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Meeting Abstracts

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Role of Testosterone Signaling in Microglia: A Potential Role for Sex-related Differences in Alzheimer's Disease

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Abstract

Alzheimer's disease (AD) is more prevalent in women than in men, suggesting that testosterone, a male sex hormone, may protect against the progression of AD pathology. Microglia, which are innate immune cells in the brain, regulate to AD progression by clearing amyloid-β (Aβ) through autophagy. Thus, we examined how testosterone regulates autophagy and Aβ clearance via its non-genomic receptor, GPR class C group 6 member A (GPRC6A), a GPR for testosterone. In human microglial clone 3 and mouse microglial cell lines (MG6), testosterone suppressed extracellular signal-regulated kinase (ERK) phosphorylation, inhibited mammalian target of rapamycin activation, and enhanced Aβ-induced autophagic flux, as indicated by increased microtubule-associated protein 1 light chain 3-II and decreased sequestosome 1/protein (p62)). Silencing GPRC6A in MG6 cells eliminated these effects, confirming the necessity of GPRC6A in signaling. In male 5xfamilial AD model mice, orchiectomy increased Aβ deposition and p62 accumulation in ionized calcium-binding adapter molecule 1-positive microglia. However, testosterone supplementation restored Aβ pathology. Consistently, orchiectomy increased ERK phosphorylation, which was normalized with testosterone replacement. Analysis of postmortem human brain samples from patients with AD revealed lower Aβ accumulation and reduced colocalization of Aβ with p62 aggregates in male microglia compared to female microglia, suggesting enhanced autophagic activity in males. Taken together, these findings demonstrate that testosterone–GPRC6A signaling promotes microglial autophagy and facilitates Aβ clearance, which may explain why men are less susceptible to AD.

Resilience to Alzheimer's Disease by Targeting Adult Hippocampal Neurogenesis

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Abstract

Alzheimer's disease (AD) is the most predominant form of age-related dementia, characterized by progressive deterioration of memory and cognitive function. One of the first brain regions to be affected in AD patients is the hippocampus, where adult hippocampal neurogenesis (AHN), a relatively novel form of brain plasticity that refers to the creation of new neurons, occurs. Despite being shown to persist throughout life in humans, AHN is impaired significantly in AD patients. Therefore, it becomes important to consider whether AHN impairment is a contributing factor to the learning and memory impairments seen in AD and whether restoring AHN could ameliorate or delay cognitive dysfunction. We found that suppressing AHN early in AD transgenic 5x familial AD (FAD) mice led to worsened cognitive performance and loss of preexisting dentate neurons. Notably, while stimulation of AHN alone did not ameliorate cognitive dysfunction, increasing both AHN and brain-derived neurotrophic factor could improve cognition in 5xFAD mice. These results suggest that adult-born neurons play critical roles in maintaining hippocampal neuronal populations in AD and that increasing AHN could be a potentially powerful adjunct therapeutic for AD that provides functional recovery. In this presentation, we will go into an in-depth exploration of the implications of AHN impairment to AD progression and examine the therapeutic potential of AHN to this complex neurodegenerative disease.

Bridging Integrator 1-Ras and Rab Interactor 3 Interaction Regulates Endosomal Pathology in Human Induced Pluripotent Stem Cell-derived Neurons

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Abstract

Alzheimer's disease (AD) risk is strongly influenced by genetic variants that disrupt endosomal homeostasis. Among these, bridging integrator 1 (BIN1) and Ras and Rab interactor 3 (RIN3) have emerged as susceptibility genes, but their functional relationship in AD is poorly understood. Here, we investigated how BIN1–RIN3 interactions regulate Ras-related protein Rab-5 (RAB5) activity and endosomal pathology. Using RIN3 constitutive knockout mice, in which exon 3 of RIN3 is flanked by locus of crossing over (X) in P1 bacteriophage sites, we found that partial loss of RIN3 expression attenuates endosomal enlargement compared to wild type. In human induced pluripotent stem cell (hiPSC)-derived neurons, clustered regularly interspaced short palindromic repeats (CRISPR)/CRISPR-associated protein 9-mediated knockout of BIN1 enhanced RIN3-mediated RAB5 activation and endosomal enlargement. Whole-genome sequencing of the National Institute of Mental Health multi-ethnic AD family dataset identified two rare missense mutations in exon 6 of RIN3 (rs74074811 and rs74074812), located within the BIN1-binding domain and in tight linkage disequilibrium with AD-associated genome-wide association study single nucleotide polymorphism (SNP). Introduction of these mutations into hiPSC-derived neurons disrupted BIN1 binding, enhanced RAB5 hyperactivation, and caused endosomal enlargement, an early AD hallmark. Notably, these SNPs have also been linked to Paget's disease of bone, suggesting pleiotropic effects of RIN3 variants. Transcriptomic profiling of BIN1-deficient hiNeurons revealed dysregulation of neurodegeneration-related pathways, further supporting BIN1's role in maintaining neuronal endosomal homeostasis. In summary, we identify BIN1 as a critical negative regulator of RIN3-driven RAB5 activation. Disruption of BIN1–RIN3 binding, either by genetic deletion or pathogenic RIN3 variants, leads to endosomal enlargement and gene expression changes associated with AD pathogenesis.

The Gain-of-function Mutation, T96K, in the Triggering Receptor Expressed on Myeloid Cells 2 Gene Increases Risk for Alzheimer's Disease by Leading to Dysfunctional Microglia

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Abstract

Genetic studies of Alzheimer's disease (AD) have revealed triggering receptor expressed on myeloid cells 2 (TREM2) among dozens of AD-associated genes involved with microglial function. TREM2, an innate immune receptor, regulates microglial activation, phagocytosis, and biosynthetic metabolism in AD. Our prior work established that the T96K mutation in TREM2 confers a gain-of-function *in vitro*, based on its ability to increase ligand-dependent activation. Most recently, we reported that the gain-of-function mutation, TREM2T96K is associated with increased risk for AD using a whole-genome sequencing dataset comprised of both family-based and case-control samples. To investigate the effects of a TREM2 gain-of-function mutation in AD, we generated a novel TREM2T96K knockin mouse model. We show that TREM2T96K markedly reduces the capacity of microglia to encase amyloid beta (Aβ) plaques, decreases levels of soluble TREM2, impairs the transition of homeostatic microglia into disease-associated microglia (DAM), and reduces total area of phagocytic microglia, specifically, in female 5x familiar AD mice. We also observed lower levels of soluble TREM2 and impaired uptake of Aβ in TREM2T96K knockin microglial cells. Single-cell RNA sequencing revealed concomitant suppression of DAM gene signature, activation of homeostatic genes, and defects in microglial trajectories from homeostatic state into DAM. Thus, we show that like the loss-of-function mutation R47H, a gain-of-function TREM2 mutation (T96K) also leads to impaired microglial function in a sex-dependent manner. Since TREM2 is currently one of the most targeted AD genes in the pharmaceutical industry (e.g., VG-3927 and MNA-001), our findings should help guide the development of TREM2-targeted therapies for the prevention and treatment of AD.

From Bedside to Bench: Using Natural Language Processing and Dimensional Psychiatry to Advance Post-mortem Brain Research

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Abstract

Post-mortem brain research is vital to advancing biological psychiatry, but traditional approaches rely on categorical diagnoses that obscure the complexity of psychiatric illness. To address this, we developed a pipeline using natural language processing (NLP) aligned with the research domain criteria (RDoC) framework to extract dimensional phenotypes from electronic health records (EHRs). In collaboration with McLean Hospital and the Harvard Brain Tissue Resource Center, we validated this approach by benchmarking NLP-derived RDoC scores against psychometric instruments including quick inventory of depressive symptomatology, Snaith–Hamilton pleasure scale, perceived value of the stimulus scale, and generalized anxiety disorder 7-item scale. In 358 psychiatric inpatients, NLP scores for positive and negative valence systems showed strong correspondence with clinical symptoms: depression correlated with both negative ($\beta = 0.45$, $p = 0.015$) and positive valence ($\beta = -0.42$, $p = 0.008$); anxiety was associated with negative valence ($\beta = 0.29$, $p = 0.049$). We then applied this NLP-RDoC method to post-mortem donor EHRs from a well-characterized Alzheimer's disease cohort. Dimensional cognitive scores were significantly associated with neuritic plaque load and Braak stage and enhanced the explanatory power of gene expression models beyond traditional diagnoses. Region-specific transcriptomic changes, including in the insula and BA32, mapped onto cognitive symptom severity and highlighted pathways involved in neuroinflammation and synaptic plasticity. This work bridges clinical psychiatry and molecular neuroscience, enhancing the specificity and translational potential of post-mortem research. Our framework allows for lifespan phenotyping, facilitates mechanistic discovery, and supports the development of precision treatments across neuropsychiatric conditions.

Development of Clinical-grade Autologous Human Induced Pluripotent Stem Cells-derived Dopaminergic Progenitors for Personalized Cell Therapy in Parkinson's Disease

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Abstract

Parkinson's disease (PD) is a common neurodegenerative disorder characterized by the progressive loss of midbrain dopaminergic (mDA) neurons in the substantia nigra, leading to debilitating motor symptoms. Cell replacement therapy using human pluripotent stem cells including human embryonic stem cells and human induced pluripotent stem cells (hiPSCs) has therefore emerged as a promising therapeutic strategy. To enable hiPSC-based personalized cell therapy for PD, we have developed an integrated platform comprising three core technologies: (1) a second-generation reprogramming method that combines Yamanaka factors with metabolism-regulating microRNAs; (2) a scalable, reproducible *in vitro* differentiation process that generates high-quality mDA progenitors; and (3) a chemical purification step that selectively eliminates residual undifferentiated cells from the final product to enhance safety. Using this platform, we established clinical grade hiPSC lines from fibroblasts of multiple PD patients. These patient-derived hiPSC lines exhibit genomic integrity, unbiased pluripotent differentiation potential, and no detectable oncogenic variants. From these hiPSCs, we produced mDA progenitors with confirmed identity and high purity, free of residual undifferentiated cells and without pathogenic alterations in cancer-associated genes. Across multiple patient-derived lines, preclinical studies established safety and efficacy benchmarks and informed practical release criteria for manufacturing. Collectively, these advances supported recent Food and Drug Administration clearance of a phase 1 clinical trial in the United States enrolling eight patients with sporadic PD. In this presentation, we will describe our source technologies, manufacturing and quality-control framework, and the preclinical evidence supporting autologous hiPSC-based cell therapy for PD.

Intraputaminal CaV1.3 RNAi Therapy in Aged Parkinsonism Male and Female Nonhuman Primates Reverses Parkinsonian Behavioral Deficits and Reveals Evidence of Nigrostriatal Dopamine-phenotype Restoration

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Abstract

We developed an adeno associated virus (AAV)-CaV1.3-short hairpin (sh)RNA that provides potent/target specific reduction of CaV1.3 channels, which delivered to the parkinsonian putamen of aged macaques reverses long-standing behavioral deficits 'OFF' medication, enhances levodopa motor response, and prevents levodopa-induced dyskinesias (PMID: 40437755). Studies exploring mechanisms underlying these unprecedented benefits suggest that our RNA-interference (RNAi) therapeutic provides neuroregenerative efficacy to injured nigrostriatal dopamine (DA) neurons. In our model, monkeys first receive the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) into the right carotid artery which induces rapid/extensive death of right substantia nigra (SN) DA neurons. Subsequent repeated weekly intravenous MPTP results in bilateral parkinsonism. However, left SN DA neurons subjected to systemic dosing show less overt cell death, often demonstrating down-regulation of tyrosine hydroxylase (TH) without/prior to cell death. AAV is well-known for retrograde transduction and we observe AAV-CaV1.3-shRNA retrograde transport to SN that appears to restore DA-phenotype (i.e., TH-immunoreactivity) in subpopulations of presumably quiescent DA neurons in the left SN. Despite similarities in TH-fiber denervation in left putamen, AAV-CaV1.3-shRNA monkeys have significantly more TH-ir neurons in left compared to right SN and compared to left and right SN in AAV-SCR-shRNA controls. Further, evidence suggests that left SN DA neurons have restored DA function indicated by elevated/normalized DA-transporter levels in left putamen despite continued sparsity of putaminal TH. In SN, CaV1.3 channels are implicated in DA neuron vulnerability to neurodegeneration in PD. It is reasonable to suggest that retrograde transduction of SN DA neurons with AAV-CaV1.3-shRNA restores physiological balance, promoting DA phenotype and motor function restoration.

A Common miRNA-mediated Inflammatory Signature Links Gut Dysfunction, Depression, and Parkinson's Disease

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Abstract

Parkinson's disease (PD) is characterized by debilitating non-motor symptoms, such as depressive disorder (DD) and gastrointestinal (GI) dysfunction, which often precede motor signs. The "body-first" hypothesis suggests that α-synuclein pathology originates in the gut, primarily affecting serotonergic pathways in the midbrain and driving these early comorbidities. Chronic inflammation links PD, DD, and inflammatory bowel disease (IBD) in a pathological triad, but the molecular mechanisms are unclear. Our hypothesis is that certain microRNAs (miRNAs) associated with IBD are a common pathophysiological factor connecting these conditions. We analyzed post-mortem brain tissue from patients with PD and DD and PD-like mouse model with depressive phenotype. Using RNAseq and quantitative reverse transcription polymerase chain reaction, we identified the expression of three specific miRNAs associated with IBD (miR-199a-5p, miR-219a-5p, miR-200a-3p) and key immune inflammatory pathways that are also altered in a concordant manner in the brains of patients with PD and DD, a finding that was replicated in the mouse model. This distinctive miRNA signature was strongly correlated with a proinflammatory state, evidenced by elevated levels of cytokines interferon gamma and tumor necrosis factor alpha, as well as nuclear factor kappa b subunit 1 in the brain and GI. Our findings provide compelling evidence for a common miRNA mediated inflammatory mechanism underlying the pathological triad of PD, DD, and GI dysfunction. This shared molecular signature deepens our understanding of the gut-brain axis pathology in neurodegeneration. It is critical to highlight that these miRNAs are promising biomarkers for identifying individuals at risk and as new therapeutic targets for treating the debilitating non-motor symptoms of PD.

Age-associated Cortical Thinning in Speech Motor Regions Precedes Hippocampal Decline: Implications for Alzheimer's Disease

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Abstract

Background: Speech-motor and cognitive impairments are central to neurodegenerative diseases such as mild cognitive impairment and Alzheimer's disease (AD). Although motor and cognitive systems are linked, speech motor control is an overlooked domain that may decline independently and precede cognitive impairment, confounding measures of higher cognition. Associations between cognition and neural indices of speech motor control remain unexplored. This study identifies cortical thickness in speech motor regions across adulthood and evaluates whether changes emerge earlier than hippocampal decline.

Methods: Participants were 699 cognitively healthy adults (56% female, aged 35 - 90) from the Human Connectome Project (HCP)-aging dataset. Cognition was assessed using trail making tests A/B, Rey auditory verbal learning test, and a composite score. Whole-brain T1/T2 magnetic resonance imaging data from 3 Tesla scanners underwent HCP minimal preprocessing. Cortical thickness in premotor, motor, somatosensory, insular, and prefrontal regions was compared to hippocampal volume; volumetric measures were adjusted for intracranial volume.

Results: Age strongly correlated with cognition, hippocampal volume, gray matter, and speech motor cortical thickness. Although hippocampal volume predicted cognitive performance after controlling for age, these associations attenuated following multiple-comparison correction. Notably, premotor and motor regions showed stronger and earlier age associations than hippocampal volume, suggesting these regions may be more sensitive to early neurodegenerative change.

Discussion: Findings identify speech motor regions as underrecognized but promising indicators of aging and preclinical decline. Their earlier, steeper structural deterioration relative to the hippocampus suggests speech motor systems could serve as sensitive targets for early detection and monitoring of AD-related neurodegeneration, informing refinement of diagnostic criteria and intervention timing.

Targeting Macrophage Migration Inhibitory Factor as a Novel Therapeutic Target in Amyotrophic Lateral Sclerosis

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Abstract

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disorder marked by progressive loss of upper and lower motor neurons. Both familial and sporadic ALS involve protein misfolding and aggregation, with misfolded superoxide dismutase 1 (SOD1) contributing to mitochondrial dysfunction, endoplasmic reticulum stress, and proteostasis failure, and mislocalized transactive response DNA-binding protein-43 (TDP-43) disrupting nuclear gene regulation. We previously identified macrophage migration inhibitory factor (MIF) as a chaperone that suppresses mutant SOD1 toxicity *in vitro* and *in vivo* and demonstrated that MIF levels are inherently low in murine motor neurons, ALS patient–derived induced pluripotent stem cell motor neurons, and human postmortem spinal cord tissues. To assess the therapeutic potential of MIF in ALS, we applied gene therapy and protein engineering approaches in cellular and mouse models. Screening of MIF variants in neuronal cells identified a triple-mutant protein with enhanced stability and anti-aggregation activity. *In vivo*, we delivered adeno associated virus (AAV)-MIF via a single peripheral intravenous injection to symptomatic SOD1 and TDP-43 transgenic mice and monitored disease progression through behavioral testing, neuroscore, and molecular analyses. Peripheral AAV-MIF treatment in SOD1G37R mice improved motor performance, reduced neuroinflammation and misfolded SOD1, and partially normalized dysregulated inflammatory and stress-response pathways. Preliminary findings in the TDP-43Q331K model indicate that IV AAV-MIF also mitigates disease severity. Although MIF does not directly bind TDP-43, it enhances its nuclear localization *in vitro*, suggesting an indirect chaperone effect. Together, these results support MIF-based therapies—delivered peripherally

or through engineered variants—as a promising strategy to modulate both SOD1 and TDP-43 pathology and potentially slow ALS progression.

Neurophenomenology in Retraining Musicians with Task-specific Focal Dystonia: Three Cases

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Abstract

Introduction: Musicians experience a high rate of playing-related pain and injury due to the rigorous nature of repetitive mind-body training, specifically musculoskeletal disorders, performance anxiety, and task-specific neurological disorder (TSND). TSND is the least understood by the performing artists. Neurophysiological evidence has demonstrated that extensive training and excessive practice can yield maladaptive changes in certain neural networks, leading to task-specific focal dystonia. Mindfulness mental training with biomechanical pedagogy has shown promise in modifying the neurological behavior pattern.

Purpose: In this presentation, we share retraining coaching session details of three professional task-specific focal dystonic musicians. We use neurophenomenological first- and second-person methods to frame our mindfulness and mind-body integrative rehabilitation process.

Approach: We experimented with mindfulness mental training with three professional musicians with task-specific focal dystonia at the height of their careers. The sessions consist of deep breathing, in-the-moment focus, mental imagery, anatomical and proprioceptive awareness, and deliberate slow playing.

Results: Musicians showed progress in rehabilitation after a few sessions; however, we did not test long-term effects. Sustained and permanent behavioral change will require an extended period of second-person neurophenomenological retraining to increase awareness and proprioception.

Conclusion: Gradual retraining process of mindfulness mind-body experience-based neurophenomenological process is an appropriate model to teach our non-dystonic pre-professional collegiate musicians in a hurried and quantity-oriented modern environment. A third-person external feedback device and brain image monitoring can add to the efficacy of the neurophenomenology.

Neuropsychological Assessment of Higher Cortical Functions: An Early Interventional Approach to Prevent Mild Cognitive Impairment in The Mexican Population

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Abstract

Non-communicable diseases (NCDs) account for a 13.42% mortality rate in Mexico and require complex, long-term care. As life expectancy rises and fertility declines, the population is aging, increasing the prevalence of neurological disorders such as Alzheimer's, Parkinson's, and other dementias. Dementia cases are expected to double every 20 years, with significant economic and social burdens, especially for low-income populations who face higher risk and reduced access to specialized care. Primary care centers often lack the capacity to diagnose or treat neurological disorders adequately, and the shortage of neuroscience specialists limits early detection. This increases the likelihood of executive function impairment, particularly among individuals with a family history of cognitive decline. NCDs disproportionately affect people living in poverty, rural areas, or with limited access to health services. This project aims to provide early neurological treatment to vulnerable populations and deliver preventive, specialized first-level care to reduce treatment abandonment. It also seeks to collaborate with the Regular State Educational System to identify students with special educational needs and prevent future neurocognitive disorders. Using government databases, school surveys, and Comprehensive Mental Health Center estimates, projections indicate that by 2025 the mid-zone of

San Luis Potosí will have 16,025 people with neurological disorders, requiring approximately 1,60,250 specialty consultations and benefiting up to 64,100 individuals. Strengthening donor support, intersectoral collaboration, and community-based rehabilitation is essential. Early neuropsychological assessment improves diagnostic accuracy, enables timely intervention, and can prevent or reverse mild cognitive impairment.

Euglycemia as a Prerequisite for Optimal Antioxidant and Anti-neuroinflammatory Treatment with Resveratrol in the Prevention of Neurodegenerative Diseases – Rationale from an *In Vitro* Study

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Abstract

The rising prevalence of impaired glucose tolerance and diabetes is primarily due to the global pandemics of obesity and physical inactivity. Furthermore, abnormal glucose control is accompanied by mitochondrial dysfunction, chronic inflammation and oxidative stress, increasing the risk of neurodegenerative diseases. One of the most studied natural anti-diabetics, anti-inflammatory, and neuroprotective compounds is resveratrol (RSV), a polyphenol. RSV activates the enzyme adenosine monophosphate-activated protein kinase (AMPK), which is a key energy sensor in the cell. Activating AMPK can then lead to the activation of sirtuin 1 (SIRT1), an enzyme associated with longevity and cellular health. The aim of the study was to assess the effect of glycemia on the concentration of selected cytokines (Interleukin (IL)-10, IL-12, IL-17A, tumor necrosis factor alpha, granulocyte-macrophage colony-stimulating factor) and SIRT1 production by astrocytes in conditions of lipopolysaccharide (LPS)-induced neuroinflammation and on the efficacy of subsequent RSV therapy. An *in vitro* model of the blood-brain barrier was formed from endothelial cells and astrocytes, divided by a semi-permeable membrane, which represented the microvascular (MC) and brain compartments (BC), respectively. RSV decreased the level of all cytokines in the BC after 24 h following its administration for all glucose concentrations in the MC ($p < 0.02$) The greatest decline was observed at glucose concentration of 90 mg/dL, corresponding to euglycemia ($p < 0.05$), compared to both hypoglycemia (40 mg/dL) and hyperglycemia (450 mg/dL). Moreover, RSV partially counterbalanced the inhibitory effect of LPS on SIRT1 secretion, acting most strongly in euglycemia. Our results may indicate that ensuring proper glycemic control is crucial for the maximum neuroprotective activity of RSV.

From Digital Dissociative Symptoms to Resilience: A Neuroscience-informed Case Study of Prefrontal Attunement and Recalibration Through Connectedness

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Abstract

Youth with autism spectrum disorder and social anxiety may engage in digital escapism, heightening risk for digital dissociative symptoms (e.g., time loss, depersonalization, derealization, and shutdown episodes) and functional impairments. Evidence-based interventions directly targeting these mechanisms and adapted to neurocognitive-behavioral profiles remain limited. This presentation introduces the socioecological prefrontal attunement and recalibration through connectedness (SePARC) intervention grounded in Dixon's Appraisal-by-Content model. This intervention targets eight prefrontal subregions via structured co-regulation routines, cue and environment redesign, and networked supports. We report a 16-week single case application with a 14-year-old youth on the autism spectrum (Level 1 support needs) and comorbid social anxiety, trauma history, and digital dissociative symptoms. We outline neurocognitive-behavioral profile assessment tools, intervention planning, scaffold design, session structure, and progress monitoring procedures to support replication and training. Improvements were observed on 95% of clinical indicators (i.e., emotion regulation, sensory awareness, social connectedness, academic participation, digital dissociative symptoms). Observed changes are consistent with movement toward homeostatic balance across prefrontal subregions and reduced allostatic load. SePARC leverages connectedness, felt safety, and belonging to increase resilience. The model operationalizes Dixon's ABC theory into clinical practice for biobehavioral health professionals, aligning person-centered goals, socioecological scaffolds, and neurocognitive skills across sessions and settings. While findings are limited by a single case design, they provide proof of concept and a rationale for larger, controlled studies to examine efficacy, reliability, validity, and generalizability. Future research should integrate electroencephalography and functional magnetic resonance imaging data to test and refine cognitive-behavioral strategies and to confirm prefrontal attunement/recalibration.

Utilizing Fuzzy C-means Clustering to Identify Emotional Subtypes of Psychosis

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Abstract

Emotional processing and recognition deficits are well-established and socially isolating symptoms associated with psychotic disorders. However, previous clustering efforts have fallen short in distinguishing emotional deficits across specific diagnoses. This study utilized electroencephalography (EEG) and emotional scene rating data from 389 adults with a psychotic disorder and 129 healthy controls. Principal component analysis was performed on EEG and valence-arousal self-ratings data to create a matrix of principal components. This matrix was used as input for fuzzy C-means clustering, which produced 3 distinct clusters. One way analysis of variance testing and multiple regression analysis were used to assess which clinical features characterize the emotional subgroups. The three clusters identified were roughly those with heightened, dampened, and unchanged EEG event-related potential (ERP) compared to health controls. The most significant determinant of clustering was mid-latency ERPs ($p < 0.01$), social functioning ($p = 0.01$), socioeconomic status (Hollingshead $p = 0.01$), general functioning (Global assessment of functioning $p = 0.02$), and impulsivity (Barratt impulsiveness scale $p = 0.02$). While there was significant overlap of emotional clusters to diagnosis and biotypes, the strongest associations were with cognition and other social and behavior features. This suggests that emotional deficits among individuals with psychosis could be more heavily influenced by behavioral and environmental factors, rather than diagnostically related symptoms. Understanding these differences could help clinicians to better develop treatment plans, such as social cognition training therapies.

From Foramen Magnum to Sacrum: Clinical Characteristics of Holospinal Epidural Abscesses

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Abstract

Background holospinal epidural abscess (HSEA) is a rare form of spinal epidural abscess (SEA) characterized by infection involving the epidural space across all spinal segments, accounting for approximately 1% of SEAs. Given its limited characterization in the literature, this study aimed to synthesize available evidence on its clinical features. Methods sixty-seven studies met inclusion criteria based on the reported extent of spinal involvement. Extracted variables included demographics, comorbidities, presenting symptoms, treatment strategies, and clinical outcomes assessed using the modified Frankel score (mFS). Results fifty-seven cases (52 male; median age 51 years) were analyzed. The median number of affected spinal levels was 23 (Interquartile range (IQR): 5). Among patients with available data, 60% (28/48) presented with the classical triad of fever, back pain, and neurological deficit. At a median (IQR) follow-up of 20 (44) weeks, 57% recovered without neurological deficit (mFS 1), 20% had ambulatory paraparesis (mFS 2), 16% had non-ambulatory paraparesis (mFS 3), and 7% had paraplegia (mFS 4 – 5). Two deaths were attributed to HSEA. Compared with patients with poor outcomes (mFS > 1), those with good outcomes (mFS = 1) had significantly lower rates of bladder motor involvement ($p = 0.01$), bowel motor involvement ($p = 0.03$), and C-reactive protein levels ($p = 0.01$). Conclusion HSEA represents an aggressive SEA subtype with a markedly higher prevalence of the classical triad (~60% vs ~10% in typical SEA). Despite severe presentations, most patients achieved favorable outcomes and mortality was low. Poor outcomes were associated with bladder and bowel dysfunction at presentation.

Multiple Intracranial Hemorrhages with Venous Stagnation on Cerebral Angiogram in Neurosarcoidosis

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Abstract

This is an interesting case of a 22-year-old male with a past medical history of intellectual disability and seizures. About one year after his first seizure at age 21, the patient presented to our institute for evaluation of his second lifetime seizure and was found to have an acute left frontal intracranial hemorrhage (ICH) on a head computed tomography (CT). A cerebral angiogram showed venous stasis but was otherwise unremarkable. A follow-up head CT one month later showed a new right frontal ICH for which he was admitted. During this hospital stay, the patient developed new hemorrhages in the left cortical frontal area and in the right subcortical frontal area. He underwent an extensive workup, including rheumatologic, hematologic, and neurologic evaluation. A repeat cerebral angiogram showed significant stagnation of contrast at the late venous phase with persistent contrast opacification indicating venous congestion. Ultimately, a brain biopsy was performed, which showed non-necrotizing granulomatous inflammation consistent with neurosarcoidosis. ICH in the setting of neurosarcoidosis is extremely rare and can progress rapidly to multiple acute hemorrhages, as exhibited by our patients. Diagnosis requires an extensive work-up to identify the underlying disease process, allowing patients to receive appropriate treatment and halt further disease progression. The proposed mechanism of pathology in our patient was the meningovascular infiltration by non-caseating granulomas, which led to venous congestion and stasis, an increase in venous pressure, and ultimately hemorrhage.

Neurobiological Mechanisms Underlying Mood-motor Interactions in Parkinson's Disease: A Scoping Review

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Abstract

Background: Parkinson disease (PD) affects more than 10 million people worldwide and is characterized by progressive dopaminergic neuron loss producing tremors, rigidity, bradykinesia, and gait disturbance. Individuals with PD frequently experience mood disturbances including depression, anxiety, and apathy, which may precede motor manifestations and substantially reduce quality of life. Despite recognized mood–motor comorbidity, the neurobiological mechanisms linking affective and motor symptoms remain incompletely characterized.

Objective: To map neurobiological mechanisms tested against both validated mood and validated motor outcomes within the same PD cohort, and to identify structural gaps constraining the field.

Methods: This scoping review follows Joanna Briggs Institute methodology and PRISMA-ScR reporting. The protocol was registered on the Open Science Framework. PubMed, Embase, PsycINFO, Web of Science, and Cochrane Library were searched from January 2000 to December 2025. Studies were included only if they directly measured a neurobiological mechanism against both a validated mood scale and a validated motor scale in the same cohort (triple-intersection criterion). Records were screened in Rayyan by two reviewers; conflicts resolved by consensus. Extraction included study characteristics, mood and motor assessments, mechanism domain, and reported associations.

Preliminary Results: Of 5,221 records screened, 129 studies met inclusion criteria (2001 - 2025; 64% post 2020; cross-sectional case-control 63%, longitudinal 30%; median cohort size 55). Studies spanned 15 mechanism domains, with five accounting for approximately 70% of the corpus: monoaminergic imaging (n = 33), peripheral biomarkers (n = 17), DBS/lesion intervention (n = 15), RS-fMRI/functional connectivity (n = 13), and electrophysiology (n = 13). Coverage was depression-anchored, with apathy fragmented across multiple instruments (n = 48) and reward/anhedonia scattered (n = 12). Cohorts derived predominantly from China (45%), USA (12%), and UK (12%). All figures are preliminary pending second-reviewer verification of data extraction.

Conclusion: This preliminary synthesis identifies depression-centric coverage, apathy instrument fragmentation, and geographic narrowness as structural constraints on the field, and points to substrate dissociation across imaging modalities as a candidate organizing principle. Findings may inform future integrated treatment strategies.

Oligodendrocyte Maturation Factor Drives Myelination in Mouse Anterior Commissure

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Abstract

The oligodendrocyte maturation factor (OMF) gene is located on mouse chromosome 17. A mutational analysis identified a 59-base pair long deletion that occurred in the first intron of the OMF gene that caused developmental neurological abnormalities. Homozygous OMF null mice were born healthy and appeared anatomically normal up to two weeks of age. However, after postnatal day 14 (P14), they were smaller than normal, exhibited ataxic gait, became lethargic, and died shortly after P24. Transcriptome analysis at P14 and P23 revealed significantly reduced expression of mitochondrial genes in OMF null brains compared to wild-type (WT) controls, including mt-Nd4, mt-Cytb, mt-Nd2, mt-Co1, mt-Atp6, and others. Similarly, genes specific for myelinating oligodendrocytes also showed reduced expression in P23 OMF null brains compared to controls. OMF:CNP:EGFP mice demonstrated significantly impaired myelination and widespread vacuolation. As such, our hypothesis is that OMF defects reduce mitochondrial energy metabolism, which disrupts oligodendrocyte maturation, leading to impaired myelination and neurological abnormalities. Neurofilament heavy chain (NF-H) staining in OMF null anterior commissure showed looser, less organized NF-H–positive axon bundles, with occasional swellings or spheroid-like structures along the tract. Oligodendrocyte lineage analysis revealed a significant reduction in OLIG2-positive cells (a marker for oligodendrocytes) in OMF null mice, with abnormal distribution compared to the aligned organization seen in WT counterparts. At the ultrastructural level, OMF-deficient animals showed defects in myelin membranes such as decompaction. Examination of nodes of Ranvier demonstrated a reduced number and shortened spacing, indicating that OMF deletion disrupts nodes. Together, these findings identify OMF as a critical factor for oligodendrocyte maturation, myelin stability, and nodal organization, while also implicating mitochondrial dysfunction in OMF deficiency. Understanding OMF's role may reveal new insights into the molecular mechanisms underlying myelin-related disorders and highlights potential therapeutic options targeting myelin integrity.

Development of a Novel Self-report Tool to Aid in the Assessment of Autism in Adulthood: The Autism Spectrum Trait Scale

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Abstract

Background: The current state of formal autism assessment poses a significant ethical concern, as systemic barriers make evaluations increasingly difficult to obtain, particularly in adulthood. Self-report scales can be used to help identify the possible presence of autism. However, no self-report measure has evaluated autistic traits in adults while encompassing the most recent diagnostic criteria and meeting the highest standards of psychometric scale development. Therefore, the autism spectrum trait scale (ASTS) was developed to establish a new self-report measure that addresses limitations in existing instruments.

Methods: Exploratory factor analysis (n = 764) determined the factor structure, and confirmatory factor analysis (n = 761) was performed in a non-overlapping sample to assess the model's fit. Additional analyses were conducted to further evaluate the scale's psychometric properties and examine group differences.

Results: The ASTS had four factors and good model fit, strong internal consistency, and criterion validity. Metric measurement invariance, acceptable sensitivity and specificity, and the presence of a superordinate factor were also established.

Discussion/Conclusions: The findings provide preliminary support for the ASTS as an effective assessment tool for autism in adults. The study included both self-identifying and clinically diagnosed autistic adults, and analyses examining group differences yielded valuable insights. Clinical implications, including the use of the ASTS in diagnostic contexts, neuropsychological assessments, and research, as well as future directions for evaluating its utility, will be discussed.

Association of Deep Phenotypical Features with Severity of Autism in Children

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Abstract

Background: Autism spectrum disorder (ASD) is a very complex disorder characterized by deficits in social communication, social interaction along with the presence of repetitive behaviors, interests, and attitudes. Several studies have indicated that patients with autism have higher rates of minor physical anomalies (MPAs) and that MPAs may serve as a diagnostic tool. The autism dysmorphology measure (ADM) is used to assess dysmorphology in ASD and it measures the physical structure of 12 body parts. Our study aimed to find out the deep phenotypical features in children with ASD in a tertiary care hospital that would help pediatricians, who were not extensively trained in dysmorphology to apply ADM scores on ASD children.

Objective: To identify deep phenotypical features in children with ASD and their association with the severity of the disease.

Methods: This was a cross-sectional study conducted at the Department of Developmental-Behavioral Pediatrics, at University of Child Health & The Children's Hospital, Lahore. Study duration was 6 months (July 2021 - December 2021). A total of 100 children with ASD between the ages of 3 - 18 years were collected through purposive sampling. 12 types of measurements were taken and based on ADM score, participants were grouped into dysmorphism and non-dysmorphism. Demographic questionnaire and scores of childhood autism rating scale 2 were noted.

Results: Based on ADM scoring, 91 children were non-dysmorphic and 9 were dysmorphic. Out of 9 dysmorphic children, 66% (n = 6) children had mild to moderate severity of autism and 33% (n = 3) had mild autism. However, out of 91 non-dysmorphic children, 38.4% (n = 35) had mild autism, 56% (n = 51) mild-moderate autism and 5.4% (n = 5) severe autism. Results showed that 8 dysmorphic children were males and 1 was female while 72 were non-dysmorphic male and 19 were non-dysmorphic females.

Discussion/Conclusion: Dysmorphism on the basis of ADM scoring was found in small number/some of children with ASD and is associated with mild to moderate severity of disease.

Investigating the Effects of Two Computerized Interventions on Cognitive and Emotional Measures in Children with Autism

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Abstract

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social and communication atypicalities and restricted and repetitive behaviors. Although deficient attention is not a core component of ASD, accumulating evidence suggests that it plays a major role. Previous cognitive training studies demonstrated encouraging results, yet methodological issues limit our understanding. This study is an ongoing multi-arm study aimed at assessing shared and unique effects of two computerized interventions in children with ASD: computerized progressive attention training (CPAT) and intervention of joint attention and communication (iJAC). Assessment occurs at 4 time points (T1 - T4), where children complete cognitive tasks (e.g., Go/No-Go, emotion recognition) while parents complete questionnaires assessing ASD, attention-deficit/hyperactivity disorder, and anxiety symptoms. Thus far, twenty-four autistic children (6 - 10 year old) participated in 12 intervention sessions, with 11 assigned to the CPAT protocol, and 13 to the iJAC protocol. Preliminary comparisons of pre (T1) and post-test (T2) assessments showed a significant improvement in response inhibition in the CPAT group and a tendency of improvement in the iJAC group. A significant interaction between time and group on response latencies revealed that CPAT participants slowed down their responses in the Go/No-Go task whereas iJAC participants speeded up their responses. Interestingly, there was a significant interaction between group and time on anxiety, indicating a reduction in anxiety following CPAT but not iJAC. Further analyses are required to explore the consistency of these effects and to identify predictors of the various interventions' outcomes, which may lead to tailored-made interventions for children with ASD.

How Parents of Autistic Children Deal with Their Child's Challenging Behavior: The Role of Parents' Explicit and Implicit Attitudes Toward Autism Spectrum Disorder

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Abstract

Objectives: This research investigated the influence of explicit and implicit attitudes toward autism spectrum disorder (ASD) on how parents of autistic children address their child's challenging behavior.

Methods: The implicit association test and online survey were conducted with 106 parents of autistic children. Data was analyzed using the Statistical Package for the Social Sciences Process Macro model 1.

Results: Implicit attitudes toward ASD, rather than explicit attitudes, moderate the relationship between the child's challenging behavior and parenting behaviors. Specifically, when the level of negative implicit attitudes toward ASD was low, the higher level of child's challenging behavior was significantly associated with more warmth-acceptance behavior and less rejection-restriction behavior. The reverse pattern was observed when the parents' negative implicit attitudes were strong.

Conclusions: This study highlights the importance of implicit attitudes among parents of autistic children. The practical implications of these findings for interventions aimed at enhancing parenting behaviors in parents of autistic children are discussed.

Ischemic Stroke Despite Anticoagulation

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Abstract

Patients with atrial fibrillation who have ischemic stroke despite taking oral anticoagulation therapy (OAT) have a high risk of recurrence. The mechanism is not known; therefore, prevention and treatment are difficult. Direct oral anticoagulants have been considered to be the best to prevent ischemic stroke; however, perfect prevention is impossible. It was considered that recurrence of ischemic stroke occurs due to inappropriate treatment dose of OAT, but it occurs even with appropriate dose. As 90% of the emboli are considered to be derived from left atrial appendage (LAA), LAA closure is often times performed to prevent ischemic stroke. Recently, an implant device to close LAA was invented to prevent ischemic stroke; however, it cannot completely prevent ischemic stroke. We will discuss the possible mechanisms of recurrent ischemic stroke despite taking OAT, reviewing literature, and sharing our experiences.

Long-term Outcomes of Paired Vagus Nerve Stimulation for Motor Rehabilitation After Stroke

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Abstract

Chronic upper extremity (UE) motor impairment is common after ischemic stroke and has been shown to negatively impact independence and quality of life. Paired vagus nerve stimulation (VNS) is a promising Food and Drug Administration-approved treatment for chronic moderate to severe UE impairments following ischemic stroke. Paired VNS combines pulses of VNS with intense, task-specific rehabilitation to drive improved motor recovery and function. In this presentation, we will discuss the long-term outcomes of the pivotal VNS-REHAB trial, including pooled 1 year results from 74 participants, along with secondary analyses and emerging evidence regarding additional applications of paired VNS. Participants in the VNS-REHAB trial received 18 sessions (three times weekly for six weeks) of in-clinic therapy, followed by three months of home exercise with

either sham VNS (control) or active VNS. Control participants then crossed over to receive 18 additional sessions of in-clinic therapy with active VNS. All participants were instructed to complete home-based exercises with self-activated VNS for 1 year. The primary outcome measure was the Fugl-Meyer Assessment – UE, and secondary measures included the Wolf Motor Function Test, Motor Activity Log, and Stroke Impact Scale. Pooled results showed improvements in motor impairment, function, and patient-reported outcomes following 1 year of paired VNS rehabilitation as compared with baseline. These findings highlight the durable, long-term benefit of paired VNS on UE motor recovery.

Assembly of Amyloid Beta in Aggregates, Remodeling of the Membranes and Dysregulation of Cholesterol Homeostasis are the Major Pathological Features Aβ for the Initiation and Progress of Alzheimer's Disease

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Abstract

The aggregation of amyloid beta (Aβ) is a self-assembly process that results in the production of fibrillar structures along with neurotoxic aggregates. However, our recent studies revealed a few more pathological features of Aβ. Based on these studies, we proposed a model in which the interactions of Aβ monomers membranes is a key step of transition of Aβ from physiology to pathology. Our studies revealed that the interaction of Aβ with membranes enables this protein to self-assemble in aggregates at physiological low nanomolar concentration. We discovered a novel feature of Aβ42 monomers, namely its capability to extract cholesterol from membranes. Extracted cholesterol catalyzes the assembly of Aβ42 in aggregates, which is the mechanism by which physiologically important Aβ42 monomers are converted into their pathological form. Additionally, release of cholesterol from membranes leads to its accumulation in the brain, which is one of the risk factors associated with disease development and progression. Finally, cholesterol depletion decreases membrane stiffness, which can result in deterioration of the function of numerous membrane-bound proteins resulted in such an evet as a synapse loss, a common pathological feature of Alzheimer's disease.

Descriptive Study of 32 Cases of Alzheimer's Disease in Senegal

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Abstract

Objectives: The main objective of the study was to describe the epidemiological, diagnostic, and therapeutic characteristics of patients with Alzheimer's disease seen at the Geriatrics and Gerontology Department of the Fann National University Hospital in Dakar, in order to improve knowledge and accurate, early diagnosis of the disease in Senegal and Africa.

Method: This was a prospective, descriptive study covering a 10 month period (December 1, 2022, to September 30, 2023), involving a population of people aged 60 and over seen in geriatric consultations. Cognitive assessment was performed using the Senegal test, and epidemiological, diagnostic and therapeutic characteristics were collected and analyzed using Epi Info 7 Software.

Results: Out of 695 patients seen in consultation, 32 presented with Alzheimer's disease, i.e., a prevalence of 4.6%. The average age of our patients was 77 ± 8 years, with extremes ranging from 60 to 92 years. The 75 - 79 age group (31.25%) was the largest. A predominance of females (68.75%) was noted, with a sex ratio M/F of 0.45. The main antecedents and comorbidities were arterial hypertension (62.65%), diabetes (34.38%) and falls (15.63%). Suspicion of familial dementia was found in 17.25% of cases. The main reason for consultation was memory impairment (100%). Thirteen (40.6%) patients had a disease duration of 6 months to 1 year. Cognitive disorders according to the Senegal test appeared severe for 7 patients (21.87%), moderate for 4 patients (12.5%) and mild for 21 patients (65.63%). Geriatric syndromes were dominated by frailty (56.25%) and loss of functional independence (43.75%). None of our patients were able to undergo tau, phosphorylated tau and amyloid protein assays. The most common biological abnormalities were anemia (89.28%) and hypoalbuminemia (81.28%). Brain magnetic resonance imaging on all our patients showed hippocampal atrophy (100%). All our patients received anti-cholinesterase treatment with donepezil or antiglutaminergic treatment using memantine. 4 patients were treated with melatonin, 4 with risperidone and 4 with selective serotonin reuptake inhibitors.

Conclusion: The incidence of Alzheimer's disease is rising steadily as a result of longer life expectancy in our regions. Diagnosis is often made at a late stage. This work could enable practitioners in developing countries to envisage Alzheimer's disease in the event of suggestive symptoms, in order to establish an early diagnosis and treatment.

What is the Big Picture in Alzheimer's Disease?

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Abstract

Like the dots in a pointillist painting, we know many facts about Alzheimer's disease causes, biology, pathology, and clinical manifestations. Unfortunately, unlike the pointillist painting, when we view all these facts together, no complete, "big picture" emerges. However, a group of scientists compiled data from over 130,000 papers describing gene expression in Alzheimer's followed by pathway analyses. Their analyses showed that gene expression changes in Alzheimer's involved 91% of the known Kyoto encyclopedia of genes and genomes pathways and accounted for the facts known about Alzheimer's. So, massive change in gene expression becomes the "big picture" in Alzheimer's disease. This then raises the question of what the upstream cause of such massive change in gene expression may be. Little known stress granules provide a potential upstream mechanism for these changes in gene expression. Stress granules form in response to cell stresses and a number of risk factors for Alzheimer's and have been described in the cytoplasm of Alzheimer's cells, as well as other neurodegenerative diseases and cancer. They typically sequester more than 1,000 proteins and RNA species that serve a variety of normal functions within the cell, including the expression of specific genes. By sequestering a panel of selected RNA species, for example, they control the expression of large numbers of genes – as required by the big picture of massive change in gene expression in Alzheimer's disease. This leads to the following sequence of events:

Risk factors > stress granules > massive, altered gene expression > biological and clinical Alzheimer's disease.

Approach to Masses in the Head and Neck Spaces

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Abstract

The neck is anatomically complex but can be organized into compartments or spaces, in order to organize an approach to neck masses. The spaces of the head and neck are based on compartments defined by the layers of the deep cervical fascia. Each space contains specific content and understanding the anatomy is critical in determining underlying pathology. These spaces can be grouped as suprahyoid or infrahyoid, with some spaces, like the carotid and retropharyngeal spaces, spanning both levels. Displacement of parapharyngeal fat can help distinguish the site of origin of a suprahyoid mass. Visceral space is the only space contained entirely in the infrahyoid neck and contains the thyroid and parathyroid glands, as well as portions of the aerodigestive tract below the oropharynx. The spaces of the head and neck have common and uncommon pathology, often based on the structures confined within it. Therefore, once a lesion is localized to the correct anatomical space, the radiologist can utilize imaging features and clinical information to provide a tailored list of differential considerations, or in some cases, a specific most likely diagnosis. Common pathologies within the spaces of the head and neck will be reviewed to help guide the next steps in patient management.

Neurologic Implications of Glucagon-like Peptide-1 Receptor Agonists

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Abstract

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are increasingly common medications used in the management of type 2 diabetes mellitus and obesity. These medications exert numerous systemic metabolic effects. They also impact the nervous system in ways independent of blood glucose concentrations, including mitigation of neurologic injury through reductions in neuroinflammation, neuroapoptosis, and oxidative damage. GLP-1 RAs have been studied in multiple neurological disease states, including Alzheimer's disease, Parkinson's disease, and stroke. Their potential benefits include both symptom improvement and disease modulation. The objectives of this presentation are to review the physiology of GLP-1, examine the impact of GLP-1 receptor agonists on the neurologic system, and discuss current clinical evidence evaluating the effects of GLP-1 RAs on several neurological disease states.

The Brain-heart Connection: Value of Concurrent Electrocardiogram and Electroencephalogram Monitoring in Epilepsy Management

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Abstract

Simultaneous electrocardiogram (ECG) and electroencephalogram (EEG) monitoring, both during and between seizures, offers significant value in epilepsy management. Concurrent recording improves diagnostic accuracy by distinguishing epileptic from cardiac events, detecting cardiac abnormalities, and identifying autonomic dysfunction. It also plays a key role in understanding the mechanisms behind cardiac morbidity and sudden cardiac death in epilepsy, and in guiding treatment decisions. The "Epileptic Heart Syndrome" results from chronic epilepsy, whereby recurrent seizures may alter cardiac structure and function, increasing the risk of interictal arrhythmias, morbidity, and mortality. Thus, standardized protocols that incorporate routine ECG-EEG monitoring in epilepsy monitoring units to enhance care, improve outcomes, and reduce epilepsy-related cardiac risks are needed.

Sex-dependent Effects of Ashwagandha Root (*Withania somnifera*) Extract and Ubisol-Q10 on White Matter Integrity and Neuronal Structure in a 5XFAD Mouse Model of Alzheimer's Disease

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Abstract

Sex differences play an important role in the progression and treatment of Alzheimer's disease (AD) but are often overlooked in preclinical therapeutic studies. This study examined sex-specific effects of treatment with a "Tonic" combination solution of ethanolic ashwagandha root (*Withania somnifera*) extract and water-soluble coenzyme-Q10, "Ubisol-Q10," in 5Xfamilial AD (5XFAD) transgenic mouse model of AD. In the current study, 24 male and 24 female mice were divided into three groups: 5XFAD treated with Tonic (n = 8 male + 8 female), 5XFAD untreated (n = 8 male + 8 female), and wild-type untreated (n = 8 male + 8 female). All mice underwent three repetitions of structural magnetic resonance imaging (MRI) over their lifetime of 11 months. White matter regions of the brains were assessed for volumetric changes from early-, middle-, and late-stage degeneration (3, 6, and 9 months of age, respectively). Post-mortem Golgi-Cox and Nissl stains were used to assess dendritic morphology and cell density to corroborate MRI findings. These analyses aim to determine whether combined treatment influences white matter integrity and neuronal structural complexity, and whether responses differ between male and female mice. A secondary aim is to evaluate MRI-identified white-matter structural changes as a potential focal point for early-diagnostic study. This work provides insight into the role of sex in therapeutic responses targeting neurodegeneration and may help inform future treatment strategies and diagnostic methods for AD.

Simultaneous Craniotomy for Resection and Placement of Responsive Neurostimulation for Treatment of Medically Refractory Epilepsy: Illustrative Cases

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Abstract

In cases of medically refractory epilepsy that is multifocal or involving eloquent cortex, a combined resection and neuromodulatory approach may offer greater seizure reduction than resection alone. Here we describe indications, technical considerations, and outcomes of patients in whom resection and responsive neuromodulation (RNS) implantation was performed in the same

operation. Three patients met inclusion criteria for this study. Pre-operative stereo-electroencephalography demonstrated multifocal or eloquent cortex-involving seizure onset zones in all patients. Two patients were implanted with one depth electrode in the contralateral hippocampus and a cortical strip electrode ipsilateral to the resection. One patient received bilateral hippocampal depth electrodes and resection. Technical considerations include prioritization of stereotactic accuracy, avoidance of pulse generator discharge, and distant implantation of the generator relative to resection site. At 1 year, two patients achieved Engel IIIA, and one IIB. We suggest consideration of this combined approach of simultaneous RNS implantation and craniotomy for resection when intracranial data indicates a role for resection but is unlikely to achieve seizure freedom alone.

A Novel Approach to Gait Rehabilitation: Cortical Priming for Better Step Initiation in Chronic Stroke

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Abstract

Evidence suggests that transcranial direct current stimulation (tDCS) can enhance motor performance and learning of hand tasks in persons with chronic stroke (PCS). However, the effects of tDCS on the locomotor tasks in PCS are unclear. This pilot study aimed to: (1) determine aggregate effects of anodal tDCS combined with visuomotor step training (VST) on improvements of the neural and biomechanical attributes of stepping initiation in a small cohort of PCS over a 4-week training program (3 sessions x 4 weeks); and (2) assess the feasibility and efficacy of this novel approach for improving voluntary stepping initiation in PCS. The step initiation (SI) tests at pretraining, post-training, 1-week and 1-month follow-ups. The primary variables were baselining vertical ground reaction force (B-vGRF), response time (RT) to initiate anticipatory postural adjustment, and the retention of B-vGRF and RT. The preliminary findings suggest a 4-week VST results in improved paretic limb weight bearing during SI in PCS. Furthermore, VST combined with a-tDCS may lead to better retention of gait improvements.

Prenatal Maternal Stress and the Broad Autism Phenotype in Adolescents: The Mediating Role of DNA Methylation

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Abstract

Prenatal maternal stress (PNMS) has been linked to an increased risk of autism spectrum disorders (ASD), though the underlying mechanisms remain unclear. Given that both prenatal stress and ASD-related traits have been associated with DNA methylation, this study examined whether epigenetic mechanisms mediate the pathway from PNMS to autistic-like outcomes in adolescence. Using data from the longitudinal study of the 1998 Quebec Ice Storm, we examined PNMS, including objective hardship, subjective distress, and cognitive appraisal of the disaster, in relation to DNA methylation measured from children's blood samples at age 13, and autistic-like traits assessed at ages 15, 16, and 19. We also investigated the mediating role of DNA methylation in the association between PNMS and children's autistic-like traits. Results showed that greater maternal objective hardship and more negative cognitive appraisal were associated with DNA methylation differences at age 13, which in turn predicted higher scores on aloof personality and pragmatic language deficits, but not on rigid personality. Mediation effects were observed in genes involved in the PI3K/AKT/mTOR pathway, previously linked to neurodevelopmental disorders. Interestingly, although higher PNMS predicted more severe ASD-like traits, the mediation effects of DNA methylation appeared to buffer this association, suggesting a potential protective or adaptive role of epigenetic modifications.

The Convergence of Autism Spectrum Disorder and Intimate Partner Violence: Neurobiological Implications for Biobehavioral Intervention Development

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Abstract

Trauma-specific care denotes best practice when working with individuals with histories of childhood abuse, maltreatment, or intimate partner violence. Youth with autism spectrum disorder and exposure to intimate partner violence form a subgroup of trauma-impacted individuals, posing specific challenges for practitioners and researchers. The comorbidity of trauma and autism spectrum disorder requires an in-depth understanding of the complex interrelationship of childhood experiences of intimate partner violence, the associated traumatic sequelae, and the neurobiological characteristics of autism. Current research on the intersectionality of intimate partner violence, trauma, and autism is limited. This presentation draws attention to the implications of working with autistic adolescent clients with a history of trauma from childhood exposure to intimate partner violence. A case study is utilized to contextualize concerns, prioritize needs, and identify treatment outcomes. A systematized review is used to examine the literature on interventions for youth with autism spectrum disorder and trauma. The objective is to highlight the gap in literature and offer behavioral health clinicians a review of interventions available for this population. Recommendations include advancing socioecological prefrontal attunement and recalibration through connectedness (SePARC), a neuroscience-guided intervention model, aligning person-centered goals, socioecological scaffolds, and neurocognitive skills, targeting eight prefrontal subregions. In a 16-week single-case SePARC application, improvements were noted in 95% of indicators. To establish mechanistic validity and evidence base, trials should integrate electroencephalography and functional magnetic resonance imaging to test and refine cognitive and behavioral strategies and confirm prefrontal attunement/recalibration. Our findings highlight the need for specialized care and further research to meet the unique needs of this underserved population.

Innovative Biomarker Exploration in Autism Spectrum Disorder: Combining Graph Neural Networks and Permutation Testing on Functional Magnetic Resonance Imaging Data

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Abstract

This study employed graph neural networks (GNNs), specifically an unsupervised GNN, to extract node embeddings from brain regions in both autism spectrum disorder (ASD) and control groups. The objective was to identify potential biomarkers by analyzing node embeddings extracted from a graph model based on functional magnetic resonance imaging data. Permutation tests were conducted to identify regions with significant differences in their embeddings between the two groups. Our results revealed several regions exhibiting significant differences, including the cerebellum, temporal lobe, and occipital lobe. These findings align with previous studies on ASD. Moreover, novel regions such as Vermis 3, Vermis 4 5, Fusiform areas, Parietal, and Cuneus were identified, emphasizing the need for further investigation. This study underscores the potential of GNNs in analyzing brain networks for ASD biomarker discovery. The identified regions warrant additional validation and exploration to understand their association with specific domains of ASD symptoms. Our approach presents a promising avenue to advance the diagnosis of ASD and to improve our understanding of its underlying neural basis.

In Silico Identification of Alpha-synuclein Fibril Inhibitors through Large-scale Virtual Screening and Molecular Dynamics Simulations

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Abstract

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by the loss of nerve cells in the brain and currently has no known cure. Alpha-synuclein is a protein that plays a vital role in the progression of PD, as it misfolds and accumulates into clumps called Lewy bodies, the pathological hallmark of PD. Small molecules show promising potential for

inhibiting alpha-synuclein aggregation due to their ability to interact with the target protein and prevent the formation of toxic fibrils. This research aims to identify and optimize small-molecule inhibitors that can effectively bind to alpha-synuclein fibrils, thereby preventing their aggregation. In this study, we have employed virtual screening to identify six promising small-molecule inhibitors after screening 1,092 chemical compounds and filtering billions of compounds from the ZINC20 database. Chemical compounds were prioritized based on their potential binding affinity and number of interactions to the fibril, from which LVI showed the highest binding affinity. Furthermore, chemical alterations were also performed on the LVI to generate five variants (LVIM1-5) to optimize ligand binding properties. Molecular dynamics simulations confirmed that the ligand remained stably bound to the receptor throughout the simulation, as indicated by root mean square deviation and root mean square fluctuation analyses, which demonstrated structural stability. Finally, absorption, distribution, metabolism, excretion, toxicity properties of all ligands were computed to predict pharmacokinetics, which shows that LVI has the best count among the rest of the ligands. This research highlights the potential of virtual screening methods in identifying therapeutic small-molecule inhibitors that target alpha-synuclein fibrils in PD.

Scalp High-frequency Oscillations as Predictors of Seizure-free Duration in Pediatric Epilepsy

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Abstract

Objective: In epilepsy research, traditional electroencephalography (EEG) interpretation has focused on the Berger frequency band (1 - 25 Hz). However, recent advances in recording techniques have enabled analysis beyond this range. High-frequency oscillations (HFOs), defined as EEG activity above 80 Hz, have emerged as promising biomarkers of epileptogenicity. In scalp EEG, HFOs are more closely associated with seizures than spikes alone. Given the limitations of self-reported seizure-free durations, this study aims to determine the relationship between scalp-recorded HFOs and the time since the last reported seizure in patients with epilepsy.

Methods: We analyzed data from 169 pediatric epilepsy patients (91 males; age range: 0 - 20 years). A holdout validation approach was employed to develop and test a predictive model—the multivariate HFO model—designed to estimate the time since the last reported seizure.

Results: The multivariate HFO model incorporated four variables: scalp HFO detection rate, presence of developmental delay, epilepsy duration, and use of antiepileptic drugs. The area under the curve (AUC) of the multivariate HFO model was higher than that for the HFO and spike models in all four discriminations for time since the last reported seizure (≥ 2 years: AUC = 0.95, ≥ 1 year: 0.91, ≥ 2 months: 0.82, and ≥ 2 weeks: 0.76).

Conclusions: The multivariate HFO model showed the highest predictive accuracy in patients with longer seizure-free intervals (≥ 1 year).

Significance: This model provides an objective tool for assessing epilepsy remission, potentially guiding treatment decisions and long-term management.

Efficacy of Bortezomib, Cyclophosphamide, and Dexamethasone for Newly Diagnosed POEMS Syndrome Patients

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Abstract

POEMS syndrome is a rare plasma cell disorder. The preferred treatments vary according to the preference of the physician and anecdote. The best first-line treatment has not been established. We propose a new treatment mode for POEMS syndrome that uses the four-cycle treatment as the induction regimen, followed by sequential transplantation as the consolidation

regimen for transplantation-eligible patients, while another two-cycle treatment can be used as the consolidation regimen for transplantation-ineligible patients. The overall vascular endothelial growth factor (VEGF) response rate of this new treatment mode was 100%. The cumulative VEGF complete remission (CRV) rate was 67.9%, and the cumulative complete hematological response (CRH) rate was 55.6%. During the median 49-month follow-up, the 5-year-overall survival (OS) rate was 90.7%, the 3-year-progression-free survival (PFS) rate was 78.4%, and the 5-year-PFS rate was 73.8%. The bortezomib, cyclophosphamide, and dexamethasone (BCD) induction regimen achieved a 75% CRV rate (median time from diagnosis to CRV = 130 days) and 66.7% CRH rate (median time from diagnosis to CRH = 218 days). In addition, the VEGF response was less than the partial response rate after four-cycle induction treatment, which, together with a decrease on the overall neurological limitation scale of < 3 points 1 year after consolidation treatment, was an independent poor prognostic factor.

Conclusion: Bortezomib was well-tolerated by patients with POEMS syndrome. Compared with CD ± T regimen, BCD as the induction regimen achieved better VEGF response and earlier hematological remission. Autologous stem cell transplantation used as consolidation therapy further improved the neurological and hematological remission rates, resulting in better OS and PFS.

Intervention Study of Health Qigong Exercise on Autonomic Dysfunction in Patients with Mild to Moderate Parkinson's Disease

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Abstract

Background and Purpose: Autonomic nervous system dysfunction is one of the common non-motor symptoms of Parkinson's disease (PD), which can significantly reduce patients' quality of life. This study uses 12-week of health qigong exercise as an intervention to observe its impact on autonomic nervous symptoms in patients with mild to moderate PD.

Methods: 28 patients with PD were randomly divided into an experimental group and a control group, with 14 people in each group. The scale for outcomes in PD for autonomic symptoms was used to compare indicators related to autonomic symptoms before, during, and after the intervention. Statistical Package for the Social Sciences 23.0 was used to perform repeated measures analysis of variance to determine the differences between groups and within groups.

Results and Discussion: After 12 weeks of health qigong exercise, the total score of the experimental group showed a highly significant difference ($p < 0.01$), and the patients' autonomic nervous function was significantly improved. A highly significant difference ($p < 0.01$) was observed between the experimental group and the blank control group after 12 weeks.

Conclusion: 12 weeks of health qigong exercise can significantly improve the autonomic nervous function symptoms of patients with PD, specifically manifesting in notable improvements in symptoms related to the digestive system, urinary system, cardiovascular function, and body temperature regulation.

Health Qigong Exercise on Dual-task Function in Parkinson's Patients

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Abstract

Background and Purpose: Parkinson's patients frequently exhibit significant dual-task impairment, adversely affecting gait and daily activities. This study investigated the efficacy of health qigong exercise in ameliorating dual-task deficits in this population.

Methods: Thirty-two participants were randomized into an experimental group (n = 16) and a control group (n = 16). Both groups maintained standard pharmacological treatment. The experimental group received an additional 12-week of health qigong exercise intervention (3 sessions/week, 60 min/session). Dual-task performance was assessed pre- and post-intervention using a combined motor-cognitive task paradigm.

Results and Discussion: Post-intervention, the experimental group demonstrated highly significant improvements in dual-task gait speed ($p < 0.01$) and significant improvements in cognitive task accuracy ($p < 0.05$). The control group showed no statistically significant changes in these parameters ($p > 0.05$).

Conclusion: Health qigong exercise is a beneficial non-pharmacological intervention for enhancing dual-task capacity in individuals with Parkinson's disease, potentially reducing fall risk and improving functional independence.

Phobia, Depression, Obsessive Compulsive Disorder: Symptoms of One Disorder or Multiple Disorder?

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Abstract

A series of 5 patients who presented with different neurobehavioral symptoms like phobia, depression, obsessive compulsive disorder in 0 to 10 years underwent evaluation and was found to have one common deficiency that is vitamin D deficiency. These children were treated with vitamin D and recovered. No medications other than vitamins were given. These cases illustrate that a simple deficiency can lead to major neurobehavioral dysfunction and before starting any psychiatric medication, vitamin D should be checked and treated by keeping the vitamin D levels between 50 ng/l and 80 ng/l. Further retrospective study of all patients presenting with these symptoms is required to collect more evidence and significance.

GAS5-targeting Therapeutic as a Modulator of Neuroinflammation and Insulin Signaling in a Tauopathy Model

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Abstract

Long non-coding RNAs (lncRNAs) are critical regulators of gene expression and metabolism. We previously demonstrated that lncRNA growth arrest-specific transcript 5 (GAS5 (human)/GAS5 (mouse)) is decreased in an aged mouse model. We demonstrated the efficacy and specificity of NPC86, a GAS-targeting therapeutic designed in our lab. Next, we evaluated the PS19 transgenic mouse model which expresses the human tau mutant form P301S. Our results demonstrate a markedly low level of GAS5 compared to non-transgenic controls in male and female PS19 mice. Intranasal administration of NPC86 to PS19 mice increased GAS5 levels in the brain demonstrated by RNA-FISH. There was no toxicity and the body weight did not change with the treatment. Results show increased improved insulin signaling shown by increased pAKT concurrent with decrease in inflammatory markers in response to NPC86 treatment. In summary, we demonstrate the yet unknown role of the lncRNA GAS5 in a tauopathy model and demonstrate that GAS5 is a druggable target for preventing dementias.

Implementation of Virtual Reality to Enhance Psychological Well-being in Home Based-primary Care Veterans with Dementia

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Abstract

Background and problem/clinical inquiry: As the population ages, persons with dementia (PwD) grow. PwD experience behavioral and psychiatric symptoms (BPS) which can impact their quality of life (QOL) and sense of well-being. Fatigue, fear, and misperceptions can lead to BPS. Veterans with dementia enrolled in home-based primary care (HBPC) require in-home support due to multiple chronic diseases affecting their daily activities and health.

Significance of the issue and/or baseline data: Treatment of BPS in PwD includes using medications to help stabilize moods and decrease irritability and agitation. However, drugs can have untoward effects on older adults. More non-medication interventions are needed for PwD.

Population (or patient/problem), intervention, comparison, outcome (time optional): In HBPC veterans with dementia do virtual reality (VR) enhance psychological well-being after receiving three VR sessions?

Narrative synthesis of the evidence: The QOL of PwD is linked to psychological factors such as mood and cognition. QOL scores for PwD decline with higher symptoms of depression. Psychological well-being, self-esteem, and life satisfaction related to QOL directly correlate to mood. Literature consensus shows VR helps moderate some behavioral symptoms and is feasible for PwD. Researchers have found VR decreases agitation. VR decreases depression or anxiety, and BPS, and increases alertness, attention, and engagement. In addition, PwD found VR enjoyable, pleasurable, relaxing, or the preferred intervention method.

Implementation strategies:

- Supervisor, evidence-based practice (EBP) council, and leadership project approval.
- Worked with HBPC providers and leadership to identify veterans who met the criteria.
- Ascertained veteran/caregiver interest, provided project details, and scheduled initial visit.
- On first visit, caregiver completed the psychological well-being of cognitively impaired persons (PWB-CIP), which assesses 11 variables of observable behaviors, with a range of possible scores between 11 - 44. A higher score indicates greater psychological well-being. The veteran then received a VR session that enhanced relaxation, and on the following 2 days, the veteran received VR sessions.
- After all VR sessions, the caregiver completed the PWB-CIP.
- Pre- and post-aggregate questionnaire results were compared and reported.

Outcomes measurement plan or project evaluation/post-implementation data: Seventeen veterans received all 3 sessions. VR sessions lasted 20 - 90 min. The scores on the tool varied from 24 to 43. The mean score pre-VR was 35 and the mean score post-VR was 37.4. Of the 11 variables, 8 variables yielded higher scores. The greatest significance with a p-value of 0.05 showed VR reduced frustration or agitation when asked to complete an activity.

Sustainment and dissemination plan: HBPC recreational therapists are now offering VR to the HBPC veterans with dementia. The ongoing VR use may lead to expanded availability and use in other services for veterans with dementia. Published in the Journal of the American Psychiatric Nurses Association. Presented at the 6th Annual VISN 21 Nursing Research and EBP Symposium.

Inherent Dysfunctional Cellular Energy Metabolism is a Risk Factor for Late-onset Alzheimer's Disease

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Abstract

Age is the main risk factor for late-onset Alzheimer's disease (LOAD) and all processes in normal aging also occur in LOAD pathology. While some factors appear to be more specific to LOAD and may be genetically determined, others are acquired through the aging process. Because bioenergetic metabolism is among the most fundamental features of cell functions, changes thereof have profound effects on every aspect of aging and could be key determinants in the pathology of LOAD. In both fibroblasts and induced pluripotent stem cell-derived immature and mature brain cells of subjects with LOAD, we observed bioenergetic substrate deficiencies, including the redox agent nicotinamide adenine dinucleotide or glucose, diminished insulin/insulin-like growth factor-1 signaling, changes in lipidomic and transcriptional profiles, and multiple alterations of bioenergetic and metabolic cell functions. Overall, our data suggests that dysfunctional bioenergetics is an inherent cell-specific and cell-autonomous risk factor in LOAD. Together with findings in the brains of normally aging individuals or in patients with LOAD, the data support the view that in LOAD, inherent cell-metabolic dysfunctions may occur in development and early life altering the trajectory of the normal aging process already in youth and middle-age and predispose neuropathological events that lead to symptomatic features of LOAD in late life. Results of these studies may reveal targets and methods for treating individuals at risk for LOAD before illness develops or delaying disease onset and progression later in life.

Developing a Quantitative, High-throughput Platform Using Virus-infected Cerebral Organoids for Neuroinflammation in Alzheimer's disease

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Abstract

We used dissociated cells from human cerebral organoids (dcOrgs) that had been infected by herpes simplex virus 1 (HSV-1) and observed several molecular features associated with Alzheimer's disease (AD). In parallel, we conducted bulk RNA sequencing on HSV-1-infected dcOrgs and observed that the differentially expressed genes were enriched for AD-associated genome-wide association study genes. We further dissected the signal by using single-cell spatial and non-spatial RNA-sequencing on HSV-1-infected dcOrgs, combined with single- nuclear RNA-sequencing conducted on human post-mortem brains.

The Dawn After the Long Night: Horizons in Autoimmune Neurology

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Abstract

Autoimmune neurological diseases have traditionally been treated with chronic immunosuppression focused on symptom control rather than disease modification. Advances in immunology and cellular engineering are redefining this paradigm. Improved understanding of immune dysregulation has enabled precision therapies such as FcRn inhibitors, complement blockade, and B-cell depletion, improving outcomes in disorders including myasthenia gravis and neuromyelitis optica spectrum disorder. Emerging strategies aim to reset immunity through plasma cell targeting, antigen-specific tolerance, and immune reconstitution. At the frontier, engineered cellular and gene-based therapies—including chimeric antigen receptor t cell, chimeric antigen receptor natural killer cell, clustered regularly interspaced short palindromic repeats-mediated editing, and regulatory T-cell engineering—offer the potential for durable, possibly curative immune correction.

Sex and Menopause as Cross-cutting Modifiers of Motor and Non-motor Circuits in Parkinson's disease: A Multi-model α-synuclein Strategy

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Abstract

Parkinson's disease (PD) is the fastest-growing neurodegenerative disorder, affecting ~10 million people worldwide and imposing an escalating societal and economic burden. Despite major advances, one critical factor remains consistently underexamined: how biological sex through chromosomal and hormonal mechanisms—shapes onset, diagnosis, and treatment response for women with PD. As a result, the mechanisms linking sex, hormonal transitions, and α-synuclein–driven pathology remain poorly understood and are rarely incorporated into therapeutic development. To address these gaps, we employed two complementary α-syn–based mouse models: (1) Striatal α-syn preformed fibrils (PFF) to examine sex differences in α-syn propagation, limbic circuit remodeling, and non-motor behaviors; (2) Adeno associated virus (AAV)-mediated α-synuclein (SNCA) overexpression in the substantia nigra, combined with the physiologically relevant accelerated ovarian failure (AOF) model, to investigate menopausal influences on progressive dopaminergic degeneration and motor impairment. In the PFF model, males and females displayed divergent α-syn propagation, amygdala connectivity changes, and anxiety- and cognitive-related behaviors, revealing sex-biased functional reorganization of limbic circuits. In the AAV.SNCA + AOF model, menopause ac-

celerated motor deficits and heightened nigrostriatal vulnerability, validating AOF as a biologically relevant menopausal model for PD research. Together, these findings demonstrate that sex and hormonal state act as cross-cutting modifiers of PD pathophysiology and underscore the need to align biological questions with circuit-specific readouts and symptom-appropriate PD models. Importantly, this multi-model strategy provides a translational framework for elucidating sex- and menopause-specific mechanisms of PD, ultimately enabling the discovery and development of targeted therapies that improve outcomes for both women and men.

Childhood Trauma, Emotion Dysregulation, and Cannabis Use in Psychosis Risk: A Review and Integrative Model

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Abstract

Background: Childhood trauma (CHT) is a well-established risk factor for psychosis, yet the mechanisms through which early adversity leads to vulnerability remain unclear. Emotion dysregulation and cannabis use, also important risk factors for psychosis, have often been examined individually. This review synthesizes evidence across these domains to propose a developmental framework in which CHT, emotion dysregulation, and cannabis use interact to shape psychosis risk.

Methods: Systematic review of past 10 years literature examining relationships among CHT, emotion regulation, and cannabis use, as they related to psychosis.

Results: Across studies, CHT was consistently associated with emotion regulation difficulties, including increased stress-response symptoms. Emotion dysregulation frequently mediates associations between trauma exposure and psychosis-related outcomes, including symptom severity, psychotic experiences, and functional impairment. Further evidence suggests that individuals with trauma-related emotion regulation difficulties may be more likely to engage in maladaptive coping such as cannabis use. Cannabis use in the early teenage years, frequently and/or in large amounts, is independently associated with increased risk and earlier onset of psychosis. Additionally, multiple studies demonstrated additive effects of cannabis use when combined with childhood adversity. These findings helped us develop a multidimensional model of psychosis risk where genetic vulnerability and CHT shape neurodevelopmental alterations that promote emotion dysregulation, with cannabis use and social context amplifying proneness.

Conclusions: Emotion dysregulation appears to be a central, modifiable mechanism linking early adversity to later substance use and psychosis risk. Clinically, these findings underscore the importance of assessing trauma and emotion regulation in individuals at risk.

Neurobiology Meets the Clinic: Making Informed Exam Room Decisions in Autism Spectrum Disorder

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Abstract

Autism spectrum disorder (ASD) stands at the crossroads of neuroscience and clinical practice, challenging clinicians to translate complex neurobiological data into practical, individualized care. Over the past decade, advances in genetics, neuroimaging, and electrophysiology have revealed critical insights into the autistic brain—highlighting alterations in neural connectivity, excitation-inhibition balance, and sensory integration that shape cognition, perception, and behavior. Despite these findings, the practical integration of neuroscience into everyday clinical decision-making remains limited. "Neurobiology meets the clinic" seeks to bridge this divide by transforming cutting-edge research into actionable insights for neurologists, psychiatrists, and pediatricians working directly with individuals on the autism spectrum. This 2 h session will provide a structured overview of key neurological mechanisms underlying autism, emphasizing how these translate into observable patterns of social commu-

nication, emotional regulation, and adaptive behavior. Through case-based learning, participants will examine how to interpret clinical signs through a neurobiological lens, enhancing diagnostic accuracy, improving empathy, and supporting evidence-based care. The session will explore the role of biomarkers, digital neuro-assessment tools, and connectivity models that are reshaping modern understanding of ASD. Special focus will be placed on practical, exam room applications: adapting communication to sensory sensitivities, managing diagnostic ambiguity, and engaging patients and families through neuro-informed explanations. Participants will gain a framework for neurobiologically grounded, compassionate clinical decision-making aligning the precision of neuroscience with the art of personalized care.

Learning Objectives:

- Describe current neurobiological insights into cortical connectivity, excitation-inhibition balance, and sensory processing in autism.
- Recognize how these neurological mechanisms shape observable behaviors and patient presentation.
- Apply neuro-informed frameworks to improve diagnostic accuracy and clinician-patient communication.
- Evaluate emerging biomarkers and neuro-assessment tools that support evidence-based decision-making.
- Integrate neuroscientific principles into compassionate, individualized care across the autism spectrum.

An Analysis of Newspaper Reports on The Success of Individuals with Autism Spectrum Disorder

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Abstract

This study aims to change the negative attitudes towards individuals with autism spectrum disorder (ASD), to analyses the news published about their success. The news reports published in The New York Times and The Washington Post newspapers, which were only about the success stories of individuals with ASD, were analyzed by document analysis and content analysis, which are qualitative research methods. In this study, 40 news from the two international newspapers were analyzed, and the news items were evaluated under the headings of gender of the individual with ASD, country of publication, year of publication, supporting institution, source of inspiration and field of success. It was revealed that success stories in the area of art were published more than other areas. In this study, it is noteworthy that the role of teachers, who are important sources of inspiration in the educational life of individuals with ASD, in success stories is quite low. In this respect, it may be suggested that in future studies, newspapers in the international newspapers may publish news in which the effect of teachers on the success stories of individuals with ASD is mentioned more.

Maintenance Electroconvulsive Therapy for Treatment-resistant Irritability in Autism Due to Depression in an Adolescent: A Case Study

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Abstract

Background: Irritability is a frequent and functionally impairing symptom in individuals with autism spectrum disorder (ASD). While often managed with antipsychotics and behavioral interventions, treatment resistance may indicate underlying psychiatric comorbidities such as mood disorders or catatonia. Accurate differential diagnosis is crucial, particularly in ASD, as clinical manifestations may overlap and confound treatment. Electroconvulsive therapy (ECT) remains a potentially effective intervention in this population. This presentation aims to discuss differential diagnosis in patients with ASD through a case.

Case Presentation: We report the case of a 16-year-old male adolescent with nonverbal ASD who presented with severe self-injurious and aggressive behaviors, refractory to multiple pharmacological agents and behavioral interventions. Despite extensive pharmacotherapy no sustained clinical benefit was observed. Given the persistence of irritability and overlapping features of depression, ECT was initiated. Subsequent introduction of fluoxetine, alongside maintenance ECT (M-ECT), led to a substantial and sustained reduction in irritability, and enhanced social engagement. At the time of reporting, the patient had received his 36th M-ECT session with continued clinical stability and a marked decline in Aberrant Behavior Checklist irritability scores. Follow-up of the case included M-ECT and as of today, he has received over 80 sessions of ECT.

Conclusion: This case underlines the importance of considering affective comorbidity in adolescents with ASD who exhibit treatment-resistant irritability. ECT, when integrated with antidepressant pharmacotherapy, may represent a safe and efficacious option in selected, complex clinical scenarios. Further research is warranted to delineate the role of ECT in ASD-related irritability, particularly within the context of comorbidity.

Exosomes, Stem Cells, Tertiary Lymphoid and Cranioencephalic Functional Lymphoid Units for Restorative Neurology

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Abstract

We have shown utility of extracellular microvesicles, including exosomes, as sensitive liquid biopsy friendly biomarkers for neurological and other diseases, as well as carriers for biological, e.g., stem and immune cell, and molecular therapies. These studies also highlighted concurrent stem cell advances for neurodegenerative disease and cancer that have also led to new clinical trials for e.g., Parkinson's disease and the gliomas. Many emerging applications of immunotherapies both *in vitro* and *in situ*, including direct cellular reprogramming and also relying on data science and gene therapy with viral transductions (and with new enhancers) or gene and base editing using e.g., clustered regularly interspaced short palindromic repeats a or i, and with guide RNAs, are advancing pathway targeting and their activation or interference. With concomitant advances in immunology, e.g., our recent discovery and characterization of a cranial bone marrow with functional lymphoid units in glioblastoma, we now know that the skull, meninges and brain together harbor tertiary lymphoid-like structures available for immunotherapies in neurodegenerative and neoplastic diseases. A restorative neurology handbook might one day soon include access to rich multi-omics datasets guiding e.g., RNA and vaccine therapies, that along with stem cell plasticity and direct cellular reprogramming *in vivo*, could help target and eliminate proteinopathies associated with neurodegenerative disease, as well as tumorigenic cells in the gliomas.

Advanced Treatment of Schizophrenia

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Abstract

Schizophrenia, a chronic disabling disease, is a complex psychiatric disorder which has a prevalence of about 1% in the whole world. In this disease, positive schizophrenia symptoms, for example paranoia, hallucinations, and above all acoustic hallucinations and illusions occur. These positive symptoms can be well ameliorated by the administration of first- and second-generation antipsychotic drugs. Negative schizophrenia symptoms are social withdrawal, reduced communication and depression. These negative symptoms can only be emended to a weak extent by the current available antipsychotic drugs. Besides, cognitive symptoms, for example a reduced concentration can occur in patients with schizophrenia and unfortunately these symptoms cannot at all be improved by the current pharmacotherapy. Therefore, investigations about new or improved antipsychotic drugs, a better cognitive behavior of the patients and reduced hospitalizations are of a great importance in schizophrenia. Taking together, the scientists participating in this special issue have demonstrated in schizophrenia that risperidone In Situ Microparticles® achieved sustained and rapid therapeutic plasma levels; that patients suffering from the disease have independent risk factors to have secondary diseases from arteriosclerosis; that electroconvulsive therapy decreased positive and negative syndrome scale total score and favored a balance of pro- and anti-inflammatory cytokines; that the computer program RehaCom® improved cognitive capabilities, and that neurological soft signsis related with schizophrenic symptoms and not with extrapyramidal ones favored by neuroleptics. Moreover, the therapeutic effects of electroconvulsive therapies in schizophrenia have been reported and the pharmacological interventions to improve the negative symptoms in this disease have been stated.

Combining Early Lower Eyelid Surgery with Neuromuscular Retraining for Synkinesis Prevention After Facial Palsy: The Role of the Eye in Aberrant Facial Nerve Regeneration

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Abstract

Abstract:

Background: Facial synkinesis is a distressing sequela of facial palsy (FP) characterized by involuntary, simultaneous movements of facial muscles occurring during voluntary facial expressions. Treatment of synkinesis is challenging, and preventive methods are needed.

Aim: This study evaluated the efficacy of physical facial nerve rehabilitation (PFNR) therapy alone vs PNFR with eyelid surgery to correct lagophthalmos and prevent the onset of synkinesis.

Methods: Twenty-five outpatients were randomized to receive either PFNR alone (neuromuscular retraining and Kabat proprioceptive neuromuscular facilitation) or PNFR and early (90 days after FP onset) eyelid surgery (involving a conservative oculoplastic correction for lagophthalmos with epiphora or ectropion). Comprehensive otolaryngological assessments and magnetic resonance imaging were conducted. Synkinesis progression was measured using another disease scale at baseline, 3, 6, 12, and 24 months post-treatment. The data were analyzed with analysis of variance, τ-test, and Chi-square analyses.

Results: Patients undergoing eyelid surgery with PFNR showed faster ($p < 0.001$) and better recovery of facial movements ($p < 0.05$) than patients receiving PFNR alone comparing T0 and T12 ($p < 0.0001$). No synkinesis was observed in the PFNR plus surgery group while 37% of patients in PFNR alone had synkinesis ($p = 0.03$). At 24 months, none of the patients in the surgery group presented synkinesis.

Conclusion: Combining early surgical treatment of paralytic lagophthalmos or epiphora with PFNR accelerated functional recovery and reduced synkinesis in patients with FP compared to facial rehabilitation alone. Further investigations into larger populations with long-term follow-up are needed.

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