visual attention. Game-based cognitive assessments, especially those involving visuospatial pattern recognition, offer an engaging and ecologically valid medium for examining both memory and attention. Building on this potential, the present study employed the digital game Honey Memory, integrating a visuospatial memory task with high-resolution eye-tracking. The objectives were: (1) to evaluate its utility as a digital screening tool for LD, and (2) to perform computational modeling of individual visual attention patterns, revealing underlying attentional mechanisms.

Methods : Sixty-seven children, aged between 6 and 10 years, participated: 34 diagnosed with LD and 33 typically developing controls, matched for age and vision status. All had normal or corrected-to-normal vision. In each Honey Memory trial, a honeycomb-shaped grid appeared with all cells initially white. After a brief fixation, a subset of cells was highlighted in yellow for 2–4 seconds. Participants memorized these locations; once the grid reverted to white, they

indicated the remembered cells via mouse clicks. No time limits were imposed. An eye tracker (GP3 Gazepoint, operating at 60 Hz, at an operational distance of 50–80 cm) continuously recorded fixation locations, durations, and saccadic movements. Analysis followed three steps: 1. Group-level statistical comparisons between LD and controls. 2. Feature extraction from gameplay and eye-tracking datasets. 3. Classification and computational modeling of visual attention. For screening, tree-based classifiers (decision trees, random forests, XGBoost) differentiated LD from controls. For attention modeling, a simulation framework incorporated: • Top-down attention weighting (task-driven saliency modulation) • Focal distance computation for spatial prioritization • Working-memory capacity constraints • Particle-movement simulation (forgetting process simulation) Simulated gaze patterns were validated against empirical data.

Results : Preliminary results showed significant between-group differences in visual attention patterns, fixation duration and dispersion, and memory retrieval accuracy. Children with LD exhibited lower spatial accuracy, shorter fixations, and less efficient attentional strategies during encoding, often focusing disproportionately on irrelevant areas. Computational modeling reproduced these patterns by adjusting top-down control, working-memory constraints, and employing particle movement to simulate forgetting. The XGBoost classifier achieved the best performance, with 84% test accuracy and 86% precision and recall. Accuracy declined exponentially with longer reaction times, and forgetting rates differed significantly between groups.

Conclusion : This study presents an integrated screening-plus-modeling approach for evaluating visuospatial memory and visual attention in LD. Combining behavioral performance, eye-tracking, and mechanistic modeling—via top-down weighting, focal-distance computation, memory limits, and particle-based gaze simulation—offers a fine-grained, non-invasive, and scalable assessment method. Gaze dynamics demonstrate diagnostic value and can be computationally reproduced, bridging behavioral assessment and mechanistic insight. Honey Memory thus has strong potential as both a clinical screening tool and a platform for modeling attentional processes in neurodevelopmental disorders.

Keywords : Learning Disabilities (LD);Visuospatial Memory;Cognitive games;Eye-tracking;XGBoost Classifier;Particle Modeling

Count: 330
Abstract ID: 462
subject: Computational Neuroscience: Computational Tools
Presentation Type: Oral

Gut Microbiota-Derived Metabolites as Drivers of Hepatic Encephalopathy: A Systems Biology Approach

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Background and Aim : Background: Hepatic encephalopathy (HE) is a severe neuropsychiatric complication of liver failure traditionally attributed to hyperammonemia. Recent evidence highlights the role of gut microbiota-derived metabolites in modulating systemic inflammation and neurotoxicity. However, the molecular mechanisms linking these metabolites to HE remain insufficiently understood.

Methods : Methods: Transcriptomic data from peripheral CD4+ T lymphocytes and fusiform gyrus brain tissue of HE patients were analyzed to identify differentially expressed genes (DEGs). Genes associated with gut-derived metabolites (choline, lipids, short-chain fatty acids, tryptophan catabolites, and secondary bile acids) were retrieved from GeneCards. Overlapping genes were subjected to pathway enrichment, phenotype, cell type, and cellular component analyses. miRNA predictions and drug repurposing were performed using Enrichr and ToppGene.

Results : Results: Nine hub genes were identified in gut-systemic interactions and twenty-nine hub genes in gut-brain interactions. Key impaired pathways included IL-24 and TP53 signaling, oxidative stress, mitochondrial fatty acid β-oxidation, neuroinflammation, and glutamatergic dysfunction. Eighteen miRNAs and thirteen candidate drugs were predicted to target both metabolite-related and HE-associated genes, providing potential diagnostic and therapeutic opportunities.

Conclusion : Conclusion: This integrative systems biology approach establishes the first molecular framework linking gut-derived metabolites to HE pathophysiology beyond the ammonia hypothesis. The findings highlight novel biomarkers and therapeutic candidates, underscoring the importance of the gut-liver-brain axis as a target for precision medicine in HE.

Keywords : Hepatic encephalopathy; Dysbiosis; Gut-brain interactions; neuroinflammation; Microglia.

Count: 331
Abstract ID: 257
subject: Neurorehabilitation and Regeneration: Psycho-cognitive Rehabilitation
Presentation Type: Oral

hemispherectomy ,an excellent models for studying neuroplasticity.

Submission Author: MohammadSadegh Masoudi

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Background and Aim : Neuroplasticity is the unique ability of the brain to recover lost functions. Studying neuroplasticity can be the key to solving many questions regarding the treatment of refractory neurological diseases.

Methods : . In this study, we evaluated 30 patients who underwent hemispherectomy and analyzed their clinical outcomes, functional recovery, and neuroplastic changes.

Results : Our findings highlight the extent and variability of neuroplastic adaptations in both pediatric and adult populations, elucidating factors that influence functional reorganization post-surgery. These insights provide valuable guidance for optimizing rehabilitation strategies and improving long-term prognosis for patients with catastrophic hemispheric dysfunction

Conclusion : Patients who have undergone hemispherectomy can serve as excellent models for studying neuroplasticity.

Keywords : Hemispherectomy_neuroplasticity-neurorehabilitation

Count: 332
Abstract ID: 638
subject: Neurorehabilitation and Regeneration: Psycho-cognitive Rehabilitation
Presentation Type: Oral

An AI-driven, pulse-regulated abdominal breathing application using Rehacore software for drug-resistant PTSD patients

Submission Author: Mohammadjavad Hoseinpourfard

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Background and Aim : The human brain, the body's most complex organ, is responsible for processing a vast spectrum of emotions. The recollection of traumatic experiences specifically activates neural circuits that mediate intense emotional states such as fear, anger, and sadness. These same brain regions integrate sensory and motor signals, modulate physiological arousal, and are critical to an individual's stress response. For conditions like Post-Traumatic Stress Disorder (PTSD), pharmacological interventions are a first-line treatment, but their efficacy often diminishes over time due to drug tolerance, side effects, and variable individual response. This declining effectiveness underscores the critical need to explore and develop robust non-pharmacological treatment strategies, such as targeted neurofeedback, to directly address the dysregulated neural circuitry of trauma.

Methods : This study designed to investigate the prospective effectiveness of a non-pharmacological therapeutic intervention for drug-resistant PTSD. The core of the intervention is a structured training protocol aimed at enhancing resilience and emotional regulation. The intervention is grounded in a model of neural plasticity that targets maladaptive fear networks. The core mechanism involves the gradual and controlled exposure to aversive stimuli, combined with real-time self-regulation training. This process is intended to enhance neurocognitive flexibility, ultimately enabling participants to gain top-down control over conditioned fear and emotional responses, leading to a decrease in symptom burden.

Results : A review of the current literature reveals strong evidence for the efficacy of non-invasive brain interventions and conscious self-regulation methods across multiple clinical domains. The findings are summarized below by therapeutic area

Conclusion : Conclusion: The most significant finding of this review is the demonstrated superior efficacy of a combined therapeutic approach for treating complex neuropsychiatric disorders. This integrative model synergistically merges conscious self-regulation techniques with advanced, non-invasive brain modulation technologies. The protocol specifically leverages photobiomodulation therapy, augmented by Neurobiological Reprogramming

Artificial Intelligence (AI) and Neural Biofeedback Therapy (NBFT). We conclude that this multi-faceted intervention represents a promising, non-pharmacological frontier in clinical neuroscience. By simultaneously engaging top-down (conscious self-regulation) and bottom-up (photobiomodulation, AI-driven reprogramming, NBFT) pathways, it offers a powerful framework for restoring neural homeostasis and achieving sustained clinical outcomes in treatment-resistant conditions such as PTSD.

Keywords : Post-Traumatic Stress Disorder (PTSD), Self-Regulation, Resonance Breathing, RehaCom, Neurofeedback, Brain-Computer Interface (BCI), Heart Rate Variability (HRV).

Count: 333
Abstract ID: 959
subject: Neurorehabilitation and Regeneration: Psycho-cognitive Rehabilitation
Presentation Type: Oral

Harnessing Personalized Immunology: Intranasal Autologous Conditioned Serum Ameliorates Stroke-Induced Behavioral and EEG Abnormalities

Submission Author: SAREH KAZMI

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Background and Aim : Ischemic stroke demands personalized therapeutic strategies due to the high variability in patient recovery and limited treatment options. We investigated a novel, individualized immunomodulatory approach using autologous conditioned serum (ACS)—a patient-specific biologic—in a mouse model of photothrombotic stroke.

Methods : ACS was prepared on an individual basis from each animal's own blood, creating a personalized profile of anti-inflammatory cytokines and growth factors. Following stroke induction with Rose Bengal and a 532 nm laser, mice received daily intranasal deliveries of either ACS or normal saline (NS) for seven days. We subsequently assessed anxiety and stress-related behaviors and recorded electroencephalographic (EEG) activity in the medial prefrontal cortex (mPFC) and globally.

Results : Our results indicates,this personalized treatment regimen significantly mitigated stroke-induced anxiety-like behaviors and restored normal electroencephalographic (EEG) rhythms, which were profoundly disrupted in the untreated stroke group.

Conclusion : In conclusion, leveraging the body's own signaling molecules through ACS provides a tailored and effective intervention for post-stroke neurological deficits. This work establishes proof-of-concept for ACS as a promising personalized medicine platform to improve functional recovery after ischemic stroke.

Keywords : Ischemic stroke;Immunotherapy;ACS;EEG

Count: 334
Abstract ID: 804
subject: Neurorehabilitation and Regeneration: Psycho-cognitive Rehabilitation
Presentation Type: Poster

The Impact of Digital Exposure on Structural Brain Maturation During the Critical Period of Development (The "Digital Baby" Phenomenon)

Submission Author: Zahra Mahdavian

Zahra Mahdavian[1]

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Conclusion : Early and increasing exposure of young children to digital devices (The "Digital Baby" Phenomenon) has raised major concerns regarding neuroplasticity during critical developmental periods. This systematic review synthesizes 5 original articles from the last 5 years that utilized advanced neuroimaging techniques, specifically Diffusion Tensor Imaging (DTI) and Structural Magnetic Resonance Imaging (MRI). The objective is to identify persistent alterations in White Matter (WM) structures, Grey Matter (GM), and brain network organization associated with high Screen Time (ST) in infants and preschoolers. Findings provide direct evidence of micro- and macrostructural alterations in the brain that carry serious implications for cognitive, linguistic, and socio-emotional development. The 0–5 age period constitutes a critical window for myelination and synaptic pruning. The aim of this systematic synthesis is to aggregate structural evidence to address this key question: Does digital media exposure interfere with the optimal pattern of structural brain maturation during this sensitive period?

Keywords : Neuroplasticits,Digital Baby,Brain Network Development,Connectomics Analysis,Grey Matter Volume,Early Childhood,Cognitive Neuroscience,Neurobehavioral Correlates,Impact of Modern Technologies.

Count: 335
Abstract ID: 199
subject: Neurorehabilitation and Regeneration: Psycho-cognitive Rehabilitation
Presentation Type: Poster

The effectiveness of Repetitive Transcranial Magnetic Stimulation on executive function and cognitive errors in patients with multiple sclerosis

Submission Author: Zahra Omidi

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Background and Aim : his study aimed to investigate the effectiveness of repetitive transcortical magnetic stimulation (rTMS) on executive function and cognitive errors in patients with multiple sclerosis.

Methods : The present research, in terms of purpose, application, and method, was of a semi-experimental design with a pretest–posttest approach and a control group. The statistical population of this study consisted of men and women with multiple sclerosis in Tehran in 1404. The statistical sample was 30 men and women with multiple sclerosis. The study sample was selected purposively and randomly (drawing lots) into two groups: repetitive transcortical magnetic stimulation (RTMS) (15 people) and control group (15 people). Repetitive transcortical magnetic stimulation (RTMS) was performed in 12 sessions for the experimental groups, and the control group did not receive any training. The research instruments were the Coolidge Executive Function Questionnaire (2002) and Possel Cognitive Errors Questionnaire (2009).

Results : Data were analyzed using analysis of covariance in SPSS version 27. The results showed that repetitive transcortical magnetic stimulation was effective in improving executive functions and reducing cognitive errors in multiple sclerosis patients.

Conclusion : Based on the results, repetitive transcortical magnetic stimulation protocols can be used to intervene in executive functions and cognitive errors in people with multiple sclerosis in mental health centers and psychiatric hospitals.

Keywords : effectiveness,Repetitive Transcranial Magnetic Stimulation, executive function ,cognitive errors,multiple sclerosis

Count: 336
Abstract ID: 192
subject: Neurorehabilitation and Regeneration: Physiotherapy
Presentation Type: Poster

Spastic Hamstring dry needling in multiple sclerosis: A randomized controlled trial to study electromyographic, biomechanical, and clinical effects

Submission Author: Homa Seyedmirzaei

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Background and Aim : Multiple sclerosis (MS), the most common demyelinating disease, affects about three million people worldwide, with spasticity present in up to 80% of patients. This symptom, often accompanied by pain, fatigue, and sleep disturbances, significantly reduces quality of life and functional independence. Current management includes medications, neuromuscular blocks, and physiotherapy. However, their efficacy remains suboptimal, and side effects are common. Dry needling (DN), a relatively new technique, has shown promise in stroke-related spasticity, but evidence in MS remains limited. In this study, we aimed to assess the electromyographic, biomechanical, and clinical effects of dry needling in people with MS who suffer from hamstring muscle spasticity.

Methods : We conducted a single-blind, randomized controlled trial at the Iran MS Association physiotherapy clinic. Forty-eight adults with relapsing-remitting MS (RRMS) and hamstring spasticity were randomized by sealed envelopes to dry needling (DN; n=24) or control (n=24). Inclusion criteria were an Expanded Disability Status Scale (EDSS) score of 5–6.5, a Modified Modified Ashworth Scale (MMAS) score ≥1 in hamstrings, and the ability to walk with or without an aid. Exclusion criteria included other neurological disorders, knee flexor contracture, recent relapses, botulinum toxin injection, or contraindications for DN. An assessor blinded to allocation measured outcomes at baseline (T0), immediately after the

intervention (T1), and one week after the intervention (T2). DN (prone position; sterile 0.3×50 mm needle) was applied for one minute to the long head of the biceps femoris and the semitendinosus. Outcomes included spasticity (MMAS), mobility (Timed 25-Foot Walk [T25FW]), stiffness (biomechanical torque-angle method), and surface electromyography (sEMG) at rest and during spasticity assessment. Nonparametric tests were used, with statistical significance set at $\alpha=0.05$.

Results : All 48 participants (36 females, 12 males; mean age 40.1 years in DN, 44.3 years in controls) completed the study, and baseline characteristics were comparable between groups. Friedman test results showed significant changes across time points (T0, T1, T2) in the DN group for MMAS, T25FW, stiffness, and sEMG at rest and during spasticity assessment (all $p<0.01$). In contrast, the control group showed significant changes only in stiffness and lateral hamstring sEMG at rest ($p<0.01$). Between-group comparisons (Kruskal–Wallis tests) revealed greater reductions in spasticity and more favorable sEMG outcomes in the DN group immediately after treatment (T1, $p<0.05$). Notably, differences between groups remained significant at follow-up (T2) for hamstring sEMG at rest (medial $p=0.02$; lateral $p=0.01$).

Conclusion : Our study demonstrated the beneficial short-term effects of DN on spasticity, stiffness, mobility, and hamstring electromyographic activity in people with MS. These findings support DN as a promising adjunctive physiotherapy intervention, although standardized treatment protocols remain to be investigated. Further research with larger samples, repeated sessions, and extended follow-up is required to optimize therapeutic parameters and elucidate the underlying neurophysiological mechanisms.

Keywords : Multiple sclerosis; dry needling; spasticity; electromyography; stiffness.

Count: 337
Abstract ID: 603
subject: Neurorehabilitation and Regeneration: Physiotherapy
Presentation Type: Oral

Investigating the effect of Transcutaneous electrical nerve stimulation on lumbar fascia tissue and lumbar curvature in healthy people .

Submission Author: Afsaneh Dadarkhah

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Background and Aim : The thoracolumbar fascia is of great importance due to its decisive role in maintaining body alignment and balance. Therefore, changes in the thickness of the lumbar fascial can lead to symptoms such as reduced flexibility, increased pressure on the spine due to improper distribution of the applied forces, and as a result, injury or functional limitation in the lumbar region. As a result, making a change in the thickness of the lumbar fascia can be the key to solving many functional problems or better strengthening the muscle tissues.

Methods : The present study investigated the thickness of the lumbar fascia and the flexibility of the lumbar region in healthy individuals using ultrasound, spinal mouse, flexible ruler, and sit and reach test. 100 healthy individuals in two groups of 50 people were subjected to active TENS and placebo TENS. Fascia thickness, lumbar arch, lumbar flexion angle and lumbar flexibility were evaluated in three stages, before the intervention, after the intervention and two weeks after the intervention (fallow-up) were done .

Results : The results of the study showed that in the TENS group, there was a reduction in the thickness of the lumbar fascia ($P \leq 0.005$), an increase in the maximum lumbar flexion angle ($P \leq 0.005$), an increase in the lumbar curvature angle ($P \leq 0.005$) and also an increase in the Sit & test results ($P \leq 0.005$) was significantly compared to the control group. Also, the results of the follow-up evaluations showed the stability of the changes made following the TENS .

Conclusion : The results of the present study showed that TENS in healthy people can increase the flexibility of the lumbar region by reducing the thickness of the lumbar fascia and increasing the angle of the lumbar curvature and lumbar flexion and thus prevent possible injuries to the lumbar vertebrae. The findings stated that TENS can be considered as a factor in the prevention of back pain by improving the parameters of the lumbar area .

Keywords : Thoracolumbar fascia, Electrical stimulation, TENS, Flexible ruler, Spinal mouse, Ultrasonography.

Count: 338
Abstract ID: 24
subject: Neurorehabilitation and Regeneration: Occupational Therapy
Presentation Type: Poster

Investigating the Effects of Transcranial Direct Current Stimulation (tDCS) of the Thalamic Region on Cognitive Performance in Individuals with Neurodevelopmental Disorders

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1. PhD in Cognitive Neuroscience

Background and Aim : The thalamus is a central brain structure crucial for sensory information processing and cognitive functions. This study aimed to investigate the effects of transcranial direct current stimulation (tDCS) on cognitive performance in individuals with neurodevelopmental disorders, focusing on thalamic region stimulation.

Methods : In this quasi-experimental design, 30 participants diagnosed with Autism Spectrum Disorder (ASD) were divided into experimental and control groups. The stimulation protocol involved 1.5 mA direct current applied for 25 minutes across 15 sessions. Electrode placement adhered to the 10–20 system, using Fz as the anodal and Oz as the cathodal site.

Results : Analysis of variance (ANOVA) showed significant improvements in sustained attention, working memory, and visual processing in the experimental group compared to the control group. A reduction in behavioral symptoms associated with neurodevelopmental disorders was also observed.

Conclusion : tDCS targeting the thalamus may serve as an effective approach to enhance cognitive performance and reduce clinical symptoms in individuals with neurodevelopmental disorders such as autism. The findings highlight the thalamus as a promising target for neurostimulatory interventions in cognitive neuroscience.

Keywords : Transcranial Direct Current Stimulation (tDCS), Thalamus, Cognitive Performance, Neurodevelopmental Disorders, Autism, Working Memory, Sustained Attention, Visual Processing, Neural Plasticity, Non-inv

Count: 339
Abstract ID: 501
subject: Neurorehabilitation and Regeneration: Occupational Therapy
Presentation Type: Oral

The Effect of a Structured and Progressive Rhythmic-Cognitive Dual-Task Exercise Program on Impulsivity in Children with ADHD

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Background and Aim : Background: Impulsivity is a core diagnostic feature of Attention deficit and hyperactive disorder (ADHD) in children. Deficits in inhibitory control can lead to behavioral issues and difficulties in interpersonal communication. Due to the limited use of medication at early ages and the broad impact of impulsivity on developmental domains, there is an increasing need for effective interventional approaches.

Methods : Objectives: This study aimed to evaluate the effectiveness of a structured dual-task exercise program that incorporates rhythmic movements preferred by children, along with visual and auditory cues, in reducing impulsivity and its related symptoms. This randomized clinical trial was conducted on 60 children aged 6 to 12 years diagnosed with ADHD. Participants were allocated equally into intervention (dual-task) and control groups using Covariate Adaptive Randomization. Both groups received 18 intervention sessions of 45 minutes during 6 weeks. Evaluations were performed at baseline, post-intervention, and one-month post-intervention to assess the sustainability of outcomes using the Integrated Visual and Auditory Continuous Performance Test (IVA-2) and Cambridge Neuropsychological Test Automated Battery (CANTAB) assessments.

Results : Significant improvements were found between the two groups in all response control and prudence measures, including auditory and visual subdomains ($P < 0.001$). Reaction time indicators also demonstrated significant between-group differences ($P < 0.001$). At the onemonth follow-up, response control and auditory response control measures showed no significant difference; however, other outcomes maintained significant improvements.

Conclusion : Conclusion: The findings suggest that a rhythmic-cognitive dual-task training program can positively impact impulsivity symptoms and enhance inhibitory control in children with ADHD.

Keywords : ADHD, Impulsivity, Inhibition, Dual-task, Rhythmic movement, Attention

Count: 340
Abstract ID: 585
subject: Neurorehabilitation and Regeneration: Speech and Language Therapy
Presentation Type: Poster

A Neuroplasticity-Based Voice Therapy Approach for Adductor Spasmodic Dysphonia

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Background and Aim : Adductor spasmodic dysphonia (ADSD) is a focal laryngeal dystonia characterized by involuntary hyperadduction of the vocal folds, leading to a strained and effortful voice. The standard treatment usually involves botulinum toxin injections; however, some patients show insufficient therapeutic response. In such cases, rehabilitation approaches based on speech and voice therapy may serve as valuable alternatives.

Methods : We report the case of a 40-year-old female with a history of progressive voice breaks, strain, and vocal fatigue. Videostroboscopic examination revealed hyperadduction of the true vocal folds during phonation with no structural lesions. Neurological assessment ruled out other movement disorders. The patient underwent an integrated voice therapy program combining traditional behavioral techniques with neuroscience-informed strategies targeting sensorimotor training.

Results : After 5 therapy sessions across 5 weeks, the patient demonstrated reduced frequency of voice breaks, improved perceptual voice quality (GRBAS scale), and decreased handicap scores (VHI). Importantly, she reported greater communicative confidence and reduced effort during speech.

Conclusion : This case illustrates the potential benefit of combining voice therapy with rehabilitative neuroscience principles in the management of ADSD. A neuroplasticity-based approach may offer a valuable complement to established treatments, supporting long-term functional improvements and enhanced patient quality of life.

Keywords : spasmodic dysphonia; neuroplasticity; voice quality

Count: 341
Abstract ID: 572
subject: Neurorehabilitation and Regeneration: Speech and Language Therapy
Presentation Type: Poster

Restoring Speech After Stroke: A Neuro-engineering Approach

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Conclusion : Introduction: Stroke often results in aphasia and motor speech disorders, creating a profound communication barrier that traditional therapy cannot always fully resolve. Neuro-engineering, which merges neuroscience with engineering principles, is emerging as a transformative field to address this challenge. This systematic review evaluates the current state of neuro-engineering technologies designed to decode and restore speech in post-stroke patients, highlighting a paradigm shift from rehabilitation to functional restoration of communication. Materials and Methods A systematic search was conducted across major scientific databases for studies published between 2015 and 2024. The review included clinical trials, case studies, and proof-of-concept reports involving stroke survivors with chronic speech impairments. Interventions were categorized into two main approaches: 1) Speech Brain-Computer Interfaces (BCIs) using intracortical or electrocorticographic (ECoG) arrays, and 2) Non-invasive neuromodulation techniques like transcranial direct current stimulation (tDCS). Outcomes measured included speech decoding accuracy, improvement on standardized language scales, and neural signal fidelity. Results The evidence supports the efficacy of both strategies. For severely paralyzed individuals, invasive Speech BCIs have successfully decoded attempted speech into text or synthesized audio by interpreting neural activity from the speech motor cortex. These systems can achieve significant decoding accuracies for dozens of words and phonemes. In parallel, non-invasive neuromodulation techniques, particularly t-DCS applied to language areas, demonstrate a significant adjunctive benefit. When paired with conventional speech therapy, they enhance neuroplasticity and lead to greater improvements in verbal fluency and naming accuracy compared to therapy alone. Conclusion Neuro-engineering offers powerful new tools to circumvent damaged speech pathways. While speech-BCIs are currently in experimental stages and neuromodulation requires further protocol optimization, together they represent the future of post-stroke communication restoration. The ultimate goal is the development of fully implanted, wireless, and real-time communication systems that can give a voice back to those who have lost it, fundamentally improving autonomy and quality of life

Keywords : Stroke, Neuromodulation, Brain-Computer Interfaces, Neuro-engineering

Count: 342
Abstract ID: 403
subject: Neurorehabilitation and Regeneration: Medication and Stem Cell Therapy
Presentation Type: Oral

Role of Aquaporin4 in parkinson

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Background and Aim : Aquaporin-4 (AQP4), a modulator of motor symptoms and synaptic plasticity, may contribute to freezing of gait (FOG)—a significant gait disturbance in Parkinson's disease with an unclear pathophysiology—and its associated impairments in balance learning. However, this potential relationship has not been investigated until now. This preliminary study explores the potential role of AQP4 in FOG and its associated balance learning deficits.

Methods : The study involved fifteen patients with FOG, fifteen patients without FOG, and fifteen healthy controls. Serum AQP4 levels were measured using enzyme-linked immunosorbent assay, and balance learning was assessed using a voluntary dynamic balance task performed on a stabilometer.

Results : Notably, patients who were FOG-positive exhibited significantly higher serum AQP4 levels compared to the other two groups ($p < 0.001$). These elevated levels showed a positive correlation with FOG severity ($\rho = 0.51$, $p = 0.004$). Furthermore, the serum AQP4 levels were inversely correlated with both the slope ($\rho = -0.65$, $p < 0.001$) and rate ($\rho = -0.46$, $p = 0.01$) of balance learning in PwPD. FOG-positive individuals exhibited impaired voluntary balance learning compared to both FOG-negative and healthy participants. FOG-negative participants showed initial improvement, while healthy individuals demonstrated continuous enhancement across consecutive learning blocks (the first significant main effect of time on balance performance, comparing Blocks 1 and 2, was $p = 0.02$ for FOG-negative and $p = 0.03$ for healthy groups), with both groups maintaining their acquired skills over time.

Conclusion : These findings suggest that AQP4 may play a significant role in FOG and its associated learning impairments, warranting further investigation for potential treatments. Additionally, alternative balance learning protocols may be necessary for FOG-positive patients

Keywords : Aquaporin 4, Motor learning, Neurological diseases

Count: 343
Abstract ID: 425
subject: Neurorehabilitation and Regeneration: Medication and Stem Cell Therapy
Presentation Type: Oral

Novel Combination Therapy: Injectable Amniotic Membrane Hydrogel and Metformin Synergize to Promote Sciatic Nerve Regeneration

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Background and Aim : Peripheral nerve injury repair remains a significant clinical challenge. Injectable hydrogels derived from the human amniotic membrane (AM) present a promising strategy, offering a neurosupportive scaffold rich in growth factors and anti-inflammatory agents. Metformin (MET), a drug with emerging neuroregenerative properties, may further potentiate this recovery. This study investigates the combinatorial effect of an injectable AM-derived hydrogel and metformin on sciatic nerve repair.

Methods : Sixty male rats with induced sciatic nerve compression injury were randomly allocated into five groups (n=12/group): (1) uninjured control, (2) injury model (no treatment), (3) hydrogel treatment, (4) metformin treatment, and (5) combination therapy (hydrogel + metformin). Functional recovery was assessed using the Sciatic Functional Index (SFI), Static Sciatic Index (SSI), and von Frey filament test. Electrophysiological measurements of nerve conduction velocity and extensive histological analyses were performed 14 days post-injury.

Results : Treatment with the AM hydrogel alone significantly improved functional indices (SFI, SSI), electrophysiological latency, remyelination, and the expression of regeneration markers (NF-200 and S-100β). The combination therapy group demonstrated superior outcomes, including enhanced sensitivity to mechanical stimulus, increased myelin density, robust axonal regeneration, and improved myelin sheath reconstruction. A reduction in gastrocnemius muscle mass index and an increase in collagen deposition were observed in all injured groups, consistent with denervation and repair processes.

Conclusion : Our findings demonstrate that an injectable decellularized amniotic membrane hydrogel effectively promotes peripheral nerve regeneration. The therapeutic effect is significantly augmented by co-administration of metformin, suggesting a potent synergistic strategy for improving functional and structural recovery after nerve injury.

Keywords : Decellularized amniotic membrane, amniotic membrane hydrogel, metformin, axonal regeneration, nerve functional recovery

Count: 344
Abstract ID: 894
subject: Neurorehabilitation and Regeneration: Cognitive Rehabilitation
Presentation Type: Oral

Enhancing Problem-Solving Ability through Transcranial Direct Current Stimulation of the Dorsolateral Prefrontal Cortex: A Quasi-Experimental Study in Female Students

Submission Author: Saleh Lashkari

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Background and Aim : Problem-solving is a crucial part of executive functioning. It involves planning, working memory, attention control, and cognitive flexibility. These higher-level thinking skills are mainly supported by the dorsolateral prefrontal cortex (DLPFC). This area is essential for reasoning and decision-making. Recently, non-invasive brain stimulation techniques like transcranial direct current stimulation (tDCS) have shown promise as tools to improve brain activity and cognitive performance. However, we still need more information about how the intensity of tDCS affects problem-solving skills, particularly in healthy young adults. This study aimed to explore how bilateral tDCS applied to the DLPFC influences problem-solving performance in female university students. We focused on comparing two stimulation intensities—1 mA and 1.5 mA—against a sham condition.

Methods : The study used a quasi-experimental pretest and posttest design with a sham-controlled approach. Fifteen healthy female volunteers aged 18 to 30 were randomly assigned to three groups (n = 5 each): 1 mA stimulation, 1.5 mA stimulation, and sham control. We delivered stimulation using a constant-current stimulator with saline-soaked sponge electrodes (5×5 cm). The anode was placed over the left DLPFC (F3 site, following the 10–20 EEG system), while the cathode was positioned on the opposite supraorbital area. Each participant received eight 20-minute stimulation sessions on alternate days. We assessed cognitive performance before and after the intervention using the Tower of London (TOL) task from PEBL software, which measures planning efficiency and cognitive control. The parameters evaluated included total moves made, minimum required moves, deviation from the optimal path, total task time, average time per trial, and total completed trials. We analyzed data using paired and independent t-tests, with significance set at $\alpha = 0.05$.

Results : The findings showed notable improvements in cognitive efficiency after active stimulation. In the 1 mA group, post-stimulation performance indicated mild enhancements in movement accuracy and time reduction, although these observations were not statistically significant ($p > 0.05$). Conversely, the 1.5 mA group displayed a significant reduction in deviation from the optimal path and total completion time ($p < 0.05$), indicating better problem-solving accuracy and planning efficiency. The sham group did not show significant differences between pre- and post-tests across the measured parameters. Comparisons between groups also confirmed that the higher stimulation intensity (1.5 mA) produced greater cognitive improvement than both the lower intensity and sham stimulation. Notably, no adverse effects were reported during the sessions.

Conclusion : The results suggest that anodal tDCS over the DLPFC can effectively enhance executive functions and improve problem-solving skills in healthy individuals. The intensity of stimulation appears to be a key factor in its effectiveness. Increased activity in prefrontal neural circuits likely helps with the efficient integration of planning, monitoring, and decision-making. These findings point to the potential use of tDCS as a safe, non-drug method for cognitive enhancement in educational, clinical, and rehabilitative settings. Future research should involve larger sample sizes, include participants of both genders, and incorporate neurophysiological measures like EEG or fNIRS to better understand the mechanisms and long-term effects.

Keywords : transcranial direct current stimulation (tDCS); dorsolateral prefrontal cortex (DLPFC); executive functions; problem-solving; Tower of London; cognitive enhancement.

Count: 345
Abstract ID: 895
subject: Neurorehabilitation and Regeneration: Cognitive Rehabilitation
Presentation Type: Poster

Bilateral Transcranial Direct Current Stimulation of the Dorsolateral Prefrontal Cortex Improves Cognitive Flexibility in Female Students: An Experimental Study

Submission Author: Saleh Lashkari

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Background and Aim : Cognitive flexibility, the capacity to shift attention and modify strategies in response to environmental change, is a key component of executive function primarily governed by the dorsolateral prefrontal cortex (DLPFC). Impairments in this domain are associated with disorders such as depression, ADHD, and schizophrenia. Recently, transcranial direct current stimulation (tDCS) has emerged as a non-invasive neuromodulatory technique capable of altering cortical excitability and enhancing cognitive performance. However, optimal parameters for improving cognitive flexibility remain unclear. This study examined the effects of bilateral tDCS applied to the DLPFC at two intensities (1 mA and 1.5 mA) on cognitive flexibility in healthy female university students.

Methods : A semi-experimental pretest–posttest design with a sham-controlled group was employed. Fifteen healthy right-handed females aged 18–30 were randomly assigned to three groups (n=5 each): 1 mA tDCS, 1.5 mA tDCS, and sham stimulation. Stimulation was administered for 20 minutes per session over eight sessions, conducted every other day for three weeks, using saline-soaked sponge electrodes (5×5 cm). The anode was positioned over the left DLPFC (F3) and the cathode over the right supraorbital region (Fp2), based on the 10–20 EEG system. Cognitive flexibility was assessed before and after the intervention using the Berg Card Sorting Test (BCST) implemented in PEBL software. Key indices included Perseverative Errors, Total Errors, Categories Completed, Conceptual Level Responses, Trials to First Category, Learning-to-Learn, and Failure to Maintain Set. Statistical analyses were performed with paired t-tests and one-way ANOVA at $\alpha = 0.05$.

Results : Both active stimulation groups exhibited notable improvements compared to baseline, whereas the sham group showed no significant change. In the 1 mA condition, significant reductions in Total Errors and increases in Conceptual Level Responses ($p < 0.05$) indicated enhanced abstraction and problem-solving. The 1.5 mA group showed numerical improvements across most indices, but differences did not reach significance, implying that higher intensity may not yield additional benefits. Within-group comparisons confirmed measurable behavioral enhancement without side effects such as discomfort or fatigue. Between-group ANOVA revealed a significant main effect of stimulation versus sham ($p < 0.05$), confirming that tDCS facilitated executive performance. However, there were no significant differences between the two active intensities, suggesting a plateau in efficacy beyond 1 mA.

Conclusion : Bilateral anodal tDCS over the DLPFC effectively enhanced cognitive flexibility and executive functioning in healthy young women. The improvements likely stem from polarity-dependent modulation of prefrontal cortical networks, leading to better set-shifting and conceptual reasoning. Importantly, increasing stimulation intensity from 1 mA to 1.5 mA did not enhance outcomes, indicating that lower-intensity stimulation may suffice for cognitive enhancement. These results highlight tDCS as a safe, affordable, and non-pharmacological approach to boost executive performance. Future studies should include larger and more diverse samples and integrate neurophysiological measures such as EEG connectivity or fNIRS activation to clarify underlying mechanisms and optimize stimulation protocols.

Keywords : transcranial direct current stimulation (tDCS); dorsolateral prefrontal cortex (DLPFC); cognitive flexibility; executive function; Berg Card Sorting Test (BCST); non-invasive brain stimulation.

Count: 346
Abstract ID: 791
subject: Neurorehabilitation and Regeneration: Cognitive Rehabilitation
Presentation Type: Poster

The Effect of COGMO Cognitive-Motor Training on Improving Cognitive Function, Processing Speed, and the Acquisition of a New Motor Skill in Older Adults

Submission Author: MANOCHEHR GHALKHANI

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Background and Aim : The rapid increase in the aging population has made maintaining the independence and quality of life for this demographic a priority [1]. A primary challenge in this regard is the natural decline in cognitive functions and reduced processing speed, which impairs the ability to learn new skills [2, 3]. In response to this need, the novel approach of cognitive-motor training has emerged. Based on the neural overlap of these two systems, it creates a powerful stimulus for neuroplasticity [4, 5]. Among these, the "COGMO" program is distinguished by designing tasks that require sensory-motor integration and a gradual increase in cognitive load. This approach creates a challenge that is greater than the sum of its separate cognitive and motor components and is expected to induce higher levels of neuroplasticity [6]. However, evidence regarding the effect of this specific protocol on processing speed and the ability to learn a completely new motor skill in older adults is scarce and requires further investigation. Accordingly, the main objective of this study is to investigate the effect of a period of COGMO training on improving cognitive function, processing speed, and the learning of a new motor skill in older adults. The hypothesis of the present research is that this intervention will lead to a significant improvement in these variables compared to the control group.

Methods : This study was conducted as a quasi-experimental study with a pre-test/post-test design. The statistical population consisted of older adults aged 60 to 75 years in Tehran in 2023. Using convenience sampling and based on inclusion criteria, 45 individuals were selected and randomly assigned to three groups of 15: 1. The CogMo cognitive-motor training group 2. The computerized cognitive training group 3. The control group The Cogmo device's reaction time test was used to assess processing speed, the Corsi Block test for spatial working

memory, the Pegboard test, and the Purdue Pegboard test to evaluate the learning of a new motor skill. The COGMO and computerized cognitive training groups underwent integrated cognitive-motor training for 12 weeks, three 60-minute sessions per week. The training was designed based on the principles of sensory-motor integration and a gradual increase in cognitive load. The control group engaged only in their daily activities during this period.

Results : Data analysis using Analysis of Covariance (ANCOVA) showed that both experimental groups demonstrated significant improvement in all variables compared to the control group ($p < 0.05$). However, a comparison between the experimental groups revealed: The COGMO group performed significantly better than the computerized cognitive training group in the variables of processing speed ($p < 0.01$, $F=12.3$) and learning a new motor skill ($p < 0.01$, $F=14.2$). In the domain of cognitive function, although both groups showed improvement, the COGMO group showed a statistically significant superiority in the spatial working memory subtest ($p < 0.05$, $F=7.1$).

Conclusion : The findings of this study indicate that while computerized cognitive training alone can be effective, the integrated cognitive-motor approach of COGMO, by creating a dual challenge and enhancing sensory-motor integration, has a higher efficacy, particularly in the domains of processing speed and learning new motor skills. These results confirm the importance of simultaneously engaging cognitive and motor systems in interventions for the elderly and suggest that integrated protocols like COGMO should be used in senior centers and rehabilitation clinics.

Keywords : cogmo,cognition, motor learning, elderly

Count: 347
Abstract ID: 471
subject: Neurorehabilitation and Regeneration: Cognitive Rehabilitation
Presentation Type: Poster

Non-invasive brain stimulation effects on post-stroke cognitive impairment: A network meta-analysis

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Conclusion : Introduction: stroke is the second common cause of death and the third leading factor for disability. Non-invasive brain stimulation (NIBS) has emerged as an effective option, for preserving and enhancing cognitive function following stroke. This Network Meta-Analysis attempts to compare the impact of NIBS techniques, including Repeated transcranial magnetic stimulation (rTMS) and Transcranial direct current stimulation (tCDS), on post-stroke cognitive function. Methods: A structured literature review of interventional studies was conducted across Medline, Scopus, and Psycinfo databases. After screening 220 records, published between January 2018 and August 2024, 12 studies involving 510 participants were included. The risk of bias in the included reports was assessed using the Cochrane collaboration tools. The global cognition severity was quantified using the MMSE and MoCA scales (Mini-Mental State Examination and Montreal cognitive assessment), and the functional abilities in activities of daily living (ADL) were evaluated using the MBI and FIM scales (Modified Barthel Index and Functional Independence Measurement). A Network Meta-Analysis (NMA) was conducted to compare NIBS modalities effects on cognitive function improvement. Additionally, the efficacy of NIBS was investigated across different stages of stroke recovery. All analyses were performed using the Netmeta package in R. Results: The NMA revealed significant improvement in cognitive function in NIBS treated groups compared to controls. Among the modalities, the rTMS showed the most substantial effects on global cognitive enhancement, by both the MoCA ($p < 0.0001$) and MMSE ($p = 0.001$) scales. And the most noticeable improvements were observed during the subacute phase ($p < 0.0001$). no significant

improvements were found in ADLs, likely due to variability in measurement scales. Conclusion: The Present evidences suggests that both rTMS and tDCS significantly improve cognitive function in post-stroke patients; however, the rTMS demonstrates superior outcomes. The findings highlight the critical role of prompt intervention in optimizing the rehabilitation potential. In conclusion, NIBS could be considered as a recommended approach for global cognitive performance enhancement, in PSCI patients.

Keywords : Post-stroke cognitive impairment; Non-invasive brain stimulation; Repeated transcranial magnetic stimulation; Transcranial direct current stimulation; Meta-analysis, stroke stages

Count: 348
Abstract ID: 853
subject: Social Neuroscience: Developmental approaches
Presentation Type: Poster

Applying Neuromarketing in Clinic Management: Leveraging Neuroscience to Enhance Patient Experience, Trust, and Operational Efficiency

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Conclusion : Neuromarketing provides a new perspective for understanding patients' behaviors, emotions, and decision-making processes beyond traditional self-reported data. By applying neuroscience-based tools and insights, clinic managers can design more effective environments, improve patient communication, and build stronger emotional connections that foster trust and loyalty. However, the implementation of neuromarketing in clinical settings requires careful consideration of ethical principles, data privacy, and methodological validity. It should not be used to manipulate patients but to enhance their experience, reduce anxiety, and support informed decision-making. The integration of neuromarketing with digital technologies, such as AI-driven data analytics and wearable sensors, opens new opportunities for evidence-based management in healthcare. Clinics that embrace this interdisciplinary approach — combining marketing, neuroscience, and patient-centered design — will likely achieve higher satisfaction, better outcomes, and sustainable growth in an increasingly competitive healthcare environment. In conclusion, neuromarketing can serve as a bridge between science and empathy in healthcare management, enabling clinics to understand not only what patients say but how they truly feel — and to use that knowledge to create a more humane, effective, and trustworthy clinical experience.

Keywords : Neuromarketing, neuroscience, patient behavior, trust;health marketing

Count: 349
Abstract ID: 166
subject: Social Neuroscience: Self perception and regulation
Presentation Type: Poster

From Self to Selfless: Using Virtual Reality to Induce Out-of-Body Experiences, Ego Dissolution, and Awe

Submission Author: Zahra Kheirabadi

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Background and Aim : Modern theories conceptualize the self not as a fixed entity, but as a constructed model fundamentally anchored in the body. Disrupting this constructed model provides a powerful method for studying the nature of consciousness. While pharmacological agents are known to induce states of "ego dissolution," non-invasive experimental paradigms remain scarce. We propose that the Out-of-Body Experience (OBE)—a targeted disruption of spatial and bodily self-consciousness—offers a unique, non-pharmacological tool to manipulate the self-model. This study tested whether a VR-induced OBE could experimentally induce a measurable state of self-diminishment and its associated self-transcendent emotion, awe.

Methods : Forty female students were randomly assigned to an experimental (OBE) or an active control group. Using a Meta Quest 3 headset, all participants were immersed in a VR environment and embodied in an avatar. The core manipulation involved shifting the experimental group's perspective from a standard first-person viewpoint to a disembodied, elevated third-person location. The active control group experienced a sustained, embodied first-person perspective throughout. Primary outcomes were measured using the Awe Experience Scale (AWE-S), including its dedicated "Self-Diminishment" subscale.

Results : The VR protocol produced a robust OBE illusion. The intervention caused a significantly greater experience of overall awe in the OBE group ($p < .001$). Crucially, the experimental manipulation also led to a statistically significant increase in Self-Diminishment ($p = .034$, $d = 0.71$), confirming that the perceptual decoupling from the body induced a quantifiable sense of ego-fading. This core finding was bolstered by significant increases in perceived vastness and connectedness, painting a cohesive picture of a multifaceted self-transcendent experience.

Conclusion : Our findings provide direct experimental evidence that temporarily displacing the self's perceived location from the body is a potent method for inducing a state akin to ego dissolution. The significant increase in felt self-diminishment lends empirical weight to constructivist models of the self, demonstrating that the very foundation of our "I" is malleable. This positions the VR-OBE as a valuable paradigm for the scientific study of selflessness and consciousness. The powerful induction of awe further suggests that this manipulation may serve as the underlying mechanism for the therapeutic and transformative effects reported in previous literature, opening new avenues for interventions aimed at promoting psychological flexibility, a core mechanism for enhancing well-being.

Keywords : Selflessness; Out-of-Body Experience (OBE); Virtual Reality (VR); Bodily Self-Consciousness; Awe

Count: 350
Abstract ID: 140
subject: Social Neuroscience: Interpersonal processes
Presentation Type: Oral

Judging who you are based on what I see: A Hierarchical Bayesian Drift Diffusion Account of Rapid Social Categorization

Submission Author: Mohammad Rahmani

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Background and Aim : We form a preliminary judgment of people whom we meet based on their different characteristics and expressions in less than a few seconds. This judgment, however, has significant consequences for our future transactions and communications with the person, making it a prime topic to investigate. The overarching aim of the current study was to examine how humans, as social creators, integrate multiple characteristics of a person to form a single judgment regarding whether a person would be classified as an in-group or out-group.

Methods : Sixty-nine adults first completed a group-priority task to identify, for each participant, important versus unimportant groups. Afterwards, participants completed the main task, which consisted of 144 trials where two group labels for a target were displayed, followed by a neutral face. Participants then made a speeded classification task, where they decided whether the person belonged to the in-group or the out-group. We fit a hierarchical Wiener-DDM to joint choices and reaction times (RTs). Drift rate (v) was modeled by Importance × Type; boundary separation (α), non-decision time (Ter), and starting-point bias (z) had varying intercepts.

Results : The mixed Type at unimportant Importance served as the baseline. Drift rate was near zero but slightly positive (mean = 0.15, 95% CI −0.00–0.30), consistent with behavior approaching chance under dual-membership ambiguity. Importance increased the drift rate, strengthening evidence accumulation relative to baseline (posterior shift in v = −0.38, 95% CI −0.47, −0.28; under our coding, the sign indicates direction, while the magnitude indexes speed/strength). Type showed negligible main effects: both-in-group Δv = +0.08 (CI −0.02, +0.18); both-out-group Δv = −0.10 (CI −0.19, 0.00). Critically, Type interacted with Importance was significant, showing that when the memberships were personally important, drift rate moved far from baseline in the direction of the corresponding decision—a large positive shift for in-group targets (Δv = +0.65, CI +0.51, +0.79) and a large negative shift for out-group targets (Δv = −0.35, CI −0.49, −0.22). Other parameters suggested moderate caution and efficient encoding (α≈2.4; Ter≈0.40 s; z≈0.48). Together, the hierarchical Bayesian DDM results reveal that the classification of the characteristics alone makes a negligible contribution. However, the class of characteristics becomes decisive when the characteristics are considered

personally important. Thus, motivational relevance transforms ambiguous dual-membership cues into strong, relevant evidence, yielding faster and more decisive social categorizations.

Conclusion : By mapping social constructs (Importance, ambiguity) onto mechanistic DDM parameters, this work advances a computational account of how the social brain resolves multiple group cues in real-time and highlights the utility of Bayesian DDM for social neuroscience research.

Keywords : intergroup relations- drift diffusion model

Count: 351
Abstract ID: 714
subject: Social Neuroscience: Interpersonal processes
Presentation Type: Poster

Neural Networks Underlying the Social Brain

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Conclusion : Background and Aim: The social brain encompasses a set of interconnected neural systems that allow individuals to perceive, interpret, and respond to socially relevant cues. These systems support higher-order cognitive abilities, including empathy, emotional understanding, and Theory of Mind (ToM). Converging evidence from neuroimaging and electrophysiological research indicates that social cognition relies on a distributed neural network involving the medial prefrontal cortex (mPFC), temporoparietal junction (TPJ), posterior superior temporal sulcus (pSTS), amygdala, and precuneus. Exploring the functional interactions and oscillatory dynamics among these regions can shed light on the neural substrates underlying social behavior and its disruption in neuropsychiatric and developmental conditions. This review aims to synthesize current findings on the structural and functional organization of the social brain and its neural oscillatory mechanisms. Methods: A systematic literature search was performed across PubMed, Scopus, Web of Science, and ScienceDirect databases. Eligible studies employed fMRI, EEG, or MEG to examine functional connectivity, network topology, and oscillatory synchronization during social cognition and ToM tasks. Extracted data included key brain regions, frequency bands, and patterns of connectivity involved in processing social information. Results: Evidence across modalities suggests that social cognition emerges from dynamic coordination within large-scale cortical and subcortical networks. Theta and alpha oscillations facilitate long-range integration between the mPFC and TPJ during perspective-taking tasks, while beta and gamma band activity underlies emotional mirroring and affective resonance. Moreover, time-resolved functional connectivity analyses highlight that network configurations flexibly adapt according to the type of social inference—whether cognitive or affective in nature. Conclusion: The social brain functions as an integrated neural network in which multiple oscillatory processes cooperate to support the comprehension of others' beliefs, intentions, and emotions. Mapping the spatial and temporal dynamics of these networks advances our understanding of both typical and impaired social functioning and may pave the way for identifying neurophysiological biomarkers of social cognitive deficits in disorders such as autism spectrum disorder (ASD), schizophrenia, and frontotemporal dementia.

Keywords : Social Brain, Theory of Mind, Functional Connectivity, EEG/fMRI, Neural Oscillations

Count: 352
Abstract ID: 340
subject: Social Neuroscience: Interpersonal processes
Presentation Type: Poster

The Behavioral Effect of Social Dominance Orientation on Decision-Making Under Uncertainty

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Background and Aim : Social Dominance Orientation (SDO) is a socio-attitudinal construct reflecting the extent to which individuals endorse group-based hierarchies and dominance over others. While SDO has been extensively studied in relation to intergroup attitudes and social behavior, less is known about its potential impact on cognitive processes such as decision-making under uncertainty. This study investigated the influence of SDO on decision-making under uncertainty using a modified probabilistic gambling paradigm. The aim of this study was SDO influences behavioral responses—specifically, reaction times) (RTs)—during expected and unexpected uncertainty in decision-making.

Methods : Participants were 30 university students (18–45 years old) recruited through convenience sampling at the University of Tabriz. Based on the SDO questionnaire (Sidanius & Pratto, 1999), participants were classified into two groups: high SDO and low SDO groups. Thirty participants first completed a 60-trial training session to learn fixed cue–response–feedback contingencies (cue A: 100% reward for response 1; cue B: 50% reward for response 2; cue C: 0% reward regardless of response). During the 500-trial testing phase, contingencies were altered so that cue A yielded a reward with 80% probability, cue B remained at 50%, and cue C yielded a reward on 20% of trials. This design generated three uncertainty/expectation levels: expected–certain, expected–uncertain, and unexpected–uncertain, each with positive or negative feedback outcomes. Data were analyzed using repeated-measures ANOVA with feedback type (positive vs. negative) and uncertainty level (expected-certain, expected-uncertain, unexpected-uncertain) as within-subject factors, and SDO group (high vs. low) as a between-subject factor.

Results : Results showed a significant main effect of SDO group, indicating that individuals with high SDO consistently responded faster than those with low SDO across all conditions of uncertainty. No main effects were observed for uncertainty level or feedback type alone. Post hoc tests indicated that, in general, positive feedback in expected–certain conditions elicited the fastest RTs, while in unexpected–uncertain conditions, negative feedback was associated with accelerated responding. Decomposition by group revealed that high-SDO individuals

exhibited faster responses to positive feedback in expected contexts and to negative feedback in unexpected contexts. In contrast, low-SDO participants tended to be slower overall and showed a pattern of relatively faster responding to negative feedback under violated-expectation trials.

Conclusion : These findings suggest that individuals with higher SDO scores are associated with faster decision-making across varying levels of uncertainty, regardless of the social context. Rapid responding may reflect a behavioral — and possibly biological — marker of dominance-related motivation, confidence, and approach orientation. Consistent with human and animal evidence, such speed (without loss of accuracy) could confer advantages in competitive environments and help explain links between dominance, leadership emergence, and competitive behavior.

Keywords : Social Dominance Orientation; decision-making; uncertainty; reaction time

Count: 353
Abstract ID: 657
subject: Social Neuroscience: Interpersonal processes
Presentation Type: Poster

Adolescent trust development: Comparing trust behaviors toward peers and interactive bots

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Background and Aim : Trust, a fundamental social and cognitive phenomenon, plays a pivotal role in human interactions and decision-making, particularly during the critical developmental period of adolescence—a time when peer relationships become increasingly important. The present study aimed to examine adolescents' trust behavior across different social partners.

Methods : Fifty-four adolescents (aged 13–18) participated in a computer-based Trust Game programmed in PsychoPy. The experiment employed a within-subjects design. In each trial, participants were endowed with 20 points and then decided how much money to invest with three types of agents: a peer, an adult, and an artificial agent. Each agent appeared in two conditions—trustworthy and untrustworthy—resulting in six agent conditions in total.

Results : Data were analyzed using a repeated-measures ANOVA with agent group (peer, adult, artificial agent) and agent type (trustworthy, untrustworthy) as within-subject factors. The analysis revealed a significant main effect of trustworthiness, indicating that adolescents invested more with trustworthy agents than with untrustworthy agents. However, the main effect of agent group was not significant, suggesting that the difference in trust was not systematically influenced by whether the partner was a peer, an adult, or an artificial agent. No significant interaction effect was observed.

Conclusion : The findings demonstrate that adolescents are sensitive to the trustworthiness of their interaction partners, showing greater willingness to invest when agents behave in a trustworthy manner. Future research could further explore the conditions under which peer identity becomes a stronger determinant of trust during adolescence.

Keywords : trust; trustworthiness; adolescents; social learning; trust game

Count: 354
Abstract ID: 329
subject: Social Neuroscience: Gamification
Presentation Type: Oral

Genetic Algorithm–Based Level Generation for a Pattern Memory Game

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Background and Aim : Serious games have emerged as valuable tools for the assessment and rehabilitation of cognitive functions. Among these, games designed for visual working memory play an important role in identifying and mitigating cognitive impairments such as attention deficits and age-related decline. A major challenge in such games is designing stages that are both engaging and appropriately matched to the player's ability level. Traditional approaches depend on storing large sets of predefined stages, which limits variability and adaptability. To overcome this limitation, we propose a genetic algorithm (GA)-based stage design module for a tile-based pattern memory game. The module dynamically generates new stages with targeted difficulty levels, defined as 5% performance intervals of a human memory simulator, thereby ensuring continuous novelty without the need for extensive stage libraries.

Methods : Levels are represented as tile-based matrices, constructed from three distinct tile types. Each chromosome encodes one stage. The GA begins with a randomly initialized population, while a single high-quality chromosome is introduced to improve convergence. Fitness Function: Evaluation involves two components: (1) structural validity, ensuring that each matrix satisfies minimum requirements for a playable level, such as containing a sufficient number of tiles, and (2) difficulty alignment, where the level is tested against the memory simulator to verify that the player's simulated performance falls within the desired 5% accuracy range. Selection and Elitism: Parent selection is performed using the roulette wheel method, with 20% of the top-performing chromosomes directly retained in each generation. Crossover and Mutation: Crossover occurs at a rate of 0.7, with a vertical crossover point fixed at the fifth column of the matrix. Mutation is applied at a rate of 0.05 to preserve diversity in the population. Termination: The algorithm runs for a fixed limit of 10 generations, after which the final population is evaluated for stage diversity and difficulty alignment.

Results : The proposed module successfully generated playable and diverse stages across multiple difficulty levels. Convergence occurred more rapidly in simpler stages, as their structural and difficulty constraints were easier to satisfy. At higher difficulty levels, convergence was slower because the distribution of tiles in the generated patterns approached a uniform distribution, reducing variability. Nevertheless, the GA consistently produced novel stage matrices that matched the desired simulator-based performance range. Importantly, the

method allowed each gameplay session to feature new stage instances, eliminating the need for predesigned libraries while preserving difficulty control.

Conclusion : This study demonstrates the effectiveness of a GA-based stage design module for a serious memory game targeting visual working memory. By leveraging the human memory simulator and defining difficulty as 5% performance intervals, the approach ensures that generated stages are both structurally valid and tailored to player ability. The method supports dynamic and scalable stage generation, enhancing replayability and adaptability. Future work will explore adaptive GA parameters and hybrid optimization methods to further improve convergence, particularly for higher difficulty levels.

Keywords : Genetic Algorithms; Serious Games; Visual Working Memory; Pattern Memory Game

Count: 355
Abstract ID: 300
subject: Special topics: Converging Technologies (NBIC: Nano-Biotech-Information-Cognitive), euroscience and Nanotechnology, Neuroscience and Biotechnology, Neural Tissue Engineering
Presentation Type: Poster

Application of Nanomaterials in Diagnosis of Brain Diseases and Disorders

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Conclusion : The blood-brain barrier (BBB) poses a challenge for diagnosing the brain diseases and disorders through conventional methods. Nanotechnology, by modifying and manipulating particles at the molecular level, creates nanomaterials with the intriguing physicochemical and biological properties such as the small size, large surface area, high surface-to-volume ratio, specific structural characteristics, potential for surface functionalization, and ability to cross the BBB. The combination of nanotechnology and medicine has the potential to revolutionize the diagnosis and treatment of many diseases, including the brain diseases and disorders. This study aimed to investigate the role of nanomaterials in improving early diagnosis and accuracy of the brain diseases and disorders. For this purpose, Scopus, PubMed, and Google Scholar search engines were searched for the keywords of brain, blood-brain barrier, nanomaterials, nano-contrast agents, nanosensors, and nano-neurodiagnostic. Then, the most relevant articles were reviewed. The integration of nanotechnology in diagnosing develops state-of-the-art imaging agents and nanosensors that lead to high accuracy, sensitivity, and efficiency in diagnosis. As first application, the development of nanoparticles revolutionizes the early detection of Alzheimer, Parkinson, and brain tumors through nano-contrast agents used in magnetic resonance imaging (MRI), computed tomography (CT) scan, and fluorescence imaging. Additionally, contrast agents have the ability to bind to nanocarriers that not only can penetrate the BBB but also exhibit high biocompatibility. The contrast agents can also be considered as diagnostic portion of the nanotheranostics which are loaded with the drugs. Furthermore, functionalizing the surface of these nanoparticles with the ligands can improve the contrast media sensitivity, create high spatial resolution, and increase detection accuracy. Moreover, nanocarriers conjugated with radiopharmaceuticals such as 99m technetium are able to detect neurodegenerative disorders and the brain diseases like tumors in their early stages using single photon emission computed tomography (SPECT) or positron emission tomography (PET). The second application of nanotechnology in this field is using the nanomaterials as nanosensors. Nanoparticle-based biosensors have demonstrated remarkable capabilities in detecting biomarkers for the brain disorders such as Alzheimer, leading to the early diagnosis of neurological abnormalities with high efficiency and specificity. Although using nanomaterials in medicine has many benefits,

there are also some limitations such as safety issues and retention times within targets. Indeed, overcoming these challenges will open the door to translating the approaches from animals to widespread medical use of the nanomaterials in human. Finally, the convergence of nanotechnology with machine learning algorithms and artificial intelligence has the potential to modify diagnostic accuracy. As conclusion, nanomaterials offer a promising platform for diagnosing the brain diseases and disorders with a high accuracy. The ongoing advancements in nano-neurodiagnostics shine a light of hope for patients with the brain diseases and disorders.

Keywords : Brain; Blood-brain barrier; Nanomaterials; Nano-contrast agents; Nanosensors; Nano-neurodiagnostics

Count: 356
Abstract ID: 837
subject: Special topics: Converging Technologies (NBIC: Nano-Biotech-Information-Cognitive), euroscience and Nanotechnology, Neuroscience and Biotechnology, Neural Tissue Engineering
Presentation Type: Poster

Bioinformatics-Based Analysis of Effective Signaling Pathways in Glioblastoma

Submission Author: Mehregan Noghreh

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Background and Aim : Glioblastoma multiforme (GBM) is a World Health Organization (WHO) grade IV glioma and represents the most common and aggressive form of primary brain cancer. The highly infiltrative nature of GBM, poor prognosis, marked genetic, genomic, and epigenetic heterogeneity, and protection by the blood–brain barrier (BBB) lead to significant treatment challenges. In addition, GBM is characterized by poor survival, with a median overall survival of approximately 15 months. It is difficult to determine definitively the incidence of GBM, as it varies depending on the report, ranging from 3.19 to 4.17 cases per 100,000 person-years. In addition, the incidence in the pediatric population (0–18 years) is 0.85 per 100,000, with pediatric glioblastoma multiforme (p-GBM) accounting for 3–15% of primary brain tumors in this age group. Primary tumors of the central nervous system are the second-most-common type of cancer in children and the most common among solid tumors. These factors make GBM a significant challenge in the field of oncology. Due to the limitations in the discovered molecular mechanisms of GBM, this study directly focused on the potential role of EZH2 and SOX2 based on bioinformatics and signaling pathway statistics.

Methods : Microarray analysis was performed in the field of glioblastoma stem cells from the GEO database. After GEO2R analysis, a gene showing significant upregulation was identified. Gene expression, survival analysis, and isoforms of the chosen gene were evaluated using GEPIA2. Also, the TCGA database was used to examine the expression of these genes in various cancers. The signaling pathways of the selected genes were analyzed using the KEGG pathway. Subsequently, the interactions between proteins related to our genes were identified using STRING. Then, the related miRNAs were extracted from miRTarBase, TargetScan, and miRDB, and the common miRNAs were identified using Cytoscape.

Results : Genes EZH2 (LogFC = 6.677, p-value = 2.53×10^{-19}) and SOX2 (LogFC = 3.563, p-value = 4.71×10^{-19}) were selected from dataset GSE4290 as significantly upregulated genes in glioblastoma. Expression validation in the GEPIA2 and TCGA databases confirmed their

high levels in GBM samples compare with normal tissues. Survival analysis revealed that high expression of these genes was correlated with increase cell survival and as result reduced overall survival in glioblastoma patients. Subsequently, 8 miRNAs were identified for EZH2 and three for SOX2 from miRTarBase, TargetScan, and miRDB databases. The shared miRNA confirms a possible co-regulatory mechanisms between EZH2 and SOX2 and they may participate in similar signaling pathways involved in GBM progression

Conclusion : According to STRING database analysis, EZH2 and SOX2 show a functional association that indicates a dependable regulatory relation in glioblastoma. EZH2, as a histone methyltransferase and a main component of the Polycomb Repressive Complex, and SOX2, as a key transcription factor involved in maintaining stemness and promoting tumor progression, may interact functionally in GBM. The STRING network highlights direct and indirect protein–protein interactions, supporting the hypothesis that EZH2 and SOX2 are functionally connected and may cooperate in glioblastoma pathogenesis

Keywords : Glioblastoma multiform, bioinformatics, glioblastoma stemcell

Count: 357
Abstract ID: 754
subject: Special topics: Neurolaw
Presentation Type: Poster

The Role of Governments in Supporting Children's Rights Affected by Electronic Screen Syndrome in light of Neurolaw

Submission Author: Mahshid Jamali

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Background and Aim : Background and Purpose: In the age of technology, the use of electronic screens has become an integral part of children's lives. The increase in time spent in front of screens, such as televisions, tablets, smartphones, laptops, and gaming consoles—especially at a young age—has led to various physical and psychological harms known as Electronic Screen Syndrome (ESS). Research indicates a strong correlation between excessive screen use in childhood and issues such as visual problems, language and brain development difficulties, attention deficits, behavioral disorders and similar concerns. The Convention on the Rights of the Child (CRC, 1989) recognizes the child's right to health in Article 24 and assigns governments the responsibility to ensure this right. Therefore, as responsible entities for protecting children's rights, governments are obligated to take measures to mitigate these risks and promote the physical and mental health of children. This research examines the role of governments in this context and analyzes the impacts of excessive use of electronic screens on children's brains and behavior from a neurolaw perspective, as well as the violation of their right to health. It also seeks to answer the question: What role do governments play in supporting the rights of children affected by Electronic Screen Syndrome?

Methods : This research is applied in nature and employs a descriptive-analytical methodology. The materials have been collected and analyzed using library resources, a wide range of documents& practices, and other research works.

Results : Ultimately, this study asserts that according to international law principles, governments are required to design and implement educational, preventive and supportive policies that include educating and raising awareness among family units, imposing time restrictions on electronic screen use, developing interactive infrastructures in the real world and formulating laws while overseeing their implementation.

Conclusion : The conclusion of this research indicates that effective governmental support can enhance the quality of life for children affected by Electronic Screen Syndrome and facilitate the realization of their right to health.

Keywords : Neurolaw, Electronic Screen Syndrome, Children, Governments

Count: 358
Abstract ID: 871
subject: Special topics: Art and Neuroscience
Presentation Type: Poster

The Brain's Role in Art Valuation: A Neuro-Economic Study of Iranian Modern/Contemporary Art

Submission Author: Bahareh Rafiee

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Background and Aim : Art valuation involves complex cognitive and emotional processes, integrating aesthetic appreciation, personal preference, and economic judgment. Neuroeconomics combines neuroscience and behavioral economics to investigate how the brain evaluates value, reward, and decision-making. While Western art has been extensively studied, the neural mechanisms underlying the valuation of Iranian modern and contemporary art remain largely unexplored. This study examines brain activity associated with subjective art valuation in Iranian participants, using functional MRI to identify regions involved in reward processing, aesthetic judgment, and decision-making. Understanding these neural correlates may provide insights into cultural influences on art perception and the economic choices related to art markets.

Methods : Participants Fifteen healthy adult volunteers (8 males, 7 females; age range 20–45 years) with normal or corrected-to-normal vision participated. All had no history of neurological or psychiatric disorders and provided written informed consent. The study protocol was approved by the institutional ethics committee. Stimuli and Task: Participants were presented with a curated set of 60 Iranian modern and contemporary artworks, balanced for style, medium, and cultural relevance. Each artwork was displayed for 6 seconds, followed by a 4-second interval during which participants . fMRI Data Acquisition: Functional images were acquired using a 3T Siemens Prisma scanner with a 32-channel head coil. T2*-weighted EPI sequences were used (TR = 2000 ms, TE = 30 ms, flip angle = 90°, voxel size = 3×3×3 mm^3, 36 slices). High-resolution T1-weighted anatomical images were also collected for normalization. Preprocessing: Data preprocessing was conducted using SPM12, including slice timing correction, realignment, coregistration, normalization to MNI space, and spatial smoothing with a 6 mm FWHM Gaussian kernel. Volumes with framewise displacement exceeding 0.5 mm were censored. Data Analysis: Brain regions associated with art valuation were identified using general linear modeling (GLM), correlating BOLD responses with individual monetary ratings. Regions of interest (ROIs) included the ventromedial prefrontal cortex, orbitofrontal cortex, striatum, and posterior parietal cortex. Group-level contrasts were analyzed using a significance threshold of $p < 0.05$, FDR corrected. Exploratory whole-brain analyses were also conducted to detect additional neural correlates.

Results : Functional MRI analysis revealed significant activation in the ventromedial prefrontal cortex (vmPFC), orbitofrontal cortex (OFC), and ventral striatum during art valuation, correlating with participants' subjective monetary ratings. Posterior parietal and lateral prefrontal regions were also engaged, reflecting decision-making and attention processes. SMS analysis indicated stronger temporal correlations in reward-related regions, while aesthetic judgment consistently activated occipital and temporal visual areas. These findings suggest that both reward evaluation and perceptual processing contribute to art valuation. Overall, the neural response patterns highlight the integration of cognitive and affective mechanisms in assigning economic value to Iranian modern and contemporary artworks.

Conclusion : Art valuation in Iranian modern and contemporary works engages reward-related regions, including the vmPFC, OFC, and striatum, alongside perceptual and attentional networks. These findings highlight the interplay of cognitive, emotional, and economic processes in aesthetic decision-making, providing insight into the neural mechanisms underlying culturally influenced art valuation.

Keywords : art valuation, neuroeconomics, fMRI, reward, aesthetics, Iranian art

Count: 359
Abstract ID: 884
subject: Covid-19 and Nervous System: Basic and Clinical Aspects: Covid-19 and Nervous System: Basic and Clinical Aspects
Presentation Type: Poster

Impact of the COVID-19 outbreak on ischemic stroke incidence, subtypes, admission patterns, and severity: A comparative study at Shariati Hospital, Tehran (2019–2020)

Submission Author: Aysan Valinejad

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Background and Aim : Background and aim: Ischemic stroke is one of the leading causes of death and disability. According to reports, COVID-19 has caused a decline in hospitals for stroke patients, altering the patterns of patient admission. This study aims to determine the impact of COVID-19 on hospitalization rates, the severity of strokes, and the subtypes of ischemic strokes among patients admitted to Tehran's Shariati Hospital.

Methods : This retrospective cross-sectional study was conducted on patients diagnosed with ischemic stroke who were admitted to Shariati Hospital in Tehran between March 2019 and December 2020. Patient data were extracted from hospital records, including demographic characteristics, stroke subtypes, and National Institutes of Health Stroke Scale (NIHSS) scores. Stroke classification was based on findings from Magnetic Resonance Imaging with Diffusion-Weighted Imaging (MRI-DWI). The study compared the incidence, subtypes, admission patterns, and severity of ischemic stroke before and after the onset of the COVID-19 pandemic, which was defined as beginning in February 2020.

Results : No statistically significant differences were observed in the mean age at admission or overall length of hospital stay between the pre- and post-pandemic periods. However, female patients exhibited a significantly longer hospital stay compared to male patients ($p < 0.005$). Following the onset of the COVID-19 pandemic, a significant increase was noted in the number of large vessel stroke cases, whereas the incidence rates of other ischemic stroke subtypes remained relatively stable.

Conclusion : The COVID-19 pandemic was associated with an increased incidence of non-small vessel ischemic strokes and higher hospitalization rates, while a decrease in ischemic stroke admissions among women was observed. These changes may reflect delays in seeking care and fear of hospital exposure during the pandemic. Enhancing public awareness and conducting further research into the contributing factors are recommended to address these shifts in stroke patterns.

Keywords : Ischemic stroke; COVID-19; stroke severity; stroke subtype

Count: 360
Abstract ID: 295
subject: Covid-19 and Nervous System: Basic and Clinical Aspects: Covid-19 and Nervous System: Basic and Clinical Aspects
Presentation Type: Poster

Immune system reaction to concurrent injection of botulinum toxin and covid-19 vaccination: A Short Review

Submission Author: Fedra Mokhtari

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Conclusion : The COVID-19 pandemic caused by a new virus from the Coronaviridae family, has resulted in significant loss of life and disrupted global routines since December 2019. To prevent further infections, widespread vaccination is essential. However, many individuals worldwide also receive Botulinum Toxin for cosmetic or therapeutic purposes. Botulinum Toxin is a Clostridium botulinum toxin that induces temporary muscle paralysis. Reports have emerged of immune reactions and swelling in those who have received filler treatments after getting the Covid-19 vaccine, raising questions about the potential effects of the vaccine on individuals who have received Botulinum Toxin. Currently, there is no scientific data available on this subject, which poses a challenge for physicians and their patients, particularly cosmetic doctors. Therefore, this study aims to investigate whether it is feasible to neutralize the effects of Botulinum Toxin after vaccination or to trigger an immune response in individuals who receive both the Covid-19 vaccine and Botulinum Toxin simultaneously.

Keywords : Covid-19, Botox, Vaccination, Botulinum Toxins, Immunogenicity, SARS-CoV-2

Count: 361
Abstract ID: 63
subject: Covid-19 and Nervous System: Basic and Clinical Aspects: Covid-19 and Nervous System: Basic and Clinical Aspects
Presentation Type: Poster

Title: COVID-19 and neuroinflammation: PPAR agonist drugs as new therapeutic agents

Submission Author: Zahra Rezae

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Conclusion : Background: The COVID-19 pandemic, caused by severe acute respiratory syndrome (SARS-CoV-2) can cause multiple complications in the body, including effects on the central nervous system (CNS). Neurological symptoms and signs such as anosmia, ageusia, stroke, encephalopathy, and cognitive impairment have been reported in both acute and post-acute phases. Common manifestations of COVID-19 are largely attributed to neuroinflammation and dysregulation of immune system responses that lead to apoptosis and proptosis. In this context, Peroxisome proliferation-activated receptors (PPARs)-members of the nuclear receptor proteins involved in lipid and lipoprotein metabolism and immune regulation-have emerged as anti-inflammation, improvement in inflammatory states, and neuroinflammation. PPARs, especially isoforms of PPARγ and PPAR-α, have recently attracted attention as anti-inflammatory and neuroprotective factors. This review investigated current evidence on the involvement of PPARs in anti-inflammatory activity and neuroprotection. Methods: This review aims to show the role of PPARs as anti-inflammatories in the CNS. This study review was conducted using search databases, including Google Scholar, PubMed, Scopus, and ScienceDirect. This study collected articles between January 2020 and July 2025, using the keywords COVID-19, SARS-CoV-2, PPARs, neuroinflammation, and central nervous system. Selected articles included case reports, review articles, and book chapters that demonstrated the role of PPARs in neuroinflammation, particularly in the context of COVID-19. Results: COVID-19 infection is commonly associated with symptoms that take place in acute and post-acute phases. In some patients, COVID-19 causes to increase of levels of IL-6, TNF-α, and other pro-inflammatory cytokines, and finally leads to significant neuroinflammatory responses, such as apoptosis and proptosis. These responses are γ caused due to blood-brain barrier disruption, neuronal damage, and glial cell activation. PPARs, especially PPAR-γ, have been shown to inhibit NK-Kb signaling, inhibit cytokine production, and reduce oxidative stress. Studies of preclinical have shown that some of the PPAR agonist drugs can reduce neuroinflammatory markers and improve neurological symptoms like encephalitis, ischemic injury, and neurodegeneration. Even though direct clinical evidence in COVID-19 patients remains limited, these studies suggest that PPAR activation may be beneficial in neuroprotection of the CNS against SARS-CoV-2. Conclusion: COVID-19 infection is commonly associated with symptoms that take place in acute and post-acute phases. In some patients, COVID-19 causes an increase in levels of IL-6, TNF-α, and other pro-inflammatory cytokines, and finally leads to significant neuroinflammatory

responses such as apoptosis and pyroptosis. These responses result from blood-brain barrier disruption, neuronal damage, and glial cell activation. PPARs, especially PPAR-γ, have been shown to inhibit NF-kB signaling, inhibit cytokine production, and reduce oxidative stress. Preclinical studies have shown that certain of the PPAR agonist drugs can reduce neuroinflammatory markers and improve neurological symptoms like encephalitis, ischemic injury, and neurodegeneration. Even though direct clinical evidence in COVID-19 patients remains limited, these studies suggest that PPAR activation may be beneficial in the neuroprotection of SARS-CoV-2-induced CNS pathology.

Keywords : Keywords: COVID-19; central nervous system; PPAR agonist drugs; neuroinflammation