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Deactivation of Peripheral Nerves for the Management of Migraine Headache

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Abstract

With 13% global prevalence, migraine is the most commonly diagnosed and treated neurologic disorder. Headache is the most common presenting symptom of migraine which could be disabling. Although traditionally understood as a central nervous system disorder, recent research highlights the role of the peripheral nervous system in migraine pathophysiology. Blocking of peripheral nerves always has been used as a treatment modality for migraine headaches. Deactivation of the same peripheral nerves with surgical intervention could be a more permanent solution for headache relief in migraine patients. Nerve blocks as a diagnostic method for migraine headache trigger sites can be a good predictor of response to deactivation procedures. In the last few years migraine surgery has made strides in part because of the discovery that migraines can be triggered at specific sites of peripheral nerve compression across the head and neck, although this surgery still is controversial. Numerous anatomical studies have been conducted to elucidate and pinpoint trigger sites for migraine headaches. In practice determining the trigger sites determine with a detailed history, a headache log and proper head and neck exam, then it confirms with nerve block before considering trigger site deactivation to provide more accurate and targeted surgical decompression.

Novel CMT2E Mouse Models with Neurofilament Light Chain E397K Mutation Reveal Early Chronic Axonal Neuropathy and Phenotype Rescue via AAV Knockdown-and-replace Therapy

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Abstract

Charcot-marie-tooth type 2 (CMT2) is presented as a slow, progressive disorder associated with axonal dysfunction. CMT2 clinical symptoms include distal muscle weakness and atrophy, sensory loss, toe and foot deformities, and reduced nerve conduction velocity. CMT type 2E (CMT2E) is associated with mutations in the gene neurofilament light chain (NEFL). The protein, NF-L, is one of five subunits that makes up neurofilaments and contributes to the axonal cytoskeleton. To advance the development of therapeutics for CMT2E and to better understand the underlying biology of disease progression, we generated NEFL+/E396K and NEFLE396K/E396K mice. Motor function, balance, and strength assessments were performed on the NEFL mice to evaluate disease progression. Additionally, muscle pathology, innervation status, nerve morphology, and *in vivo* measurements of neuronal function highlight a quantitative CMT2E phenotype as early as P21. Delivery of an AAV9 vector expressing a shRNA targeting NEFL and a shRNA-resistant cDNA significantly improved the CMT phenotype, including electrophysiology, neuronal and cellular pathology. Collectively, our thorough phenotyping of these novel CMT2E mouse models of disease conclusively demonstrates that there is an early quantifiable neurological phenotype that is clinically relevant, and a pre-clinical knock-down-and-replace strategy can have a significant impact upon disease severity.

Liver Survival Motor Neuron Restoration in Spinal Muscular Atrophy: The Key to Rescue of Whole-body Pathology?

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Abstract

The liver is a key metabolic organ, acting as a hub to metabolically connect various tissues. Spinal muscular atrophy (SMA) is a neuromuscular disorder where patients have an increased susceptibility to developing dyslipidemia and liver steatosis. It remains unknown whether fatty liver is due to an intrinsic or extrinsic impact of survival motor neuron (SMN) protein depletion. Using an adeno-associated viral vector with a liver specific promoter (albumin), we restored SMN protein levels in the liver alone in *Smn*^{2B/-} mice, a model of SMA. We demonstrate that AAV9-albumin-SMN successfully expresses SMN protein in the liver with no detectable expression in the spinal cord or muscle in *Smn*^{2B/-} mice. Liver intrinsic rescue of SMN protein was sufficient to increase survival of *Smn*^{2B/-} mice. Fatty liver was ameliorated while key markers of liver function were also restored to normal levels. Certain peripheral pathologies were rescued including muscle size and pancreatic cell imbalance. Only a partial central nervous system recovery was seen using liver therapeutic strategy alone. The fatty liver phenotype is a direct impact of liver intrinsic SMN protein loss. Correction of SMN protein levels in liver is enough to restore some aspects of disease in SMA. We conclude that the liver is an important contributor to whole-body pathology in *Smn*^{2B/-} mice.

Developing Modified Dihydropyridin-Compound as a Therapeutic for Anxiety Disorder, ASD, and PTSD with Sleep Problems

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Abstract

Posttraumatic stress disorder (PTSD) is a chronic and disabling anxiety disorder that affects 3.5% of US adults. Patients with PTSD are twice as likely to develop dementia and Alzheimer's disease (AD). Sleep disturbances are prevalent in 70 - 91% of patients with PTSD and traumatic brain injury (TBI), yet current treatments are often limited by adverse effects such as liver dysfunction, mood changes, metabolic issues, and suicidal ideation. The physiological mechanisms underlying these sleep disturbances are not fully understood, but they are closely linked to the GABAergic pathway, which regulates inhibitory neural activity and is crucial for sleep. Despite this, most current medications do not target this pathway. Dihydropyridin (DHP), an herbal compound extracted from *Amelops*, has emerged as a potential treatment for PTSD-induced sleep disorders. Previous studies in rodent models of acute stress disorder (ASD) have shown DHP to reduce anxiety-like behaviors and cognitive impairments. In recent clinical trials, DHP was found to significantly improve stress and depression levels, enhance sleep duration and quality, particularly deep sleep (slow-wave sleep, SWS, N3), and improve cognitive function compared to placebo. These findings suggest that DHP could serve as an effective therapeutic option for improving the mental health and well-being of individuals with PTSD and related disorders, particularly in the contexts of stress and social distancing.

Comparative Analysis of Daytime Sleepiness and Associated Factors in Patients with Parkinson's Disease

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Abstract

Daytime sleepiness (DS) manifests in various clinical forms and significantly impacts the quality of life in patients with neurological disorders. This study aimed to compare patients with isolated sudden sleep attacks (SSA, n = 10), persistent sleepiness (PS, n = 10), combined SSA-PS (n = 12), and a control group without DS; (n = 10). Key findings revealed that patients with combined SSA-PS had longer disease durations, higher levodopa dosages, and more severe motor impairments than other groups (p < 0.05). The PS group exhibited the lowest equivalent levodopa dosage compared to SSA and SSA-PS groups. Patients with SSA-PS demonstrated a significantly shorter mean sleep latency during daytime sleep studies (p < 0.05) and more frequent sleep-onset REM periods. Nighttime sleep analysis revealed reduced sleep efficiency, increased wakefulness after sleep onset, and structural alterations, including reduced REM sleep duration (p = 0.06) in PS and SSA-PS groups. Cognitive assessments indicated heightened executive dysfunction and memory impairments in SSA-PS patients compared to SSA and

controls ($p < 0.05$). The findings highlight the heterogeneity of DS and its impact on motor, cognitive, and emotional parameters. These differences underscore the need for tailored therapeutic approaches targeting specific forms of DS, especially in SSA-PS patients. Further research is warranted to explore the underlying mechanisms and optimize treatment strategies for this population.

Health Qigong Exercise on Shifting Function in Parkinson's Patients

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Abstract

Background and Purpose: Parkinson's disease (PD) patients frequently exhibit executive dysfunction due to neurodegenerative processes, with impaired shifting function contributing to diminished cognitive flexibility and multitasking deficits. Previous studies have demonstrated that non-pharmacological interventions, exemplified by health qigong exercise, alleviate PD motor symptoms by enhancing motor coordination and neuroplasticity. This study specifically investigates the impact of health qigong exercise on shifting function within the executive domain of PD patients, aiming to elucidate its therapeutic efficacy and clinical applicability.

Methods: A cohort of 28 mild-to-moderate PD patients was randomized into an experimental group ($n = 14$) and a control group ($n = 14$). The experimental group received 12 weeks of health qigong exercise (3 sessions/week, 60 min/session) alongside standard pharmacotherapy, while the control group adhered to routine treatment. The shifting function was assessed pre- and post-intervention using the standardized odder switching test to quantify task-switching efficiency and response accuracy.

Results and Discussion: 1. Significant improvements ($p < 0.05$): The experimental group demonstrated reduced reaction times and elevated task-switching accuracy, indicating enhanced cognitive flexibility and executive efficiency attributable to health qigong exercise. 2. Mechanistic insights: The intervention likely optimized neuromuscular coordination through deliberate slow movements and respiratory modulation, potentially activating prefrontal cortical regions and basal ganglia circuits to reinforce neural pathways associated with shifting function. 3. Control group outcomes: No statistically significant changes ($p > 0.05$) were observed in the control group, underscoring the specificity of health qigong exercise in targeting shifting function.

Conclusion: Health qigong exercise effectively ameliorates shifting function in PD patients, bolstering multitasking capacity and cognitive adaptability, thereby offering a scientifically grounded strategy to decelerate executive decline. These findings advocate integrating health qigong exercise into comprehensive PD management protocols to enhance functional independence and quality of life.

Exercise Reduces Transthyretin Gene Expression in the Hippocampus

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Abstract

It has been reported that elderly people who exercise regularly have a reduced risk of developing cognitive disorders. Furthermore, it has been reported that aerobic exercise and strength training can improve cognitive function and increase brain volume in elderly people. However, the mechanism of the relationship between exercise and the brain is unclear. Therefore, in this study, we aimed to clarify the genes in the hippocampus that change with exercise in mice. C57BL/6J-Aged (68 weeks old) mice were divided into two groups: a normal feeding group (control) and a group that was placed in a cage with a wheel (exercise group). After 4 weeks, the hippocampus was taken from each mouse, RNA was extracted, and genes that increased or decreased by more than twice the control group were analyzed by microarray. As a result, 63 genes were increased in expression and 139 genes were decreased in expression in males, and 391 genes were increased in expression and 268 genes were decreased in expression in females. Among these, the gene that showed the largest decreased change in expression in both males and females was the transthyretin (TTR) gene. We have reported that in cognitively normal older people, serum TTR levels were negatively correlated with cognitive function scores, independent of sex, age, and education, and that in a longitudinal study, subjects with high TTR levels showed significantly decreased cognitive function. These results suggest that exercise may reduce TTR expression in the hippocampus and affect cognitive function.

The Effect of Ketamine Infusion in the Treatment of Complex Regional Pain Syndrome

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Abstract

Complex regional pain syndrome (CRPS) is a painful debilitating neurological condition that accounts for approximately 1.2% of adult chronic pain population. Ketamine, an NMDA receptor antagonist, is an anesthetic agent that has been used for CRPS. There is a growing body of clinical evidence to support the use of ketamine in the treatment of CRPS. A search of Embase, Pubmed, Web of Knowledge, Cochrane, Clinical Trial.gov, and FDA.gov between January 01, 1950, and August 01, 2017, was conducted to evaluate ketamine infusion therapy in the treatment of CRPS. We selected randomized clinical trials or cohort studies for meta-analyses. I 2 index estimates were calculated to test for variability and heterogeneity across the included studies. The primary outcome is pain relief. The secondary outcome is the pain relief event rate, which is defined as the percentage of participants who achieved 30% or higher pain relief in each of the qualified studies. Our results showed that the Ketamine treatment led to a decreased pain score in comparison to the self-controlled baseline ($p < 0.000001$). However, there is a statistical significance of between-study heterogeneity. The immediate pain relief event rate was 69% (95% confidence interval (CI) 53%, 84%). The pain relief event rate at the 1 - 3 months follow-ups was 58% (95% CI 41%, 75%). Our findings suggested that ketamine infusion can provide clinically effective pain relief in short term for less than 3 months. Studies are needed to prove long-term efficacy of ketamine infusion in the treatment of CRPS.

A Case Study Examining a Neurological Disorder in a Culturally and Linguistically Diverse Early Elementary Student

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Abstract

Neurological conditions, including rare disorders like absence of septum pellucidum (ASP), significantly impact individuals' medical and psychological well-being. ASP involves the absence of a brain membrane integral to the limbic system, which regulates emotions, memory, and behavior. The current understanding of ASP is limited, with associations to various neurological anomalies and psychiatric disorders. Specifically, there is little information on how ASP presents in early childhood. This case study explores the neurobehavioral profile of "Charlie," a 7 year old boy diagnosed with ASP, referred for psychological evaluation due to social-emotional and behavioral concerns. Charlie's cognitive profile demonstrated overall average intelligence but normative deficits in executive functioning. Charlie experiences hyperactivity, aggression, anxiety, depressive symptoms, and impulsiveness. Following this assessment, Charlie met DSM-5 criteria for oppositional defiant disorder, intermittent explosive disorder, and attention-deficit hyperactivity disorder, combined presentation. Charlie's assessment highlights significant emotional and behavioral challenges despite average general cognitive abilities. Given that the SP is associated with the limbic system function, its absence could indicate a behavioral impact as seen in this case report. This study underscores the need for further research on ASP, particularly in culturally and linguistically diverse populations, to inform clinical assessment, prognosis, and intervention strategies.

Ferroptosis is Involved in Neurodegenerative Disorders Including Diabetes-associated Cognitive Impairment

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Abstract

As central immune cells, microglia have strong siderophilic properties. However, the role of iron deposition in microglia and the underlying regulatory mechanism remains unclear in diabetes-associated cognitive impairment (DACI). Here, we established high glucose (HG) model in BV2/HMC3 cells and diabetes model in C57BL/6J mice with HFD and STZ. Transmission electron microscopy, Western blot, assay kits of Fe²⁺, GSH/GSSG, MDA and ROS were carried out *in vitro*. Prussian blue staining, Western blot and immunofluorescence were implemented *in vivo*. Y-maze and novel object recognition were performed to assess cognitive performance. LP17 was used to inhibit TREM1 (triggering receptor expressed on myeloid cells 1) specifically *in*

in vivo and *in vitro*. We found excessively deposited iron and significant reduction in antioxidants in hippocampal microglia of mice with DAPI, concomitant with increased TREM1 (a microglia-specific inflammatory amplifier). Furthermore, LP17 (TREM1 specific inhibitor) ameliorated cognitive impairment caused by HFD/STZ through relieving iron accumulation and antioxidant inactivation. *In vitro*, ferroptosis was induced by HG in mice microglia-BV2 and human microglia-HMC3 cells, which could be blocked by a ferroptosis inhibitor-Fer-1 and LP17. Moreover, PERK pathway of endoplasmic reticulum stress was activated by HG and then reversed by LP17 in HG-cultured BV2. In summary, our results indicated that TREM1 effectively aggravates T2DM-associated microglial iron accumulation through the PERK pathway of ERS, which contributes to antioxidant inactivation and lipid peroxidation, eventually, massively boosted ROS result in microglial ferroptosis. The mechanism elucidation in our study may shed light on targeted therapy of DAPI.

Stereoencephalography Based Platform Device for Neural Recordings, Ablation and Targeted Drug Delivery into the Brain

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Abstract

Stereoencephalography (sEEG) electrodes are routinely used to identify the epileptogenic zone in patients with drug resistant epilepsy. The sEEG electrodes are FDA-cleared for temporary (<30 days) neural activity monitoring, recording and stimulation. Here we present bench and *in vivo* studies for two new clinical functions added to the sEEG electrodes: 1. temperature-controlled radiofrequency ablation (RFA) and 2. convection enhanced drug delivery (CED). sEEG-guided RFA produced lesions proportional to temperature and time (diameter 4-10 mm) in *ex vivo* tissue and *in vivo* swine brain. Drug delivery through modified sEEGs (DD-sEEG) achieved CED for infusion rates of 0.5 - 15 $\mu\text{l}/\text{min}$ and infused volumes (V_i) of 100 - 1000 μl , with V_d/V_i ratio of 2.7 - 3.4. *In vivo* in the rat and swine hippocampus, penicillin delivered through DD-sEEGs elicited seizure-like episodes, consisting of large amplitude bursts of coordinated activity interspaced by quiet periods. MRI visualization of Gd demonstrated localized infusions in the swine putamen and hippocampus. These data demonstrate the ability of a single sEEG-based device platform to perform multiple functions: record brain activity, stimulate, ablate and deliver drugs. This has the potential to increase the accuracy of diagnosis and offer treatment within one surgical procedure. Furthermore, real time monitoring of neural activity during infusion of therapeutic compounds can be used to probe the function of various brain structures, and/or evaluate onset and mechanism of action, dosing, efficacy and safety of therapeutic compounds.

Endothelial C3a Receptor Mediates Inflammatory Signaling in Murine Stroke

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Abstract

Stroke is regarded the second leading cause of death and disability worldwide with an annual mortality rate of ~5.5 million and it reduces mobility in more than half of stroke survivors in aged populations. Stroke is caused by a clot by obstructing blood flow to the brain. In Arizona, USA the highest age-adjusted stroke mortality rates are in rural counties Greenlee, Navajo and Yavapai with limited access to tertiary stroke centers. The narrow temporal window and limited availability of, and eligibility for, thrombolytic therapy and endovascular thrombectomy are major therapeutic limitations. Neuroprotective therapies that could be given early to replace or augment existing therapies could improve stroke outcomes. We showed that monosialoganglioside (GM1) containing nanoliposomes (NLGM1) composed of GM1, cholesterol and phosphatidylcholine (5/25/70% molar ratios) protect against hypoxic/ischemic injury likely through Nrf2-dependent upregulation of antioxidant enzymes. Our aims are to (1) optimize NLGM1 treatment to reduce acute stroke following middle cerebral artery occlusion (MCAO) and (2) test whether NLGM1 can confer chronic protection following photothrombotic (PT) stroke injury.

A Case of ME/CFS and Hypothalamic Adrenal Dysfunction Causing Prolonged, General Fatigue after COVID-19 Infection

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Abstract

Background: Post COVID-19 condition presents with a variety of symptoms. Central nervous system (CNS) complications are still not well recognized or reported. Many clinicians face challenges in diagnosis, and patients could be left without adequate treatment or support.

Case Presentation: We report a case of middle-aged male who developed persistent brain fog and profound fatigue after COVID-19. Even though conventional brain computed tomography (CT), and magnetic resonance imaging (MRI) were normal, brain perfusion scintigraphy revealed decreased cerebral perfusion and MRI-VSRAD revealed diffuse brain atrophy. Endocrinological testing demonstrated hypothalamic adrenal insufficiency. The patient was diagnosed with myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) complicated with hypothalamic adrenal insufficiency.

Discussion: In patients with persistent mental symptoms and general fatigue after COVID-19, CNS involvement including post-viral encephalopathy should be considered. ME/CFS and hypothalamic-pituitary-adrenal axis dysfunction are important differential diagnoses. Advanced brain imaging and endocrinological tests are required. Treatment should include steroid replacement when adrenal insufficiency is present, and symptomatic therapy for ME/CFS. Physical rehabilitation should be carefully and gradually performed to avoid post-exertional malaise (“crash”).

Conclusion: It is challenging to diagnose and treat post COVID-19 condition. We hope this case report will shed light on the investigation of such mysterious illness to save the patients in the world.

Cortical Neuron Dysfunction Occurs at Early-stage AD in the Absence of β -amyloid Deposition

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Abstract

Alzheimer's disease (AD) is a neurodegenerative disease that causes severe neurocognitive deficits. The β -amyloid ($A\beta$) theory has been considered as a causative factor of AD for decades, but therapeutic strategies based on that are unsatisfactory. Numerous upstream mechanisms that may cause AD are proposed, but none of them has been identified. Meanwhile, it is established that AD is associated with neuronal calcium dyshomeostasis (excessive Ca^{2+} influx/intracellular Ca^{2+} , $[Ca^{2+}]_{in}$), which induces dysfunction, injury, and even death of cortical neurons in the brain regions that regulate neurocognition. These studies suggest a likelihood to reduce excessive $[Ca^{2+}]_{in}$ -induced neurotoxicity, a downstream mechanism that is related to AD, by concurrently but not individually, diminishing overactivation/expression of NMDA receptors (NMDARs) and voltage-gated Ca^{2+} channels (VGCCs), including those formed by accumulated $A\beta$ at late-stage AD, in dysfunctional cortical neurons. However, the medicines currently been using for treating AD, e.g., memantine (which blocks NMDAR) and $A\beta$ antibody (that removes a small portion of $A\beta$ but does not prevent $A\beta$ production) lack the ability to abolish overactive VGCC-mediated excessive $[Ca^{2+}]_{in}$. Thus, a novel pharmacological intervention that combinedly antagonizes NMDAR and VGCC overactivation/expression would be more effective for treating AD. Our study focuses on defining dysfunction of live cortical neurons with NMDAR/VGCC-mediated Ca^{2+} dyshomeostasis and astrocytes in the AD brain; assessing the effects of different $A\beta$ subtypes and their antibodies on them; and proposing a potential novel hypothesis with a working model that may eventually help developing a new, more effective therapeutic approach to treat AD.

The Reciprocal Relationship Between the Gut Microbiota, Intermittent Fasting and Alzheimer's Disease

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Abstract

Alzheimer's disease (AD) is an enormous burden for patients, caregivers and ultimately also for countries. Due to the multifactorial nature of the disease, many biological mechanisms have been described, and today, all possible targets and mechanisms are being investigated. Recently, associations between the gut microbiota and AD, intermittent fasting and AD, and the gut microbiota and intermittent fasting have been documented. Here, we analyze and interpret the relationships in the triangle of AD, gut microbiota, and intermittent fasting by reviewing recently published studies. The main aim of this review is to identify well-defined and undefined pathways or relationships in this triangle. In this way, a new strategy for treating AD with intermittent fasting and a healthy gut microbiota can be developed, as these are non-pharmaceutical and holistic approaches that are also relatively accessible and affordable.

Breakthroughs in Alzheimer's Disease Drug Development

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Abstract

Alzheimer's disease (AD) is a neurodegenerative condition characterized by cognitive decline and memory loss. The current lack of understanding regarding the cause of AD has impeded the development of animal models for drug testing, resulting in a dearth of effective prevention and treatment methods. Despite efforts to target A β plaques and tau protein tangles, which have proven ineffective, it is evident that these factors alone do not fully account for the cognitive deficits and memory loss associated with AD. While clinical trials seek effective treatments, success remains uncertain. Recent attempts to reduce A β levels in AD treatment have proven ineffective, indicating that A β plaques and tau protein tangles are not the sole contributors to cognitive deficits and memory loss. Drugs such as rivastigmine and varenicline have demonstrated no significant benefits in treating AD patients. Although memantine is employed for mild cognitive impairment, a comprehensive analysis reveals limited improvement in cognitive function and a potential risk of gastrointestinal adverse reactions. Clinical trials continue in the pursuit of effective treatments, with outcomes remaining uncertain. Upon examining AD symptoms, it was observed that 70% of patients exhibited anxiety/depression, 17% experienced epileptic seizures, and 60 - 90% faced sleep disorders. These issues point towards dysfunction in the GABA system in AD patients, yet the precise role of GABA in AD pathogenesis remains unclear. GABA, a major inhibitory neurotransmitter, plays a crucial role in initiating and regulating sleep, participating in cognitive formation, and managing anxiety and depression. It collaborates with excitatory neurons to maintain a balance in the brain. Our development of dihydromyricetin (DHM), a positive modulator of GABA, aims to address these imbalances. Research indicates that symptoms such as depression, anxiety, and sleep disorders manifest 15 - 20 years before an AD diagnosis. Social isolation induced loneliness has been identified as a factor contributing to a 50% increase in AD incidence. To replicate the natural onset of AD, we created social isolation animal models that effectively mimic human social isolation. After a 4 week isolation period, we observed increased A β plaques and tau tangles, reduced mitochondrial ATP production, heightened neuroinflammation, and anxiety/depression/aggression. Post-synaptic gephyrin and GABAergic neurotransmitter activity were reduced, accompanied by cognitive impairment. Notably, DHM was able to reverse these pathological changes. Social isolation also induced neuroinflammation, affecting the tripartite synapses formed by astrocytes and neurosynapses, fundamental to cognitive formation. Astrocytes support neurosynapses' structure and function, maintaining cognitive processes. However, even two weeks of social isolation led to astrocyte atrophy and damage to the tripartite synaptic structure, resulting in cognitive impairment. This study provides compelling evidence that tripartite synapses form the basis of cognition, and damage to tripartite synapses is a key mechanism for cognitive impairment. The DHM we developed demonstrates unprecedented efficacy in reshaping tripartite synapses, representing a groundbreaking discovery. We posit that DHM has the potential to evolve into an effective drug for the prevention and treatment of AD.

Effect of Virgin Coconut Oil on Cognition of Individuals with Mild-to-moderate Alzheimer's Disease

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Abstract

Virgin coconut oil (VCO) is a potential therapeutic agent to improve cognition in Alzheimer's disease (AD) due to its ketogenic and antioxidative characteristics. The objectives of this double-blind placebo-controlled trial were to investigate the effect of VCO on cognition in individuals with mild-to-moderate AD and to determine the impact of apolipoprotein E (APOE) ϵ 4 genotype on cognitive outcomes. Participants were 120 Sri Lankan individuals with mild-to-moderate AD (mini-mental state examination (MMSE) = 15 - 25), aged >65 years, and they were randomly allocated to either treatment or control groups. The treatment group was given 30mL/day of VCO and the control group with the same amount of canola oil for 24 weeks. Cognition was assessed by MMSE, and executive clock drawing task (CLOX) at the baseline and at the end of the intervention. Fasting lipid profile and glycated haemoglobin (HbA1C) were analyzed at the baseline and at the end of the intervention. APOE genotyping was conducted at the baseline. There were no significant changes of cognitive scores, lipid profile and HbA1C levels between the groups. However, MMSE scores of APOE ϵ 4 carriers in the treatment group was improved compared to non-carriers (change of MMSE = 2.37, p = 0.021), whereas there was no difference in the control group. Lipid and HbA1C parameters were not compromised during the intervention. In conclusion, VCO did not improve cognition in individuals with mild-to-moderate AD compared to canola oil. However, VCO improved the MMSE scores of APOE ϵ 4 carriers compared to non-carriers. Consumption of VCO was safe as it did not compromise lipid parameters.

The Impact of Pain on the Course of ADL Functioning in Patients with Dementia

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Abstract

Background: Understanding if and how pain influences activities of daily living (ADL) in dementia is essential to improving pain management and ADL functioning. This study examined the relationship between the course of pain and change in ADL functioning, both generally and regarding specific ADL functions.

Methods: Participants were Dutch nursing home residents (n = 229) with advanced dementia. ADL functioning was assessed with the Katz ADL scale, and pain with the Dutch version of the pain assessment checklist for seniors with limited ability to communicate (PACSLAC-D). Changes in PACSLAC-D and Katz ADL scores were computed based on the difference in scores between baseline, 3 month and 6 month follow-up. Multivariate linear regression models were used to assess the relationships between change in pain score, change in total ADL score and specific ADL item scores during follow-up.

Results: At baseline, residents had a median ADL score of 18 (interquartile range 13 - 22, range 6 - 24) and 48% of the residents were in pain (PACSLAC-D ≥ 4). Residents with pain were more ADL dependent than residents without pain. A change in pain score within the first 3 months was a significant predictor for a decline in ADL functioning over the 6 month follow-up (B = 0.10, SE = 0.05, p = 0.045), and specifically, a decline in the items 'transferring' over the 6 month follow-up and 'feeding' during the first 3 months of follow-up.

Conclusion: Pain is associated with ADL functioning cross-sectionally, and a change in pain score predicts a decline in ADL functioning, independent of dementia severity. Awareness of (changes in) ADL activities is clearly important and might result in both improved recognition of pain and improved pain management.

Association of Long-term Blood Pressure Variability with Plasma Neurofilament Light Underlies the Mechanisms in the Comorbidity of Alzheimer's Disease and Cerebral Small Vessel Disease

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Abstract

Introduction: Both long-term blood pressure variability (BPV) and plasma neurofilament light (PNFL) have been identified as potential biomarkers for Alzheimer's disease (AD) and cerebral small vessel disease (CSVD). However, the relationship between BPV, PNFL, and their association with comorbidity, remains unknown.

Methods: Participants with normal cognition and mild cognitive impairment from the ADNI study were included in the data analysis. Linear regression models and causal mediation analyses were conducted to investigate the relationship between BPV over 2 years with PNFL, brain structural changes related to AD and VD, and cognitive function.

Results: BPV was associated with PNFL levels independent of average blood pressure. Mediation analyses revealed that PNFL mediated the associations between BPV and both brain structural changes and cognition.

Discussion: PNFL is linked with BPV and mediates the association between BPV, brain structural changes, and cognition, and underlies the mechanisms involved in the comorbidity of AD and CSVD.

Structural Models of Amyloid Beta Oligomers, Protofibrils, and Channels and their Role in Synaptic Transmission

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Abstract

Alzheimer's disease (AD) is associated with an over-abundance of Amyloid β (A β) peptides. The most toxic variant, A β 42, forms discrete ion channels in neurons. We now propose that our symmetric concentric antiparallel β -barrels of A β 42 channels form 2-D hexagonal lattices that include GM1 lipids and propose that larger channels can form from the fusion of smaller

ones. These A β 42 channel models promise breakthroughs in AD and fusion pore structure and functional mechanisms. A β 42 interacts strongly with synaptic membranes where it competes with the VAMP protein for binding to synaptophysin. The synaptophysin/VAMP complex and SNARE complex are major components of the fusion pore system by which neurotransmitters are released from vesicles into the synaptic cleft. In our models, A β 42 channels form a hexagonal lattice that surrounds synaptophysin and is coupled to the SNARE complex through the VAMP protein. Ca²⁺ ions stimulates the SNARE complex to apply radial tension to the junction of plasma and vesicle membranes. This tension stimulates smaller closed channels to merge to form larger channels of the fusion pore. Our atomic-scale models of A β 42 oligomer, protofibril, and channel assemblies are consistent with numerous experimental results, e.g., electron microscopy, cryoEM, atomic force microscopy, and freeze fracture images, NMR structural studies, secondary structure analyses, biochemical analyses, and single channel currents; and with well-established β -barrel and molecular modeling criteria and theory. Aspects of our models can be tested experimentally, suggest ways to stabilize specific structures for high-resolution structure studies, and identify targets for antibody development.

Hellenic National Biobank of Neurodegenerative Diseases

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Abstract

The Hellenic National Biobank of Neurodegenerative Diseases is a resource of the Panhellenic federation of Alzheimer's disease (AD) and consists of laboratory and sample storage facilities. It aims to facilitate laboratory research in the wide field of dementia, ranging from subjective cognitive decline and mild cognitive impairment to AD, dementia with Lewy bodies, vascular, and frontotemporal dementia. At this moment, the biobank contains 28,205 medical records, where demographic characteristics, disease diagnosis, neuropsychological and neurological examination, as well as additional neuroimaging and neurophysiological tests have been documented, using the Olympias database. Until now, 9,586 magnetic resonance imaging tests and 3,512 electroencephalograms have been recorded. Regarding the biological samples, 1,560 samples of blood plasma, 1,600 samples of serum, 2,300 samples of cerebrospinal fluid (CSF), 766 samples of saliva, 160 fecal samples, and brain tissue from 120 donors are currently stored. So far, all the samples of whole blood (9,316) have undergone DNA analysis for the determination of apolipoprotein E alleles, whereas the presence of markers of amyloid plaques, tangles, and neurodegeneration has been examined in more than 50 % of CSF samples, among numerous other tests performed. Although sample and data collection primarily focus on the spectrum of dementia, a significant portion of samples from other neurodegenerative diseases, including Parkinson's disease and multiple sclerosis, is also available. Through interdisciplinary and inter-institutional collaborations, the biobank enables the conduction of large-scale studies, with the ultimate aim to promote the identification of novel risk factors and biomarkers, as well as the development of innovative therapies.

Determination of the Demyelination Severity in Diabetic Neuropathy with a New Method Based on Electrochemical Aptasensor

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Abstract

Diabetic neuropathy (DN) is a series of neuropathic processes in the peripheral nervous system caused by diabetes-related hyperglycemia and pathways it triggers. Delays in the early diagnosis of neuropathy lead to disease exacerbation and ultimately to the development of diabetic ulcers and further to amputation. Herein, an electrochemical aptasensor-based prototype diagnostic method was developed for the rapid, sensitive, specific, low-cost point-of-care analysis of myelin basic protein (MBP) as a biomarker increasing in serum as a result of demyelination due to DN. Pencil graphite electrodes modified with functionalized graphene oxide nanomaterial were used as the sensor surface with immobilized MBP-specific aptamer forming bioactive binding layer. MBP binding in serum samples was monitored by electrochemical impedance spectroscopy. Serum samples of 14 healthy volunteers and 16 diabetic patients with and without DN were included in the data to be analyzed. Same samples were analyzed with enzyme-linked immunosorbent assay to compare the performance of aptasensor-based method. A statistically significant difference in serum MBP levels was found between healthy volunteers and diabetic patients with and without DN. The aptasensor-based method was able to detect MBP in serum samples with LOD 0.35 ng/mL and in clinical application range from 0.35 to 128 ng/mL. As a proof-of-concept it was shown that there is an agreement between aptasensor-based method and ELISA for detecting MBP levels in serum samples. Authors acknowledge the financial support from Health Institutes of Türkiye (TUSEB), project number 24139.

Effects of Language Delay on General Psychomotor Development. Children up to 6 Years Old

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Abstract

The latest data show that in 2022 and 2023, 666 patients with a diagnosis from the group of diagnoses “Specific speech and language development disorders” were admitted to the Children’s Clinical University Hospital in 2022 and 2023. 395 of them have not visited an audiologist. 607 speech delay is the basic diagnosis. The largest number of patients is aged between 2 and 8 years. They are mostly children aged 2 to 5 years (492) Most have not received any prior therapy. Children with speech delays develop more slowly without appropriate multisensory support. Not only speech, but also other aspects of psychomotor development. The number of patients with speech development disorders increases every year. The number of patients with this isolated disorder is also growing. To achieve maximum results in speech correction, it is not enough to work with a speech therapist. It is fundamentally important to find effective combinations of other multisensory therapy options for patients with speech delays.

Gemfibrozil Alleviates Cognitive Impairment by Inhibiting Ferroptosis of Astrocytes via Restoring the Iron Metabolism and Promoting Antioxidant Capacity in Type 2 Diabetes

Qing Li*

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Abstract

Diabetes-associated cognitive dysfunction (DACD) is considered a significant complication of diabetes and manifests as cognitive impairment. Astrocytes are vital to the brain energy metabolism and cerebral antioxidant status. Ferroptosis has been implicated in cognitive impairment, but it is unclear whether the ferroptosis of astrocytes is involved in the progression of DACD. PPARA/PPAR α (peroxisome proliferator-activated receptor alpha) is a transcription factor that regulates glucose and lipid metabolism in the brain. In this study, we demonstrated that high glucose promoted ferroptosis of astrocytes by disrupting iron metabolism and suppressing the xCT/GPX4-regulated pathway in diabetic mice and astrocytes cultured in high glucose. Administration of gemfibrozil, a known PPAR α agonist, inhibited ferroptosis and improved memory impairment in db/db mice. Gemfibrozil also prevented the accumulation of lipid peroxidation products and lethal reactive oxygen species induced by iron deposition in astrocytes and substantially reduced neuronal and synaptic loss. Our findings demonstrated that ferroptosis of astrocytes is a novel mechanism in the development of DACD. Additionally, our study revealed the therapeutic effect of gemfibrozil in preventing and treating DACD by inhibiting ferroptosis.

A New Approach for Diagnosis and Monitoring of Multiple Sclerosis Based on Electrochemical Nanoaptasensor for MBP Detection

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Abstract

Multiple sclerosis (MS) is recurrent and progressive inflammatory disease of central nervous system (CNS) associated with demyelination. The number of MS patients is increasing, but the diagnostic process is still quite difficult and costly and requires combination of various methods and analysis. Myelin basic protein (MBP) makes up to 30% of myelin and is known to be released into the cerebrospinal fluid (CSF) as a bioindicator of MS. In case of another demyelinating disease or trauma of CNS, MBP is present as biomarker in blood serum. We present the results of developing a new biosensor-based method of MBP detection and monitoring in CSF. MBP specific aptamer earlier developed for therapeutic purposes in mouse model was applied as bioreceptor for both human and mouse MBP recognition. Graphene oxide nanomaterial was integrated into the working electrodes to enlarge surface area and aptamer was immobilized to create a bioactive layer on the sensor surface for MBP binding. The measurements were carried out using electrochemical impedance spectroscopy. Using carbon-based nanomaterial with a large surface area aggregated with aptamer allowed us to achieve high specificity and affinity to the target molecule and enabled selective and sensitive MBP determination. In CSF LOD was 0.65 ng/mL and 0.01 ng/mL for human and mouse model correspondingly. In the future, this developed aptasensor can be implemented for development of prototype products for clinical use in MBP detection as PoC analysis. This research was funded by EGE University Office of Scientific Research Projects (FDK-2020-22395) and Scientific and Technological Research Council of Türkiye (120Z911).

Nonconvulsive Status Epilepticus

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Abstract

Nonconvulsive status epilepticus (NCSE) is a state of continuous seizure activity for at least 30 min, with cognitive or behavioral changes. It is sometimes difficult to diagnose. NCSE constitutes about 25 - 50% of all cases of status epilepticus. NCSE is still underrecognized, yet potentially fatal if untreated. An 82 year old male was admitted to our hospital due to coma, fever, and respiratory failure. He was diagnosed with aspiration pneumonia, heart failure, and rhabdomyolysis. Pneumonia was treated with antibiotics, and heart failure was treated by diuretics. After the treatment of pneumonia and heart failure, he still had a coma. EEG revealed abnormal discharge, and the patient was diagnosed with NCSE. Patient was positive for RPR and TPHA, so that neurosyphilis was also suspected. Other differential diagnoses were also discussed. NCSE could be underdiagnosed due to lack of characteristic symptoms. It should be considered when a patient reveals disturbance of consciousness with no obvious reasons.

Therapeutic Approaches Targeting Post-stroke Vascular Remodeling to Promote Blood-brain Barrier Maturation: Atorvastatin and Non-invasive Vagus Nerve Stimulation

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Abstract

Angiogenesis and re-vascularization are the main repair processes following cerebral ischemic stroke. However, high permeability of blood-brain barrier (BBB) in the newly formed vessels via stroke-induced spontaneously angiogenesis contributes to the disease progression. Thus, novel treatments by targeting functional BBB barrierogenesis during neurovascular remodeling are imperative for functional stroke recovery. Here we report two therapeutic options that address late-stage neurovascular remodeling and functional recovery following ischemic stroke: treatment with atorvastatin as a pre-angiogenic agent, and non-invasive vagus nerve stimulation (nVNS) at 2 weeks after ischemic stroke onset. Spontaneously hypertensive rats were subjected to a 90 min middle cerebral artery occlusion (MCAO) with 8 week reperfusion (RP). Atorvastatin-treated rats received atorvastatin (3 mg/kg) daily for 7 days starting at 14 days after MCAO/RP; and nVNS-treated rats received a nVNS treatment (5 doses of nVNS, duration: 2 min; every 10 min) twice a week from 2 - 8 weeks after MCAO/RP. The rats were studied at multiple time points up to 8 weeks with multimodal-MRI, behavior tests, immunohistochemistry, and biochemistry. The delayed treatment of atorvastatin significantly reduced infarct size and BBB permeability, restored cerebral blood flow, and improved the neurological outcome. Atorvastatin facilitated maturation of BBB properties in the new vessels by promoting endothelial tight junction formation. nVNS significantly reduced core-infarct size and BBB permeability in peri-infarct areas, and improved motor-movement and memory impairments at 8 weeks. Post-mortem histological analysis demonstrated that rat brains treated with atorvastatin or nVNS presented a higher number of micro vessels and a higher expression of presynaptic and postsynaptic proteins in the peri-infarct regions.

Potential Pathophysiological Mechanisms Implicated in New-onset Refractory Status Epilepticus Caused by Viral Agents

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Abstract

In recent years, numerous cases have emerged of patients exhibiting respiratory system symptoms following infection with the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). These symptoms can persist for days, weeks, or even months post-infection. Additionally, literature has documented several individuals experiencing neurological symptoms in the context of post-COVID conditions. Notably, patients with no prior history of epilepsy or other neurological issues may develop new-onset refractory status epilepticus (NORSE) weeks, months, or even up to a year following the viral infection. While NORSE is uncommon, it carries a high mortality rate and can result in permanent epilepsy. Therefore, clinicians should consider this possibility when assessing individuals with NORSE who have recently contracted SARS-CoV-2. This presentation compiles comprehensive information on the mechanisms of epileptogenesis linked to SARS-CoV-2, the diagnosis of NORSE

syndrome, its treatment options, and associated outcomes. These results aim to enhance physicians' understanding of the virus's pathogenesis and increase awareness of NORSE.

Cyclosporin A Inhibits the Damage of α -synuclein in Neurons of Eclampsia-like Seizure

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Abstract

Objectives: Neuroinflammation in preeclampsia damages hippocampal neurons. In inflammatory environment, α -synuclein leads to abnormal neurons excitation by aggregation in mitochondria. Cyclosporine A (CsA) can inhibit inflammatory response and protect mitochondrial function by specifically binding to cyclophilin D (CyPD) in mitochondria. We aim to determine CsA ameliorate the neurons damage induced by α -syn in eclampsia.

Methods: Pregnant rats were randomly divided into P, PE, E, and CsA groups. Rats were given pentylenetetrazol following tail vein injection of lipopolysaccharide to establish the eclampsia-like model. CsA (5 mg/kg) was administered intravenously after LPS infusion. Mean systolic blood pressure and proteinuria were detected. Seizure activity, inflammatory cytokines and pregnancy outcomes were analyzed. Immunofluorescence for quantitative analysis of α -syn and CyPD. Primary neurons were isolated and treated with 1 μ g/ml α -syn. Inhibition of CyPD by siRNA and estimate the effect of CsA by evaluation of neuron morphology.

Results: CsA significantly decreased blood pressure, proteinuria and increased fetal and placental weight. Meanwhile, CsA reduced serum IL-1 β , TNF- α , IL-6 and IL-17 levels and the seizure scores. The expression of α -syn in hippocampal neurons elevated in PE and E group, significantly decreased in CsA group. The expression of CyPD slightly increased in E group. α -syn attenuated PSD95 expression and dendritic mutations in neurons. CsA can reverse neuronal damage caused by α -syn.

Conclusions: CsA improved preeclampsia symptoms and attenuated inflammatory responses. The improvement of neurological symptoms coincidences with a decrease in α -syn expression after CsA treatment, suggesting that CsA might be an alternative management option for preeclampsia and eclampsia.

Dialogic Reading Effect on Communicative Initiations and Responses for Children with Autism: A Systematic Review

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Abstract

Dialogic reading (DR) is an evidence-based shared reading intervention targeting children's language skills. Verbal and nonverbal communication is a common difficulty for most children with autism. Therefore, this systematic review aimed to examine the effect of DR interventions on communicative responses and initiations for children with autism. The review included nine studies in which types of DR interventions were used: the original DR and adapted versions of DR. Both types were equally effective for children's verbal responses. All studies examined the effect on children's verbal communication, while only two studies investigated children's nonverbal communication. The review showed an increase in children's verbal responses, although mixed results were found when measuring verbal and nonverbal initiations. In addition, the systematic review synthesized the quality of the studies and found that all studies showed evidence of moderate to strong research quality. The review concluded that DR is a promising shared reading intervention and can benefit children with autism.

Hierarchical Brain Dysfunction: A Key Factor in Autisms Restricted Repetitive Behaviors

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Abstract

Restricted repetitive behaviors (RRBs) are the most characteristic behaviors of autism spectrum disorder (ASD). The clinical diagnosis and treatment of RRBs are extremely difficult resulting from its complex and variable etiology, highly heterogeneous clinical manifestations influenced by multiple factors (sleep quality, gastrointestinal health, age, and gender), lack of precise diag-

nostic criteria and low effectiveness of current clinical interventions. Therefore, clarifying the pathogenesis of RRBs is crucial for advancing clinical approaches. The occurrence of RRBs is thought to be influenced by both genetic and environmental factors, recent studies show sensory sensitivity of ASD is also related to RRBs. Our clinical study found that RRBs in ASD do not simply decline with age, ASD showed significant sensory gating P50 deficits, but RRBs showed no significant link to sensory gating; animal pre-pulse inhibition (PPI) experiments showed that the auditory pathway is normal. However, the PPI of the autistic rats was significantly reduced, that is, the autistic rats had defects in the inhibition of response amplitude, and maybe there were obstacles in the high-level regulation process of the brain. Cognitive flexibility is closely tied to RRBs severity. We further found that the frontal lobe function of autistic rats was insufficient, and the functional connection between the frontal lobe and the striatum and the hippocampus was reduced during the cognitive flexibility related test paradigm. These results indicate that RRBs are regulated by advanced brain center, in which the frontal-striatal circuit may play a crucial role, and these findings also provide new ideas for future therapeutic interventions.

Developing a Neuro-educational Model for the Inclusion of Children with Autism in Africa

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Abstract

In African contexts, educational challenges are often compounded by infrastructural deficits, socioeconomic disparities, and cultural perceptions, and developing a neuro-educational model (NEM) for the inclusion of autistic children is both necessary and complex. With the increasing shift to inclusive education NEM is important for addressing the multifaceted needs of children with neurological disorder that may interfere with children's learning, such as autism. NEM integrates neuroscience and educational practices to tailor learning environments, curricula, and teaching methodologies to suit the diverse neurological profiles of learners. This study introduces a NEM which draws from neuroscience of autism condition to create an adaptive education that supports cognitive, social, and emotional development of autistic students in schools. NEM ex-rays brain structures and functions that are distinct in autistic individuals, enabling educators and policymakers to design interventions that better support their needs in the classroom. The model emphasizes early diagnoses and intervention, individualized education plans (IEPs), and the use of neuro-educational strategies, such as differentiated instruction and multisensory learning. The study also highlights the cultural and systemic barriers to inclusion in Nigeria and proposes a framework for implementing the model within the existing educational infrastructure. This approach aims to enhance educational outcomes for autistic children, ensuring that they receive equitable access to quality education and fostering a more inclusive society. A NEM for the inclusion of children with autism in Africa can bridge the gap between the neurological needs of autistic children and the educational system, promoting equal opportunities for learning.

Engineering Human Neurons: Building Preclinical Models to Study Treatments for Mental Disorders

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Abstract

Autism spectrum disorder (ASD) is characterized by behavioral changes, including altered social patterns. ASD is often associated with coexisting conditions, including epilepsy, intellectual disability, and hyperactivity. Mutations affecting neuronal communication confer risk for ASD and offer possible therapeutic targets. However, the genetic heterogeneity of ASD is enormous, and transcriptional regulators, such as CHD8, have been associated with ASD. Of the 91 gene regulators most strongly associated with ASD, the transcription repressor MYT1L is unique due to its specific and continuous expression in neurons. We found that MYT1L deficiency in human neurons and mice caused neurodevelopmental delays with thinner cortices, behavioral phenotypes, and gene expression changes that resemble patients. MYT1L deficiency also causes upregulation of the cardiac sodium channel, SCN5A, and neuronal hyperactivity, which could be restored by shRNA-mediated knockdown of SCN5A or MYT1L overexpression in postmitotic neurons. Acute application of the sodium channel blocker, lamotrigine, also rescued electrophysiological defects *in vitro* and behavior hyperactivity *in vivo*. Here, we translate our findings for the first time to patient-derived preclinical models. Despite exhibiting differences in gene deregulation, we discovered that distinct mutations converge on electrical hyperactivity phenotypes in patient iPSC-derived neurons. Importantly, we found that these phenotypes can be normalized using lamotrigine. Strikingly, we could normalize social behavior alterations in our MYT1L mouse model. These findings open up new possibilities for patients, which we are currently translating to the clinic. Our work showcases how

stem cell technologies allow studying mental disorders in human neurons to decipher novel disease-causing mechanisms and translational strategies.

Profiling α -synuclein in Parkinson's Disease

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Abstract

Parkinson's disease (PD) is featured by the selective loss of nigral dopamine neurons and the proteinaceous aggregations of α -synuclein (α Syn) in the broad brain regions as Lewy pathology. The demonstration of clinical phenotype is associated with the neuronal dysfunction caused by the underpinning spatial occurrence and degree of Lewy pathology since the prodromal stage. Whereas disease trajectory is presumed under the impact of pathological species of α Syn, post-translational modifications are known to contribute to the formation and spread of pathological α Syn. However, the major post-translational modifications and pathogenic mechanisms are unknown yet to be explored. We question whether post-translational modifications of α Syn differ between wild-type (sporadic) and SNCA mutation PD cases and whether other non-neuronal cells also develop aggregations with unique post-translational modifications. By exploring these histopathology changes in post-mortem human brain tissues and reviewing the reports from previous publications, we propose the dominant forms of pathologic α Syn in the neurons and glia of the ventral midbrain and cortical regions, two representative loci responsible for motor and non-motor symptoms but have opposite degree of neuronal loss in PD. Our proposal may indicate the matrix of antibodies against different epitopes of α Syn, which can potentially predict disease progression. With α Syn being the key target for treatment and early diagnosis of PD, exploring the pathological profile of α Syn will bring value to reduce the side effects of protein-blocking treatment and enhance diagnostic accuracy, providing hope and optimism for the future of PD diagnosis and treatment.

Mature B-cell Lymphoma with Acute Myelitis as the First Presentation: A Case Report and Literature Review

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Abstract

We presented two patients with acute vertigo whose infarction located in posterolateral superior basal ganglia. We suspect that this infarction zone may involve the ascending and descending fibers of the vestibular cortical network. And it is possible that the lesion directly impaired the pathways of cerebellum to parietal and insular cortices through the thalamus or the related ascending and descending association fibers. Meanwhile, DTI showed that the lateral fibrous tracts were slightly sparse, which suggested that the lesions may involve corticocerebellar tract, inferior fasciculus fronto-occipital, arcuate fasciculus, corpus callosum and their related ascending and descending association fiber. Therefore, this explained why the symptoms of patients did not improve significantly in a short time after active treatment.

Effects of Health Qigong Exercise on Sleep Quality in Patients with Parkinson's Disease

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Abstract

Background and Purpose: Parkinson's disease (PD) is a common neurodegenerative disorder among the elderly. Sleep disturbances, as one of the more prevalent non-motor symptoms in PD patients, are closely associated with mortality rates related to the disease. Research conducted abroad has shown that the incidence of sleep disturbances in PD patients ranges from 60% - 98%. Therefore, this study aims to investigate the intervention effects of health qigong on sleep in Parkinson's disease patients.

Methods: A total of 23 participants who met the inclusion criteria were recruited, were randomly divided into the experimental group (n = 12) and the control group (n = 11). Both groups received standard pharmacological treatment, the experimental group additionally received 12 weeks of health qigong exercise, three times a week, lasting 60 min each time; the control group maintained a normal lifestyle with no exercise intervention, before and after the experiment, all people in both groups underwent test of Parkinson's disease sleep scale.

Results and Discussion: Before and after the experimental intervention, After the intervention, the PDSS scores of the two groups were higher than before ($p < 0.05$), and the test group was significantly higher than the control group ($p < 0.05$).

Conclusion: Health qigong can improve sleep disorders and sleep quality in patients with PD.

Exploring the Clinical and Genetic Spectrum of Distal Arthrogryposis Type 7

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Abstract

Distal arthrogryposis (DA) is a group of genetically heterogeneous congenital diseases characterized by non-progressive contractures predominantly distal joints of the extremities. Almost all products of known 11 identified genes are expressed in the structures of the neuromuscular system, which makes it possible to classify DA as a neuromuscular disease. Type 7 DA is a rare autosomal dominant disease characterized by two main symptoms: trismus and pseudocampodactyly, a specific symptom of limited mobility of the interphalangeal joints during hand dorsiflexion with no restriction during palmar flexion. In all patients described in the literature from different populations with type 7 DA, the same pathogenic variant c.2021G > A (p.Arg674Gln) was found in the MYH8 gene, the protein product of which is one of the myosin isoforms functioning in the embryonic period and providing the formation of muscle fiber structures. The aim of the presentation is to describe the clinical and genetic characteristics of four Russian patients with type 7 DA from two families. The patients underwent clinical examinations, electromyography, muscle MRI and molecular genetic analysis. As a result, in one family with clinical manifestations of type 7 DA, a heterozygous c.2021G > A (p.Arg674Gln) variant in the MYH8 gene, which was previously described in all patients published in the literature, was detected. In another family novel heterozygous c.2020T > C (p.Arg674Trp) variant was identified. Specific clinical signs of type 7 DA, combined with the presence of a recurrent nucleotide variant, make it possible to optimize the process of molecular genetic diagnosis of this type of hereditary arthrogryposis.

Investigation of Comparative Nonword Repetition Performance in Multiple Sclerosis: Group Differences, Subtype Variations, and Severity Effects

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Abstract

The study investigated nonword repetition (NWR) tasks in individuals' multiple sclerosis (MS) compared to healthy controls (HC), focusing on phonological working memory (WMP), speech perception, and short-term memory. Significant differences were found in NWR accuracy (NWRacc) rate between MS subgroups and HC ($H = 48.2$, $p < 0.001$). NWRacc decreased as the number of syllables increased in both groups, indicating increased cognitive load. All MS subtypes showed lower NWRacc compared to HC across varying syllable lengths (Mann Whitney U Test: two syllables $U = 64.5$, $p < 0.001$; three syllables $U = 183$, $p < 0.001$; four syllables $U = 248$, $p < 0.001$; five syllables $U = 283.5$, $p < 0.001$). However, no significant differences were found within MS subtypes based on syllable length. NWRacc did not differ between mild and severe MS groups. Overall, the NWR test effectively assessed WMP in MS, highlighting its utility in diagnosing and addressing language-cognitive challenges in individuals with MS. This underscores the importance of tailored intervention strategies to mitigate these challenges.

Barking Up the Wrong Tree-motor-sensory Elements as Prodrome in Autism

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Abstract

Autism spectrum disorder (ASD) has been intensely investigated since the term was first used over 80 years ago. The prevalence of ASD is constantly rising, and currently, 1:36 children are diagnosed with this disorder. Despite intense interest in ASD, the origins of this disorder remain obscure. This article explores motor issues and proprioceptive interoception difficulties as the prodrome of ASD. The importance of early intervention in the prognosis of ASD is common knowledge. Yet, since the communicational and social behaviors typical of ASD are observable only after the age of 18 months, diagnosis and early intervention are delayed. Therefore, the quest into the involvement of sensory-motor difficulties as a source of ASD traits, or at least as a

potential early indicator, is warranted, with the intention of enabling early diagnosis and early intervention. The present lecture examines the justification for this new avenue of early diagnosis and intervention and may open up a completely different way of viewing ASD as a motorically oriented disorder. This new point of view may suggest an original path of assessment and intervention in early toddlerhood with this group of clients, possibly leading to improved prognosis for children and their families.

Alexithymia and Illness Perceptions in Persons with Multiple Sclerosis and Their Partners: A Dyadic Analysis

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Abstract

This talk will disseminate the findings of a research study conducted in the context of a PhD program. The aim of the study was to investigate the association between alexithymia (difficulty in understanding and expressing feelings) and illness perceptions (opinions regarding a disorder) among persons with multiple sclerosis (PwMS) and their partners, as well as within the dyads composed of PwMS and partners. All participants were assessed in terms of illness perceptions and alexithymia. A dyadic data analysis was performed to test the effect of alexithymic traits both on a person's own illness perceptions (actor effect) and on the partner's illness perceptions (partner effect). Both at the individual and dyadic level, higher alexithymic traits related to more negative illness appraisals. Our findings may inform therapeutic interventions aimed at reducing alexithymic traits, which in turn may reduce negative, and potentially dysfunctional, illness perceptions.

UBOs and Neurological Involvement in Neurofibromatosis Type 1

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Abstract

Background: Unidentified bright objects (UBOs) have been detected as areas of T2-weighted hyperintensities on brain MRI in 43% - 93% of children with neurofibromatosis type 1 (NF1). These hyperintensities seem to have a relation with cognitive impairment, another common finding in NF1.

Objective: To report on a possible correlation between cognitive impairment of NF1 patients and the presence of UBOs.

Materials and Methods: we've retrospectively reviewed brain MRIs and neuropsychological reports of 17 patients with NF1 (mean age 9.7 ± 4.7 SD) and extracted data from a range of cognitive and behavioral tests, focusing mainly on Wechsler scales of intelligence (WPPSI-III, WISC-III, WAIS-R).

Results: The mean full scale intellectual quotient (FSIQ) was within the low average range (84 ± 14.54); only two patients (11.76%) had a diagnosis of intellectual disability (ID). 64.7% of the patients presented a clinically relevant discrepancy between verbal and performance intellectual quotient, in favor of the latter ($VIQ < PIQ$). The mean number of UBOs for patients was 3.94 ± 1.76 . Pearson's correlation revealed an inverse relationship between the total number of UBOs and FSIQ, although without statistical significance. The cerebellum was the region most frequently affected by UBOs (70.6%). Patients with UBOs in the cerebellum or brainstem presented significantly higher mean PIQ than patients with UBOs elsewhere, while the two patients with ID had both UBOs in the thalamus.

Conclusions: Our results suggest that the location and the number of UBOs could influence the cognitive profile of NF1, even if further investigations with larger study samples are warranted.

Successful Outcomes with Early Intervention by Combined Comprehensive Approach in Treatment of Autism: A 10 Year Retrospective Study

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Abstract

This is a retrospective study of outcomes in all children with autism who received early interventions between 15 months to 3

years of age with combined protocol treatment. The study also compares the percentage of children getting similar outcomes in age groups >3 - 5 years, >5 - 10 years, and >10 years age groups. The relation of outcomes to severity of autism at the time of diagnosis was also assessed. It was found that the children who received early intervention with combined pharmacological, applied behavior analysis (ABA), occupational therapy (OT), speech and nutritional therapy were closer to neurotypical than those who received only ABA, OT, speech and 1:1 IEP. Patients were classified according to age with assessment of maternal risk factors, birth complication, media exposure, family history, age at interventions, lab studies and imaging studies. Patients with chromosomal anomalies, seizure disorder and other medical or neurological disorders were excluded from study. The most important pre-determinants for successful outcomes were age of intervention, parental counselling, parental acceptance and regular follow up with a focal person who could counsel and direct other disciplines. For determining the long-term outcomes, the longest period of follow up was 10 years and shortest period 2 years.

Statin Induced Neurological Dysfunction

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Abstract

3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase (HMGR) inhibitors, commonly known as statins, are drugs frequently used in the treatment of hypercholesterolemia and hyperlipidemia. However, the current study has demonstrated that simvastatin induces neurotoxicity is associated with cellular coenzyme Q₄₀ (CoQ₄₀) depletion. CoQ₄₀ has an important role in the mitochondrial depletion electron transport chain (ETC), in addition to being a fundamental lipid-soluble antioxidant. Depletion of CoQ₄₀ is frequently associated with impaired mitochondrial function and increased oxidative stress. The aim of this study was to investigate the potential mechanisms of simvastatin induced neurotoxicity assessing mitochondrial function and evidence of oxidative stress in an *in vitro* SH-SY5Y human neuronal cell line. Fluorescence studies assessed via flow cytometry determined significant increases in intracellular and mitochondrial reactive oxygen species production following SH-SY5Y treatment with simvastatin compared to control cells. Additionally, spectrophotometric enzyme studies determined a significant ($p < 0.0001$) inhibition of ETC complex I and II-II activities which accompanied a significant decrease in neuronal CoQ₄₀ content ($p < 0.005$) and cell viability ($p < 0.0001$). The results of the present study have indicated evidence of mitochondrial dysfunction and increased oxidative stress, resulting in increased loss of neuronal viability following simvastatin treatment. Thus, these results demonstrate evidence of neurotoxicity associated with statin therapy.

Multi-disciplinary Approach to Evaluate the Efficacy of Combined Treatment of UbisolQ10 and Ashwagandha in a Transgenic Mouse Model of Alzheimer's Disease

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Abstract

Introduction: Alzheimer's disease (AD) has become one of the largest global health challenges, with no cure at present. Although pharmaceuticals may help alleviate some of the symptoms, their long-term use can be harmful due to potential side effects and a certain level of toxicity. Therefore, this research aims to investigate a multi-modal approach to evaluate the efficacy of a novel nutraceutical treatment, which includes UbisolQ10 (UQ) and ashwagandha root extract (ASH) in a transgenic (5xFAD) mouse model of AD. The goal is to analyze a set of behavioral, neurostructural, and biochemical parameters to gain an overall picture of the effectiveness of this intervention.

Motivation and Significance of Study: This comprehensive project embraces a broad range of scientific fields, which incorporate magnetic resonance imaging (MRI), spatial and non-spatial recognition tasks, and immunohistochemical analyses. UQ, a water-soluble formulation containing Coenzyme Q10 (CoQ10), has shown promise in mitigating neurodegeneration, while ASH is known for its potential in clearing amyloid-beta plaques. UQ is superior to its oil-soluble ancestor, CoQ10, because of its increased bioavailability of lipophilic compounds while it also maintains CoQ10's ability to cross the blood-brain barrier. Leveraging these properties, the study aims to target several complex mechanisms underlying AD progression.

Methods: To evaluate the efficacy of the combined treatment, 24 female and 24 male mice divided into three treatment groups (untreated wildtype, untreated transgenic, and treated transgenic) will be used as subjects. MRI scans will be repeated three times throughout the 11 month lifespan of mice, accompanied by sequential behavioral evaluations using novel objects and novel location recognition tests in a plus-shaped radial maze. Using advanced MRI techniques, longitudinal monitoring will be applied to structural changes in the hippocampal and entorhinal regions, as well as severity and longevity of neural

inflammation. Lastly, post-mortem biochemical analysis will delve deeper into molecular mechanisms in AD pathophysiology, as well as evaluating the preservation or non-preservation of such mechanisms and validate antemortem findings.

Conclusion: AD is complex and multi-mechanistic; thus, it is important to take into consideration multiple different perspectives when analyzing the efficacy of a medical intervention. This collaborative research study provides an in-depth look into non-toxic remedies that may stop the progression of AD.

In-vitro Model of Subcortical Alzheimer's Disease and Neuroprotective Effect of Selective Serotonin Reuptake Inhibitor

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Abstract

Tauopathies: Besides its anti-depressive and anxiolytic effects, selective serotonin reuptake inhibitor (SSRI) treatment also offers intracellular modifications that may help improve neurogenesis, reduce amyloid burden and Tau pathologies, and neuroinflammation in Alzheimer's disease (AD). Despite its multifaceted impact on the brain, the exact physiological and molecular mechanism by which SSRIs such as citalopram improve neurogenesis and synaptogenesis in dementia is poorly understood. In the current study, we investigated the protective role of SSRI, citalopram, in serotonergic and medullary raphe neurons (RN46A-B14). RN46A-B14 cells were transfected with wild-type and mutant APP and Tau cDNAs for 24 h and then treated with 20 μ M Cit for 24 h. We then assessed mRNA and protein levels of pTau, total Tau, serotonin-related proteins such as TPH2, SERT, and 5HTR1a, synaptic proteins, and the cytoskeletal structure. We also assessed cell survival, mitochondrial respiration, and mitochondrial morphology. The mutant APP and Tau transfected cells showed increased serotonin-related proteins and mRNA levels, while synaptic proteins' mRNA and protein levels were downregulated. Citalopram treatment significantly reduced pathologically pTau levels along with serotonin-related protein levels. On the other hand, there was a significant increase in the mRNA and protein levels of synaptic genes and cytoskeletal structure in the treated groups. Further, citalopram also improved cell survival, mitochondrial respiration, and mitochondrial morphology in the treated cells that expressed mAPP and mTau. Taken together, these findings suggest that citalopram could not only be a promising therapeutic drug for treating patients with depression but also for AD patients.

Brain Rejuvenation via by High Tech Products from Quantum Chinese Medicine

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Abstract

Brain rejuvenation is one of the most active studies of the brain in Western medical schools. It is a complex challenge. There are many proposed therapies. For example, 1. Stem cells are used to regenerate damaged brain tissue and stimulate neurogenesis. 2. Resveratrol is a compound found in red wine and certain plants. its neuroprotective properties may help in reducing inflammation, promoting neurogenesis, and improving cognitive function in aging brains. 3. Fasting and caloric restriction is a compound found in red wine and certain plants. It may help in reducing inflammation, promoting neurogenesis, and improving cognitive function in aging brains. Researchers are still in early phases of clinical trials for many of these therapies. We report successful cases of applying solutions with solid water particles that activate the brain rejuvenation process. The brain rejuvenation can be observed by infrared imaging system. A healthy 56 year old male with bald head was the subject of study. First, the infrared pictures of his head were taken that showed the temperature distribution on the surface of the head. Then he drank 60 ml Xenwater, and waited 15 min. Another set of infrared pictures were taken on the head. Then compared the two sets of pictures. The temperature reduction of the brain was shown, as below (Figure 1). The color code of the infrared picture is white the hottest (about 35 °C), then red not so hot (34 °C), then yellow (33 °C), and healthy green (32 °C). Many more cases will be presented. Feasible explanation of brain rejuvenations will be given. Xenwater was classified as a dietary supplement. Six FDA tests showed that Xenwater does not cause any harm.

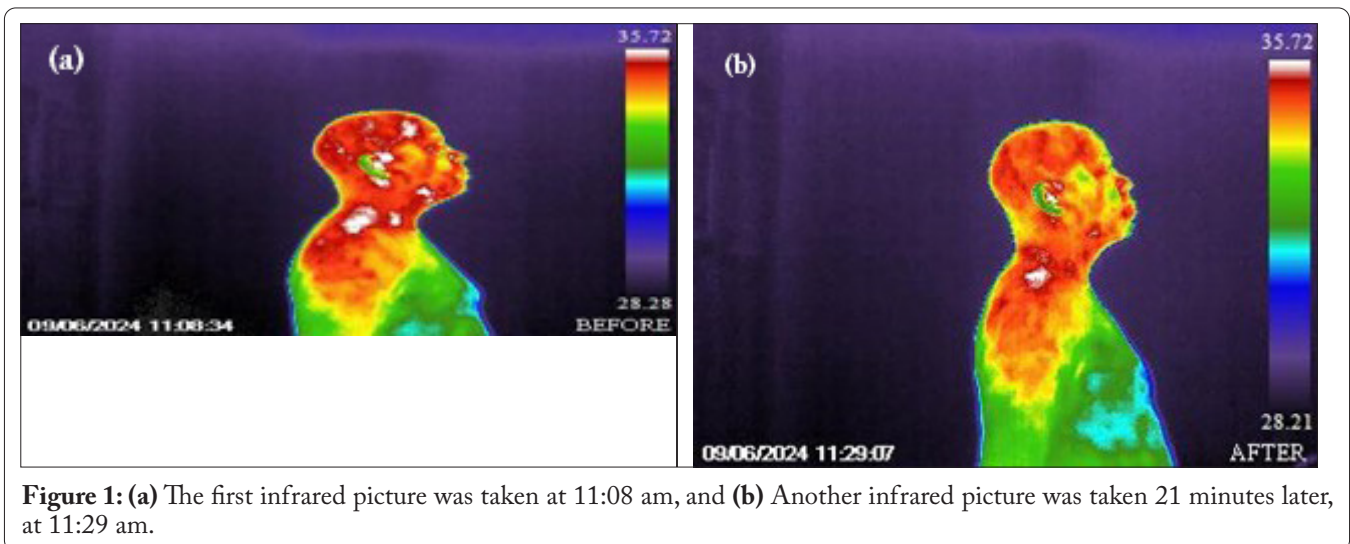


Figure 1: (a) The first infrared picture was taken at 11:08 am, and (b) Another infrared picture was taken 21 minutes later, at 11:29 am.

Parent's Experience of the Diagnosis of Autism Spectrum Disorder

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Abstract

This presentation explores the experiences of parents during the initial stages of their child's autism spectrum disorder (ASD) diagnosis, with a focus on how clinicians can support families. Through thematic analysis of qualitative data, four key themes emerged: understanding, acceptance, emotion, and coping. While many parents demonstrate resilience and proactive coping strategies, a subset faces challenges that may hinder their ability to support their child's treatment journey. This session will discuss how clinicians can address these challenges by fostering resilience, enhancing understanding, and promoting acceptance. Attendees will gain insights into the emotional and cognitive processes parents undergo, and how to provide targeted interventions to strengthen family dynamics and improve outcomes for children with ASD. This presentation emphasizes the importance of integrating family-centered approaches within multidisciplinary care teams to optimize the well-being of both the child and the family.

Relationships of Angiogenesis, Fatigue, and Walking Disability in Multiple Sclerosis

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Abstract

Multiple sclerosis (MS) is a chronic, immune-mediated neurological disease with fatigue and walking disability being the most common and debilitating MS symptoms. MS onset and relapse are associated with angiogenesis, the formation of the new blood vessels. Vascular endothelial growth factor (VEGF) is a potent pro-angiogenic factor that is elevated in the blood, cerebrospinal fluid, and nervous tissue of persons with MS (PwMS). To investigate the relationships between angiogenesis, walking disability, and fatigue, we recruited PwMS and non-MS controls for a single-center cross-sectional study. Participants completed surveys on demographics and fatigue (PROMIS-Fatigue_{MS}). For PwMS, timed 25-foot walk (T25FW) scores were collected and serum VEGF was assayed using ELISA. Statistical analyses included parametric (Pearson correlation, Unpaired t-test) and nonparametric (Spearman rank correlation, Mann-Whitney) tests with statistical significance determined at $p < 0.05$. 60 PwMS and 16 non-MS controls returned surveys. PwMS had significantly greater PROMIS-Fatigue_{MS} scores than controls ($p = 0.02$). Fatigue was not correlated to age or length of disease (LOD). In PwMS, fatigue was weakly positively correlated with serum VEGF ($n = 16$) (Pearson $r = 0.28$, $p = 0.3$). Fatigue was not correlated to T25FW in PwMS ($n = 55$). Additionally, T25FW and serum VEGF were not correlated in PwMS ($n = 55$). When PwMS were separated into "high fatigue" ($n = 43$) and "low fatigue" ($n = 15$) groups, those with high fatigue had significantly worse T25FW scores ($p = 0.04$). We found that VEGF more likely contributed to fatigue than age and LOD in PwMS. Our findings support PwMS experiencing greater fatigue than non-MS individuals. Tonic angiogenic activity may underlie fatigue in PwMS, and anti-angiogenic treatments may provide an avenue to reduce fatigue and improve quality of life in MS.

Diverse and Heterogeneous: Often Othered, People with Disabilities Defy Expectations

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Abstract

This essay explores the historical and current experiences of people with disabilities. I consider the tradition and the moral force of conformity to norms—about those who fit and those who defy them—and contradictions of faith in a creative god of eccentricity, present in the diversity of all humankind. Then, turning to the disabled god, I consider the implications of scandal against the imago Dei in discrimination, othering, and violence that people with disabilities continue to experience is engaged. People with disabilities defy the expectations of homogeneity assumed in the development and imposition of norms and standards, which few people ever attain. As a result of their embodied/en-minded diversity people with disabilities are often othered and stigmatized. The UN Convention on the Rights of Persons with disabilities recognizes “that disability is an evolving concept [today] and that disability results from interaction between persons with impairments and attitudinal and environmental barriers that hinders their full and effective participation in society on an equal basis with others” (Preamble, 5). CRPD signatories have resolved “to promote, protect and ensure the full and equal enjoyment of all human rights and fundamental freedoms by all persons with disabilities, and to promote respect for their inherent dignity” (Art 1 - Purpose). In the first World Report on Disability, the World Health Organization notes the diversity of gender, age, socioeconomic status, sexuality, ethnicity, (and) cultural heritage among people with disabilities, and that “the disability experience resulting from the interaction of health conditions, personal factors, and environmental factors varies greatly”. Disability studies literature, led by scholars with disabilities and their allies, confirms that the experience of disability is multifarious; the experience of disadvantage is likewise multidimensional. Disability studies challenge the historical trajectory of how disability has been portrayed in religious and philosophical texts, in mytho-poetic and classic literature, in scientific and medical approaches, in legal and policy determinations, and in hermeneutic assumptions of structural and social conditions that have othered/set apart people with disabilities from a presumed main. These histories include depictions of people with disabilities –of people with sensory limitations, mobility impairments, intellectual, developmental, and/or behavioral disabilities—as deviant. Such depictions alternately prize normativity, regularity, and symmetry contra, i.e., discriminating against diversity and heterogeneity. As a result, people with disabilities have been and continue to be othered by the nondisabled-normate, suggesting discomfort with the kaleidoscopic diversity of creation among the keepers of authoritative norms. Sadly, as a result of this discomfort and in opposition to the remedial efforts of the civil rights and Americans with disabilities acts, disability discrimination remains considerably widespread nationally and internationally. This essay presents a review of the historical evidence regarding people with disabilities giving particular attention to the Judeo-Christian scriptures and the Christian theological tradition. After establishing the tradition of norms—about those who fit and those who defy normative expectations—and the authority and power to enforce them, the paper examines the moral force of conformity and the contradictions that conformity presents to faith in a god of eccentricity who creates a universe teeming with diversity and heterogeneity, inclusive of humankind. Then I consider the implications of scandal concerning overt discrimination and covert violence (e.g., medical practitioners’ failure to acknowledge bias against people with disabilities re: autonomy, beneficence, maleficence, and justice) that people with disabilities continue to experience.

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