NEURO 2024

INTERNATIONAL NEUROSCIENCE CONGRESS

28-30 NOVEMBER, 2024

IVANE JAVAKHISHVILI TBILISI STATE UNIVERSITY

TBILISI, GEORGIA

ABSTRACTS BOOK

INTERNATIONAL NEUROSCIENCE CONGRESS (NEURO-2024)

CONGRESS HIGHLIGHTS:

The NEURO-2024 Congress with the slogan "Unlocking the Mysteries of the Brain" was held at Ivane Javakhishvili Tbilisi State University on 28-30 November 2024 in conjunction with the 5th Annual Meeting of International Center for Neuroscience Research in Georgia. The main topics recent advances in brain disorders and therapy, neurotechnology and new discoveries in neuroscience and neurosurgical innovations: 21st century challenges and solutions for functional neurosurgery are discussed. The congress was held very successfully, about 150 participants and many national and international speakers from different countries participated and talked on the interesting panels. The programs were included keynote lectures by leading scientists, Workshop, Q&A panels, and Oral/poster presentations by participants.

November 28-30, 2024
Ivane Javakhishvili Tbilisi State University
Tbilisi, Georgia

ABSTRACTS

Abstract ID: 1011

CRISPR and Brain Disorders: A Frontier in Therapeutic Advances

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ABSTRACT

CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats) in-novation has revolutionized hereditary inquire about, advertising uncom-mon exactness in genome altering. This paper investigates the application of CRISPR in understanding and possibly treating brain disarranges, counting neurodegenerative illnesses, neurodevelopmental clutters, and brain cancers. We talk about the most recent headways, current chal- lenges, moral contemplations, and future bearings of CRISPR-based treatments in neuroscience. Brain disorders, enveloping neurodegenerative maladies such as Alzheimer's and Parkinson's, neurodevelopmental disarranges like autism spectrum disorder (ASD), and brain cancers, speak to a few of the fore- most complex and weakening conditions influencing humankind. These clutters not as it were force a noteworthy burden on patients and their fam-ilies but too display significant challenges to the healthcare framework due to their persistent nature and the current need of successful medications.

Keywords: Autism spectrum disorder, CRISPR, Therapeutic advances

Abstract ID: 1012

Innovative neuroanatomy education and its clinical impact: A Prospective Cohort Study on Peripheral Nerve Surgery Outcomes at KFH Rwanda

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ABSTRACT

Background: Peripheral nerve injuries present significant challenges in neurosurgery, requiring advanced surgical techniques and precise anatomical knowledge. This study explores the impact of innovative neuroanatomy education on the outcomes of peripheral nerve surgeries at King Faisal Hospital (KFH), Rwanda. Our focus is on the application of cadaveric anatomical dissection and Anatomage technology to improve surgical precision and patient outcomes. **Methods:** This ongoing prospective cohort study evaluates patients undergoing peripheral nerve surgery at KFH. Cases are monitored with follow-ups on Day 3, Day 30, Day 90, 6 months, and 12 months' post-surgery. Neurophysiological assessments including NCS/EMG are performed at 1 month, 3 months, 6 months, and 12 months. The study includes five cases with various peripheral nerve injuries: Cases of brachial plexus avulsion, ulnar nerve repair with sural nerve graft, and neurotization procedures. **Results:** Initial findings from the five cases reveal diverse but promising outcomes. Case 1 involved a 40-year-old male with a brachial plexus injury who underwent DREZ-otomy for pain management followed by Neurotization of the Musculocutaneous Nerve with 3rd and 4th Intercostal Nerve Transfers 1 year and 4 months' post Trauma, showing reduced pain post DREZ-otomy but persistent motor deficits. Case 2 featured a 52-year-old male with traumatic ulnar nerve injury and Median nerve entrapment between 2 heads of pronator teres, who had significantly improved motor function post forearm Surgical Exploration, median nerve neurolysis, and Ulnar Nerve Repair with Sural Nerve Graft (e.g., OPB muscle power of 0/5 preop to 3/5 post-op). Case 3 was a 30-year-old male with brachial plexus avulsion, treated Neurotization of Spinal Accessory Nerve to Suprascapular Nerve and Oberlin technique, demonstrating partial recovery of motor functions. Case 4, a 31-year-old male with a supraclavicular brachial plexus schwannoma, achieved gross total resection with preserved motor sensory and neurophysiological functions. Case 5 features a 63-year-old male who came with intractable right upper limb pain not responding to medical management, post 8 years of having total brachial plexus injury due to RTA. He benefited from C5-T1 DREZotomy for his pain management. **Conclusions**: Innovative neuroanatomy education utilizing cadaver dissection and Anatomage technology significantly enhances surgical planning and execution. Early results suggest improved outcomes in nerve repair and transfer procedures. Continued follow-up and analysis will further elucidate the long-term benefits of these educational advancements in peripheral nerve surgery.

Keywords: Peripheral nerve surgery, neuroanatomy education, Anatomage, brachial plexus injury, nerve repair, neurosurgery, surgical outcomes, King Faisal Hospital, Kigali, Rwanda, Africa.

Abstract ID: 1013

Breaking (Blood Brain) Barriers: Harnessing Extracellular Vesicles for Targeted CRISPR/Cas9 Therapy in Neurological Disorders

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ABSTRACT

Huntington's disease (HD) and amyotrophic lateral sclerosis (ALS) are neurological disorders with massive treatment challenges owing to their genetic underpinnings and the restrictive nature of the Blood Brain Barrier (BBB). The advent of CRISPR/Cas9 gene editing technology brings new promise to target the genetic mutations underlying these disorders. However, efficient delivery of CRISPR/Cas9 into the central nervous system (CNS) still remains a formidable obstacle. This review focuses on the emerging potential of extracellular vehicles (EVs), especially exosomes, as novel delivery vehicles of CRISPR/Cas9 in the treatment of neurological diseases. EVs have become one of the potentially important drug delivery systems due to their intrinsic ability to cross the BBB, low immunogenicity and natural origin. Recent developments in this field have been able to show that EVs can successfully encapsulate and deliver CRISPR/Cas9 components into target cells within the CNS. Notably, researchers successfully demonstrated the use of engineered exosomes to deliver CRISPR/Cas9 systems targeting the mutant huntingtin (mHTT) gene in a mouse model of HD. They demonstrated a significant reduction of the mHTT protein and substantial improvement in motor function. Chinese scientists also researched the use of EVs to deliver the CRISPR/Cas9 for the SOD1 mutation in the SOD1-ALS mouse model, where they were able to show a decline in SOD1 aggregation with delay of disease progression. These benefits of EV-mediated CRISPR/Cas9 delivery concern increased stability of gene-editing components, higher cellular uptake, and lower off-target effects compared to traditional viral vectors. In addition, EVs can be engineered to display specific surface proteins for targeted delivery to specific cell types, such as neurons or glial cells. With these features, EVs bear promise as a versatile platform for CNS-targeted gene therapy. Notwithstanding these promising findings, several obstacles must be surmounted. Among the major topics that still require future investigation, the optimization of the EV loading techniques comes first, followed by scaling up the production of EVs for clinical use and assessment of their long-term safety. Finally, immune responses against repeated EV administrations could potentially be elicited; thus, this should be considered with utmost attention. We look forward to future studies that will develop tissue-specific targeting strategies, combination therapies coupling CRISPR/Cas9 with neuroprotective agents, and the development of next-generation gene-editing tools such as base editors or prime editors that offer higher precision. By exploiting the natural properties of EVs to cross the BBB and by enabling targeted delivery to specific CNS cell types, several limitations that exist in most CNS gene therapies so far can be overcome. Further development in this arena may create revolutionary treatments for neurological diseases that were previously considered intractable.

Keywords: Huntington's disease, Amyotrophic lateral sclerosis, CRISPR/Cas9

Abstract ID: 1016

Memorization Techniques for Cognitive Health: A Preliminary Study on Mitochondrial Function in the Indonesian Muslim Population through Qur'an Memorization

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ABSTRACT

Dementia is a neurodegenerative disorder that can significantly impair cognitive function and behavior, affecting millions worldwide. According to the Global Burden of Disease Study 2019, there are 57.4 million cases globally, including 4.9 million in Indonesia. One crucial factor in memory retention and neuronal health is Brain-Derived Neurotrophic Factor (BDNF) protein, which plays a significant role in neuroplasticity. BDNF levels can increase rapidly through cognitive training, and studies have shown that such interventions can positively impact cognitive abilities, particularly in the elderly. Recent research has highlighted the bidirectional circulation of BDNF between the brain and peripheral blood, emphasizing its role in maintaining mitochondrial health, which is essential for cellular energy production. This connection between cognitive function and mitochondrial health suggests that interventions targeting BDNF could be beneficial in preventing cognitive decline. This preliminary study investigates the potential cognitive benefits of Qur'an memorization, specifically its impact on mitochondrial function in monocytes. The study focused on a group of Indonesian Muslim children who engaged in Qur'an memorization compared to a control group who did not. Monocyte cells were cultured from isolated Peripheral Blood Mononuclear Cells (PBMCs) to assess mitochondrial activity, which serves as an indicator of BDNF development and overall cellular health. The findings indicate a positive trend in the proportion of healthy monocytes and mitochondrial activity in the Qur'an memorization group. Although the statistical power was limited due to the small sample size, these results suggest that Qur'an memorization may enhance mitochondrial health and, by extension, cognitive function. This aligns with previous research that has demonstrated a positive correlation between BDNF levels and cognitive training. In conclusion, this study provides preliminary evidence that Qur'an memorization could serve as a cognitive training method with potential benefits for memory preservation and dementia prevention. However, the findings should be interpreted with caution due to the limited statistical power, and further research with a larger sample size is necessary to confirm these results. This study lays the groundwork for future investigations into the use of Qur'an memorization as an intervention for maintaining cognitive health, particularly in populations at risk of dementia.

Keywords: Dementia, Mitochondria, Monocytes, PBMC, Qur'an Memorization

Abstract ID: 1022

A microglia targeted therapy in neuroinflammation for Alzheimer's patients

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ABSTRACT

This integrated literature review explores neuroinflammation, a fundamental physiological process implicated in brain disorders, with a focus on its role in neurodegenerative diseases, particularly Alzheimer's disease (AD). Alzheimer's is marked by the accumulation of amyloid beta (Aβ) proteins in the cerebral cortex and neurofibrillary tangles (NFTs) in the hippocampus and basal ganglia. The review emphasizes the role of neuroinflammation and the involvement of microglia, which are essential for detecting environmental changes, clearing toxic agents, and defending against harmful stimuli. Persistent neuroinflammation, characterized by the activation of microglia, can become neurotoxic, particularly with aging and stress, and is closely associated with neurodegenerative conditions like Alzheimer's. Genome-wide association studies have highlighted the significance of innate immune-related genes, such as Apolipoprotein E (APOE), in neurodegenerative diseases. The APOE4 allele is identified as a major genetic risk factor for Alzheimer's, leading to earlier deposition of amyloid plaques, accelerated disease progression, and increased brain atrophy. In contrast, the APOE2 allele is linked to a decreased risk of Alzheimer's and a delayed onset. The APOE4 allele's association with heightened inflammation suggests it may influence Alzheimer's pathology by altering immune responses. Recent advancements in stem cell research, particularly regarding mesenchymal stem cells (MSCs), are also examined. Studies indicate that MSC-derived secretome can improve memory and neuronal health in Alzheimer's models by enhancing neuroinflammation resolution and facilitating plaque clearance via the release of soluble intracellular adhesion molecule-1 (Sicam-1), which stimulates neprilysin, an enzyme that degrades Aβ. The review includes meta-analyses of emerging drugs targeting microglia receptors, such as CSF1R inhibitors (e.g., PLX3397), propofol, and various antibodies, currently in clinical trials. Depleting microglia with CSF1R inhibitors has shown promise in reducing amyloid plaques and enhancing cognitive function. Additionally, research into TREM2 and CD33 modulation, gene-targeted therapies, and other pharmacological approaches aims to improve microglial function and alleviate Alzheimer's symptoms. Finally, the review discusses potential diagnostic markers and therapeutic interventions, including Sirtuin 1 (Sirt1), an anti-aging protein crucial for regulating appetite, neurodegeneration, and metabolism. Sirt1 interacts with anti-aging genes to affect neuron survival and circadian rhythms. Dysregulation of Sirt1 is associated with neurodegenerative diseases like Alzheimer's and Parkinson's. Caloric restriction and diets modulating bacterial lipopolysaccharides (LPS) may enhance Sirt1 activity, potentially improving Alzheimer's prognosis. Sirt1's effects on microglial function and neuroinflammation underscore its potential as a therapeutic target. The review concludes that addressing Alzheimer's disease effectively requires early intervention during the prodromal phase with new biomarkers, advanced imaging, and stem cell technologies. Future research should integrate systemic inflammation, immune function, and microbiome impacts to better understand disease progression and modify outcomes.

Keywords: AD: Alzheimer's disease, Aβ: Amyloid beta protein, NFT: Neurofibrillary tangles, Sicam-1: Soluble intracellular adhesion molecule-1,Sirt1: Sirtuin 1, LPS: Lipopolysaccharide

Abstract ID: 1023

Navigating epilepsy: breakthroughs in minimally invasive surgical techniques for managing refractory epilepsy

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ABSTRACT

Epilepsy is a chronic neurological disorder characterized by recurrent, unprovoked seizures, affecting an estimated 50 million individuals worldwide. First-line treatments, primarily Anti-Epileptic Drugs (AEDs), often fail to achieve complete seizure control in approximately 30% of patients. Furthermore, conventional surgical interventions, such as temporal lobe resections and corpus callosotomy, are frequently associated with significant side effects and complications. Many patients continue to require AEDs post-surgery, as these procedures do not necessarily eliminate seizures entirely. This review explores innovative minimally invasive surgical procedures that offer more definitive seizure control for both generalized and focal refractory epilepsies. The aim is to enhance patients' quality of life while reducing mortality and morbidity associated with the disorder. Neurostimulation devices including EASEE (Epicranial Application of Stimulation Electrodes for Epilepsy), and Picostim Neurotransmitter Deep Brain Stimulation are discussed, highlighting their associated risks and benefits. These devices involve minimally invasive surgery, to target epileptic foci in individuals with drug-resistant focal epilepsy and Lennox-Gastaut Syndrome, respectively. Additionally, Laser Interstitial Thermal Therapy (LITT) is examined as a promising future treatment for refractory focal and lesional epilepsy. LITT involves minimal surgical intervention, offering a safer alternative to corpus callosotomy and other disconnection surgeries by causing minimal brain tissue damage and enabling rapid patient recovery. Patients undergoing LITT can typically resume their usual activities within a week. Overall, these minimally invasive techniques represent significant advancements in the management of refractory epilepsy, providing hope for improved outcomes and enhanced quality of life for patients who do not respond to traditional treatments.

Keywords: Refractory epilepsy; Laser Interstitial Thermal Therapy (LITT); Epicranial Application of Stimulation Electrodes for Epilepsy (EASEE); Deep brain stimulation

Abstract ID: 1024

Effect of s-allyl-cysteine on Alzheimer's disease-like features in transgenic drosophila melanogaster

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ABSTRACT

Background: Alzheimer's disease (AD) is an age-related progressive neurodegenerative disease. AD is described by the loss of neurons in the hippocampus and cortex of the nervous system affects the loss of cognitive function, and memory, and eventually leads to the death of patients. The most prevalent neurodegenerative dementia AD, which affects ~30 million people worldwide is presently incurable. S-allyl-cysteine is nutraceuticals that have been used in Ayurvedic medicine for thousands of years and are known for their beneficial effects when used as medicinal and dietary constituents. The present study aimed to assess the neuroprotective effect s-allyl-cysteine in the Drosophila melanogaster model of Alzheimer's disease. **Methods and design:** Drosophila melanogaster (UAS > GMR-GAL4, UAS > ELAV-GAL4, Wild-type Canton-S) was cultured on a medium containing varying concentrations of 25 mg/kg, 50mg/kg, 75 mg/kg of S-allyl cysteine. The parameters measured include larva motility, negative geotaxis, and eye imaging test. **Result:** Treated flies (UAS > GMR-GAL4) and (UAS > GMR-GAL4) in base media express significant changes compared to the Wild-type Canton-S (Control group) in the eye imaging test, climbing, and larva motility assays. Untreated flies expressed a significant decline ($^*P < 0.05$) in climbing activity, larva motility, neurodegeneration, as well as pronounced ommatidial disarrangement when compared with wild-type (Control group). S-allyl-cysteine at 75mg/kg (high dose) increased locomotor activity of larva in a dose-dependent manner in UAS > ELAV-GAL4 flies, rescued amyloid-beta expressing GMR-GAL4 flies from ommatidial degeneration. **Conclusion:** The study shows the potential neuroprotective effect of s-allyl-cysteine in the treatment of Alzheimer's disease.

Keywords: Alzheimer's disease (AD), S-allyl-cysteine, GMR-GAL4, Neurodegenerative disease

Abstract ID: 1028

Neurotoxic effect of chronic manganese administration on motor and non-motor disorders inducing Manganism similar to Parkinson-like disease in male rats: possible involvement of oxidative stress

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ABSTRACT

Manganese (Mn), a natural heavy metal essential to human and animal survival, is notably involved in the metabolism of sugars, amino acids and lipids, it plays an important role in the nervous and immune systems, cellular energy regulation, bone and connective tissue formation, and the activation of certain enzymes in many other physiological mechanisms, but becomes toxic with chronic exposure, this toxicity is often associated with a disease presenting neurological symptoms similar to those of Parkinson's disease, known as manganism, since the central nervous system (CNS) is a target organ for Mn toxicity, probably due to its passage through the blood-brain barrier (BBB), causing its accumulation in neuronal structures such as the striatum, olfactory bulb, frontal cortex, prefrontal cortex and hippocampus, and inducing oxidative stress that is potentially responsible for neurodegeneration and the onset of symptoms. The aim of this study was to evaluate the neurotoxic effects of chronic Mn exposure, in order to understand this neurotoxicity, different groups of male Wistar rats were exposed to different doses of Mn (6 mg/kg, 25 mg/kg, 30 mg/kg and 40 mg/kg) by daily intraperitoneal injections for 12 weeks. The neurotoxic effects of Mn were assessed by tests of neuroaffective behavior, locomotion, motor coordination and olfaction. The rats were then euthanized and their brains extracted for analysis of affected nerve structures and measurement of oxidative stress. Chronic exposure to Mn produced adverse effects on the organism and especially on the central nervous system, causing irreversible damage to various nerve structures, with doses of 25 mg/kg, 30 mg/kg and 40 mg/kg causing symptoms similar to those observed in Parkinson's disease, a condition known as manganism affecting locomotion, olfaction and neuroaffective behavior, and the 6 mg/kg dose, on the other hand, proved relatively harmless. Neurochemical analyses showed a significant increase in nitric oxide and lipid peroxidation levels, as well as an increase in catalase activity in the hippocampus, striatum, frontal and prefrontal cortex, and olfactory bulb. Chronic exposure to Mn induces dose-dependent neurotoxic effects, necessitating increased monitoring of this metal's levels to prevent health risks.

Keywords: Manganese; Neurotoxicity; Parkinson's disease; Manganism; Dose-dependent effects; Oxidative stress

Abstract ID: 1025

Prenatal quercetin supplementation improves working memory impairment and hippocampal astrocyte activation induced by lipopolysaccharide in an animal model of schizophrenia

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ABSTRACT

Background: Schizophrenia is a neuropsychiatric illness affecting one percent of the world population. Among the multiple factors, maternal exposure to immunogenic agents such as lipopolysaccharide (LPS) is known as a significant environmental risk factor that increases the risk of schizophrenia in the next generation. Quercetin (QE) is a natural flavonoid with multiple forms of desirable biological activity, including anti-inflammatory and antioxidant properties. In the present study, we utilized maternal LPS injection on gestational days (GD) 15 and 16 as a model for schizophrenia to investigate whether QE supplementation can prevent LPS-associated alterations. **Methods:** Pregnant rats (n=24) were randomly assigned into four experimental groups. Control and LPS groups: pregnant dams were received two consecutive intraperitoneal (i.p.) injections of either saline or LPS (0.5 mg/kg) dissolved in saline at GD15/16, respectively. LPS+QE and QE groups: pregnant dams were treated with LPS (0.5 mg/kg, i.p.) or saline on GD15/16 and meanwhile were daily supplemented with QE (50 mg/kg, suspended in water) throughout the gestational period. At postnatal day 60, Y-maze was used to evaluate the working memory performance of male offspring (n=12). After behavioral assessment, qPCR analysis was carried out to measure the expression levels of several pro-inflammatory mediators in the hippocampus of male pups (n=6). Furthermore, immunostaining was performed for the evaluation of astrocyte density on brain sections. **Results:** Male offspring prenatally exposed to LPS showed a significant working memory impairment compared to the corresponding control (LPS group vs control group Bonferroni post hoc analysis, $P < 0.001$), that returned to the control level upon maternal QE supplementation in LPS+QE group (LPS+QE vs LPS Bonferroni post hoc analysis, $P>0.05$). Furthermore, a significant effect of prenatal treatment was also detected for mRNA levels of IL-6 $F (1, 20) = 4.417$, $P = 0.0485$), TNF-α ($F (1, 20) = 6.878$ $P = 0.0163$), NF-κB ($F (1, 20) = 7.323$, $F (1, 20) = 7.323$), and GFAP ($F (1, 12) = 23.86$ $F (1, 20) = 17.83$, $P = 0.0004$), that was prevented by QE supplementation in LPS+QE group. These findings were accompanied by increased astrocyte ($F (1, 12) = 9.453$, $P = 0.0096$) density with unchanged neurons number ($F (1, 12) = 0.4689$, $P = 0.5065$) in the hippocampus of adult offspring. Interestingly, quercetin supplementation could reverse the mentioned deficits induced by LPS (LPS+QE vs LPS Bonferroni post hoc analysis, $P>0.01$). **Discussion:** These results support the idea that the deleterious effects of prenatal LPS exposure could be attenuated by natural flavonoids such as quercetin. It is of interest to suggest early therapeutic intervention as a helpful approach to prevent neurodevelopmental deficits, following maternal infection.

Key words: Schizophrenia; Natural products; Maternal immune activation model

Abstract ID: 1036

Potential neuroprotective effect of Opuntia Ficus Indica cladode extract in a Wistar rat model of Parkinson's disease induced by chronic manganese toxicity

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ABSTRACT

Manganese (Mn) is present everywhere, in soil, water, food and many other forms. Our bodies use it to contribute to energy metabolism and many other functions, but it becomes toxic to the body if exposed to prolonged exposure, inducing symptoms of a disease called manganism similar to Parkinson's disease (PD). The prickly pear is a fat plant belonging to the cactus family, and more specifically to the Opuntia genus, widely distributed in Morocco. Among its species is Opuntia ficus indica (OFI), the cladodes (Cla) of the prickly pear are little exploited, but are rich in bioactive substances with health benefits and antioxidant potential. The aim of this study is to examine the neurotoxic effects of chronic Mn exposure, in particular its effects on the central nervous system, using a dose of 25 mg/kg (ip) in male Wistar rats for 12 weeks to induce a Parkinson-like syndrome and to treat this toxicity, we chose to test the antioxidant effect of OFI Cla and looking for a neuroprotective effect, by preparing an extract at a dose of (4 mg/kg, ip) combined with an intraperitoneal injection of Mn, weight gain was recorded weekly and rats underwent various types of tests to assess their affective, motor and olfactory behaviours. Our results show adverse effects following chronic Mn exposure, producing anorexigenic and depressogenic effects, coordination and locomotion deficits, and neuroaffective disorders, whereas Mn injection combined with Cla produces antidepressant and anxiolytic effects, and improves symptoms in Parkinson-type rats. In conclusion, treatment with extract from OFI cladodes may be a potential treatment for Mn toxicity.

Keywords: Manganese, Opuntia Ficus indica, Parkinson's disease, Neurotoxicity, Neuroprotection

Abstract ID: 1040

Neuroprotective Agents in Parkinson's Disease: Evaluating Natural Compounds and Novel Drugs

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ABSTRACT

Parkinson's Disease (PD) is a progressive neurodegenerative disorder characterized by the loss of dopaminergic neurons in the substantia nigra (Francardo et al., 2017). Current treatments primarily focus on symptomatic relief rather than disease modification. This paper explores the potential of neuroprotective agents, encompassing both natural compounds and novel drugs, to particularly prevent the worsening of the disease. This paper will discuss various natural compounds, including curcumin, resveratrol, ginkgo biloba, and green tea polyphenols, for their antioxidative, anti-inflammatory, and mitochondrial protective properties. Additionally, novel pharmacological agents such as nilotinib, istradefylline, and safinamide are examined for their innovative mechanisms targeting alpha-synuclein aggregation, dopamine receptor activation, and MAO-B inhibition. Through a comprehensive review of clinical trials, we assess the efficacy, safety, and future potential of these neuroprotective strategies in PD management.

Key words: α-synuclein, clinical trials, combination therapies, natural compounds, neuroinflammation, neuroprotection, Parkinson’s disease

Abstract ID: 1032

The evidence for Th2 shift in patients with acute-phase schizophrenia

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ABSTRACT

Background: While research suggests that a disparity in Th1 and Th2 cell responses plays a role in the development of schizophrenia, the specific epigenetic processes involved are still not well understood. In this study, we utilized both bioinformatics and experimental methods to investigate changes in genes related to Th1 and Th2 cells in individuals with schizophrenia.
Methods: Utilizing bioinformatics analysis, we identified Th1-associated genes (NeST and T-bet) and Th2-associated genes (TH2-LCR and GATA3) as potential factors in the development of schizophrenia. To experimentally validate these findings, we quantified the expression levels of these genes in peripheral blood mononuclear cells (PBMCs) from individuals with acute-phase schizophrenia and healthy controls.
Results: Bioinformatics analysis identified 2 lncRNAs, and 234 transcription factors (TFs) linked to Th1 and Th2 cell lineages, which are implicated in schizophrenia. Further examination of qPCR data revealed a significant increase in the expression levels of GATA3 and TH2-LCR with unchanged Th1-related genes (NeST and T-bet) in the PBMCs of schizophrenia patients compared to controls. Notably, both TH2-LCR and GATA3 showed higher diagnostic value in female subjects.
Discussion: These results underscore the crucial involvement of Th1/Th2 imbalance and immune-related genes in the development of schizophrenia. Additional studies with larger, drug-free cohorts are needed to robustly confirm these findings.

Keywords: psychiatric disorders; immune-related genes; lncRNAs

Abstract ID: 1035

Unraveling the Interplay between Hypoxia, Angiogenesis, and Tumor Cell Invasion

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ABSTRACT

Background: Glioblastoma (GBM) is the most aggressive and lethal primary brain tumor of adults, with very rapid progression and poor prognosis despite current therapeutic strategies. Recent improvements in understanding the tumor microenvironment have focused on the role of ischemia-induced hypoxia in driving GBM malignancy. This article focuses on how hypoxia interplays with angiogenesis and the invasive process of tumor cells within the ischemic microenvironment in the context of GBM progression. **Methods:** Following PRISMA, an extensive search across PubMed, Scopus, and Google Scholar was done, including studies that ranged from 2020 to July 2024. The identification included one study on hypoxia, angiogenesis, and invasion of GBM under conditions of ischemia. A total of 1,857 articles were retrieved; 27 studies met rather stringent inclusion criteria to be included for final analysis. Data extraction and bias assessment have been independently done by multiple reviewers, thereby ascertaining the reliability of the findings. **Results:** The investigation showed a maintained active participation of important molecular pathways triggered by hypoxic conditions, especially the HIF-1α pathway, often together with the VEGF and PI3K-Akt-mTOR pathways. Angiogenesis appeared to be an important process. Most studies showed an upregulation of angiogenic factors under hypoxic conditions, which would point to a strong correlation between hypoxia and increased angiogenesis in glioblastoma. Moreover, hypoxia was recognized for promoting the migration and invasion of tumor cells through pathways such as the L-Arg/P4HA1 axis and HIF-1α-related pathways, which are responsible for the aggressive nature of glioblastoma cell invasion. From the records screened, 1702 were subjected to thorough screening and review, resulting in the inclusion of 27 studies in this review. **Conclusions:** The systematic review discusses the complex mechanisms by which the ischemic microenvironment has a promoting effect on GBM malignancy. Hypoxia is an important factor in not only enhancing angiogenesis through up-regulation of VEGF but also invasive capacity through MMP-mediated pathways. Thereupon, therapeutic strategies that would concomitantly inhibit hypoxia-driven angiogenesis and invasion would benefit the outcome of patients. The molecular underpinnings of the interactions involved have not yet been fully decoded for appropriate choice and optimization of treatment.

Key words: Angiogenesis, Glioblastoma (GBM), Hypoxia, HIF-1α pathway, PI3K-Akt-mTOR pathway

Abstract ID: 1045

Selenium and zinc protect against heavy metal mixture-induced memory impairment, and hippocampal oxidative stress by augmenting antioxidant capacity and activation of Nrf2-Hmox signaling in male albino rats.

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ABSTRACT

Heavy metals and metalloid-induced (Cd, Pb, Hg and As) toxicity in the hippocampus (HIP) and relevant limbic regions of the central nervous system CNS, as well as potential protective effects of Se and Zn which remain not well elucidated have been evaluated in this study. Five groups of Sprague Dawley rats were divided into control, toxic metals mixture (TMM) exposed rats (PbCl2, 20 mg·kg-1; CdCl2, 1.61 mg·kg-1; HgCl2, 0.40 mg·kg-1, and NaAsO3, 10 mg·kg-1)), TMM+Zn, TMM+Se and TMM-+Zn+Se groups. All treatments were by oral gavage for 60 days. Barnes maze test was conducted. Oxidative stress biomarkers [malondialdehyde (MDA), superoxide dismutase (SOD), glutathione content (GSH) and catalase (CAT)], inflammatory cytokines [interlukin-6 (IL-6) and TNF-α Nuclear factor kappa B -NFkB, N), apoptotic and redox transcription markers were assessed measuring the activity of Cas-3, nuclear factor erythriod 2- related factor 2 (Nrf2), Heme Oxygynase-1 (Hmox-1), activity of acetylcholinesterase (AChE). Cd, Pb, Hg and as bioaccumulations, and histopathological changes in the hippocampus were determined. There was increase lipid peroxidation and diminished antioxidant capacity in TMM only exposed rats. These adverse effects induced by TMM were alleviated by Zn and Se co-treatment; moreover, essential trace elements (Zn and Se) decreased activity of acetylcholinesterase (AChE), reduced Cd, Pb, Hg and as bioaccumulation and decreased levels of TNF-α. TMM treated rats had lower levels of Hmox1 (HIP) and TMM-treated of Nrf2 (HIP). TMM-treated rats significantly showed the highest time in locating the escape hole. Histopathological examination revealed neuronal degeneration in CA1 of TMM-exposed rats. Various aspects of heavy metals and metalloids-induced toxicity could be reversed by Zn and Se.

Keywords: Hippocampus (HIP); lipid peroxidation, transcriptional factors; acetylcholine.

Abstract ID: 1046

Acoustic non-periodic stimulation (ANPS), a sound created to oppose epilepsy, activates the cortical dorsal but not ventral pathway

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ABSTRACT

Epilepsy represents a major contemporary challenge. Approximately one-third of epileptic patients are considered refractory to medication. Invasive procedures such as intracranial surgery carry risks and are not always indicated, and when performed, they do not guarantee success in all cases. Given the need for alternative methods to control epilepsy, our research group developed Non-Periodic Acoustic Stimulation (ANPS). This stimulus, the ANPS, is a stereo soundtrack with a baseband of 400 Hz, interrupted by events short and slightly faster called 'pulses.' These auditory pulses are asynchronous, low-frequency, and randomized following a power-law distribution. The present research aimed to compare the activation of cortical dorsal and ventral pathways (the where/what system) in patients with epilepsy (Experimental Group – EG) and healthy controls (Control Group – CG) listening to ANPS or white noise (WN). EEG analyses were conducted before, during, and after listening to ANPS or WN in both groups. The study was based on intracortical electrical density (sLORETA) and indices of brain functional networks (BFNs). The ANPS, but not the WN, showed statistically significant activation in the theta and beta 1 frequency bands in the EG but not in the CG, mainly in the frontal and parietal regions. In the BFN analysis, the EG, but not the CG, showed a statistically significant increase in interhemispheric connectivity while listening to ANPS, but not WN. sLORETA analysis showed that ANPS promoted increased activity in cortical regions involved in the dorsal pathway (‘where system'). We believe that, as ANPS presents random and unpaired sound pulses to the right or left ear, it may interfere with brainstem´s source localization function in the nuclei involved in sound processing. Theoretically, this could change synchronicity in a button-up way, reaching the auditory primary cortex, and consequently, the dorsal pathway. These findings suggest that ANPS exhibited distinct electrophysiological characteristics from WN and that patients with epilepsy respond differently to ANPS compared to individuals without epilepsy. Also, ANPS seems to interfere with the dorsal pathway.

Keywords: Epilepsy; Acoustic non-periodic stimulation; dorsal pathway; EEG; LORETA.

Abstract ID: 1047

A Novel Deep Learning Approach for Accurate Brain Tumor Detection and Localization

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ABSTRACT

Introduction: Brain tumor localization and diagnosis using MRI scans is a difficult task, depends mainly on the experience and efficacy of radiologists. Tumors like gliomas, pituitary tumors, and meningiomas vary significantly in appearance, with visual overlaps occurring, especially in early stages. Deep learning technology has proved exceptional proficiency in accurately segmenting and classifying brain tumors. By using deep learning models such as VGG16, for classification and U-Net, for localization, both are well-known convolutional neural network (CNN), have potentials to tackle these challenges. **Aim:** The aim of this study was to develop and apply a U-Net model for the segmentation of brain tumors in MRI images. This deep learning model automatically detect and segment tumor areas and supports in identifying glioma, meningioma, pituitary tumors, and non-tumors in brain scans. Also, we intend to develop a deep learning model using a pre-trained VGG16 architecture to classify brain MRI images into four categories such as glioma, meningioma, pituitary tumor, and no tumor. **Methods:** We applied a combined framework using VGG16 for classification and U-Net for segmentation. MRI dataset available in kaggle.com containing gliomas, pituitary tumors, and meningiomas used for our evaluation and study. Images were preprocessed with augmentation techniques such as rotation, width and height shifts, and brightness adjustments. A VGG16 model, pre-trained and was fine-tuned by adding fully connected layers with dropout and batch normalization. Model performance was evaluated by using accuracy, classification report, confusion matrix, and test set predictions. We employed a trained U-Net model on these paired images and masks, using data augmentation techniques to improve accurately to predict tumor locations. **Results:** VGG16-based model achieved an overall accuracy of 95% in classifying brain MRI images. The model exhibited strong performance across all classes, with high precision and recall metrics. The model's confusion matrix showed excellent differentiation between the tumor types. The U-Net model demonstrated strong performance in segmenting tumor affected areas on test images. Predicted masks closely matched the actual tumor areas, showing high accuracy in detecting and outlining tumors in glioma, meningioma, and pituitary tumor cases. However, a notable limitation of the model was its reduced ability to accurately segment small tumors, where detection and classification performance diminished. While these results are promising, small tumor identification remains a challenge, and further testing is required to improve the detection of early-stage tumors. **Conclusions:** Early and accurate diagnosis of brain tumors is essential for reducing mortality rates. The variability and complexity of tumors make this task challenging, but deep learning models like VGG16 and U-Net offer promising solutions. By improving the accuracy of tumor classification and localization, these models can support radiologists in diagnosing tumors more consistently. With further development, deep learning could be integrated into MRI machines for real-time analysis enabling earlier interventions.

Keywords: Brain tumor localization, Deep Learning Approach; VGG16 model; MRI

Abstract ID: 1049

Early-life inflammation effects on behavior, HPA axis and brain neuroplasticity in mice with autism-like phenotype

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ABSTRACT

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by difficulties in social communication. Changes in the nervous system could be linked to observed immune abnormalities and involve a stress response system. The role of external inflammation stress is still unclear. In our study we used the BTBR T+Itpr3tf/J (BTBR) mice strain, a well-established model for ASD, to analyze the effect of early-postnatal inflammation on behavior and gene expression in the brain. Male BTBR and C57Bl/6 mice were treated with bacterial (lipopolysaccharide, LPS, 50 mcg/kg) and viral (polyinosinic: polycytidylic acid, Poly I:C, 10 mcg/kg) mimetics, as well as their combination on postnatal days 3 and 5. The controls were injected with an equal volume of saline. The dark-light box and social interaction tests were used to determine the level of anxiety and social behavior on the 23-24 days of life. After that we analyzed the expression of genes associated with astroglia and microglia activation (Gfap, Aif1), inflammatory response (Serpina3, Trem2) and HPA axis (Crh) in the frontal cortex and hypothalamus by real-time PCR method. Interstrain comparisons showed no differences in the social interaction index. We can assume that the BTBR strain has not developed social behavior abnormalities by this age. However, in light-dark test BTBR mice spent less time in the light area, had reduced frequency of rearings, and moved between compartments more often, which may indicate a higher anxiety level. Poly I:C and LPS treatment did not affect the social behavior of either strain, whereas Poly I:C treatment led to an increased average duration in light area and the frequency of rearings in light-dark test only in the C57Bl/6 strain, which may suggest a reduction in anxiety. We did not reveal the effect of early-life inflammation on markers of astroglia and microglia activation and inflammatory response in both strains. However, interstrain comparisons revealed that the expression of Aif1 and Trem2 genes in the BTBR strain was lower, while the expression of Gfap and Serpina3 genes were higher compared with the C57Bl/6 strain in both structures. However, an interesting effect of early inflammation has been found in HPA axis. The expression of Crh was higher in the hypothalamus of C57Bl/6 mice compared to BTBR, and it increased with Poly I:C injection. However, no similar effects were observed in the BTBR strain. This pattern of expression is similar to the Poly I:C effects on anxiety behavior. Thus, BTBR mice at the 24th day of life do not exhibit abnormal social behavior typical of adult mice, but they have higher levels of anxiety than C57Bl/6 mice. The BTBR strain shows less sensitivity to the early inflammation by the behavioral patterns and HPA axis genes expression. We also show multiple interstrain differences in the expression of glial activation markers and neuroinflammatory factors as well as a lack of response to early inflammation in these genes.

Keywords: Autism-like phenotype, HPA axis, Brain neuroplasticity, C57Bl/6 strain

Abstract ID: 1050

Harnessing AI for Early Detection of Neurodegenerative Diseases through Medical Imaging

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ABSTRACT

Neurodegenerative diseases like Alzheimer's and Parkinson's are among the most challenging health issues we face today. Early diagnosis is critical, but it remains difficult due to the subtle changes in brain structure that occur in the initial stages of these conditions. In recent years, artificial intelligence (AI) has shown great promise in improving the detection of these early signs, particularly through the analysis of medical images such as X-rays and MRIs. My research focuses on using advanced AI techniques, specifically deep learning and convolutional neural networks (CNNs), to analyze brain images with greater accuracy and speed than ever before. By training AI models on vast datasets of clinical brain scans, we can identify tiny, early-stage changes that may signal the onset of neurodegenerative diseases. This not only helps in diagnosing patients earlier but also enables more timely interventions, improving the chances of slowing disease progression. Beyond early diagnosis, my work also explores how AI can assist in neurosurgery by providing clearer insights into brain structure and function. In line with the congress theme of Neurotechnology and New Discoveries in Neuroscience, I will discuss how AI is becoming a critical tool in transforming the way we understand and treat brain disorders. This research has the potential to significantly impact patient care by making diagnostics more precise and accessible.

Keywords: Artificial Intelligence, Neurodegenerative Diseases, Medical Imaging, Early Detection, X-ray, MRI, Deep Learning, Brain Health

Abstract ID: 1051

Activation of α7nAChR mitigates neuroinflammation linked to cerebral ischemia-reperfusion injury, potentially through microRNA-21

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ABSTRACT

Ischemic stroke is characterized by an abrupt cessation of blood flow in an artery leading to the brain. Activating the α7-Nicotinic Acetylcholine Receptor (α7nAChR) during post-stroke reperfusion injury has been shown to provide neuroprotective effects in stroke-induced injury in rats, but whether this strategy can be used as pre-treatment remains unknown. The first part of this study employed an exploratory work in which α7nAChR agonist PNU 282987 was used as a pre-treatment to prevent cerebral ischemic reperfusion/injury in a zebrafish model by focusing on inflammatory markers. Hypoxia/reoxygenation model was established by perfusing pure nitrogen gas in a hypoxia chamber for 10 minutes followed by 1hour recovery/reperfusion in the beaker. Hypoxia/reoxygenation significantly increased the expression of proinflammatory markers such as TNF-α and IL-6, while α7nAChR agonist reduced these markers. However, there was no discernible improvement in agonist group locomotor activity and brain damage, implying that the neurological impairment was not fully reversed following PNU 282987 pre-treatment. Further, the anti-inflammatory activity of PNU 282987 pre-treatment was examined in BV2 microglia cells that were kept in a hypoxia chamber (Oxygen-glucose-deprived) to simulate ischemic injury. The gene expression of M1 (pro-inflammatory) and M2 (anti-inflammatory) markers as well as microRNA-21/NFκB/STAT3 signalling pathway which has a pivotal role for inflammation was analyzed. The pre-treatment with PNU 282987 inhibited the elevation of M1 markers in OGD/R group and increased the expression of M2 marker, while blocking microRNA-21 reversed these anti-inflammatory effects. Both in vivo and in vitro anti-inflammatory effect of PNU 282987 was shown to be regulated by NFκB downstream signalling pathway. Overall, this study provides important insights into the anti-inflammatory potential of pre-α7nAChR activation in ischemic/reperfusion conditions, with microRNA-21 identified as a potential switch in this receptor-mediated neuroprotection.

Keywords: Ischemic Stroke; microRNA-21; α7nAChR; microglia; Cerebral Ischemic Reperfusion (I/R) injury

Abstract ID: 1052

Delayed effects of early life inflammation in the hippocampus of adolescent male mice with autism-like phenotype

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ABSTRACT

Neuroinflammation is a common feature of many psychopathologies, including autism spectrum disorder, suggesting that the immune system may play a key role in the pathophysiology of these disorders. BTBR T+Itpr3tf/J (BTBR) mice exhibit an autism-like phenotype in adulthood, characterized by behavioral and immune system abnormalities. However, the timing of when these impairments emerge during development is unclear, as does the potential influence of environmental factors in exacerbating their manifestation. To address these questions, we induced early-life inflammation in BTBR mice and evaluated its long-term effects on neuroinflammation in the hippocampus. Inflammation was triggered by bacterial (LPS, 50 μg/kg) and viral (Polyinosinic:polycytidylic acid, 10 mg/kg) mimetics or their combination to male BTBR and C57BL/6 mice on postnatal days 3 and 5. Light-dark box and social interaction tests were conducted on days 39-40 to evaluate anxiety levels and social behavior. Gene expression of key neuroinflammatory markers—Gfap (astroglial activation), Aif1 (microglial activation), Serpina3n (a serine protease inhibitor involved in primarily astrocytic inflammatory response), and Trem2 (a microglial membrane receptor)—was measured using real-time PCR. At the same time, no delayed effects of inflammatory agents on behavior were observed. Significant interstrain differences were observed in hippocampal gene expression. In BTBR mice, Trem2 expression was reduced in both the dorsal and ventral hippocampus compared to C57BL/6 mice, while Aif1 expression was reduced only in the dorsal hippocampus. The consequences of neonatal inflammation were found only in the dorsal hippocampus of C57BL/6 mice: elevated Trem2 expression was observed in groups treated with LPS alone or in combination with viral mimetics, compared to controls. This suggests that BTBR mice exhibit reduced microglial activity, which may explain the absence of delayed effects of early-life inflammation in this strain. Conversely, BTBR mice displayed increased expression of Gfap and Serpina3n in both the dorsal and ventral hippocampus, indicating heightened astroglial proinflammatory activity compared to C57BL/6 mice. Notably, only in BTBR mice was a delayed effect of LPS observed: Serpina3n expression in the dorsal hippocampus was lower in LPS-treated mice compared to controls. This finding points to an abnormal response to proinflammatory stimuli in BTBR mice, which already exhibit high basal astroglial activity. Our results demonstrate that BTBR mice exhibit numerous alterations in microglial and astrocytic activity, which may contribute to the development of autism-like traits in adulthood. However, BTBR mice were less sensitive to early postnatal inflammation compared to C57BL/6 mice, possibly due to their underlying immune system abnormalities.

Keywords: Autism-like phenotype, BTBR, C57BL/6 mice, dorsal hippocampus

Abstract ID:1041

Effects of chronic acamprosate in an animal model of anxiety and depression induced by salicylate

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ABSTRACT

Background: Emotional disorders like anxiety and depression involve imbalances between the excitatory glutamatergic system and the inhibitory GABAergic system in some brain areas. Acamprosate as an effective drug for controlling alcohol dependence is suggested to act with a dual or multiple mechanism in alcohol dependence treatment via affecting on glutamatergic and GABAergic systems. This study aims to assess the effects of chronic Acamprosate on anxiety and depression in rats.
Methods: Forty-four male Wistar rats randomly were divided into five groups: Control; Saline; Sodium salicylate; Acamprosate; and Sodium salicylate + Acamprosate (treatment), and followed for two weeks. 7-day intraperitoneal (IP) administration of Sodium salicylate (400 mg/kg) was applied to establish a chronic model of anxiety and depression. Then, 7-day IP injection of chronic Acamprosate (400 mg/kg) was used to investigate its possible effects on anxiety- and depression-like behaviors. Anxiety and depression were evaluated on baseline day and 14th day by elevated plus maze (EPM), open field (OF), and tail suspension (TST) tests in all exprimental groups. One-way analysis of variance followed by post-hoc Tukey test was used for comparison of data.
Results: After fourteen days, percentage of open arm time parameter in EPM test was decresed in Sodium salicylate and Sodium salicylate + Acamprosate groups compare to Control; Saline; and Acamprosate groups ($P<0.5$). Percentage of central zone time parameter in OF test was reduced in Sodium salicylate group compare to Control; Saline; Acamprosate; and Sodium salicylate + Acamprosate groups ($P<0.5$). Percentage of immobility time parameter in TST was incresed in Sodium salicylate group compare to Control; Saline; Acamprosate; and Sodium salicylate + Acamprosate groups ($P<0.5$).
Conclusion: Chronic Sodium salicylate can induce emotional disorders-like behaviors such as anxiety and depression in rats. Chronic Acamprosate partially reversed indicators of anxiety- and depression-like behaviors. In conclusion, Acamprosate may improve anxiety and depression by modulation of glutamatergic and GABAergic systems, and thereby exert anxiolytic- and antidepressant-like effects.

Keywords: Sodium salicylate; Acamprosate; Anxiety; Depression; Animal model

Abstract ID:1057

The Power of GENUS (Gamma Entrainment) in Revolutionizing Alzheimer's Treatment

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ABSTRACT

Background: Alzheimer's disease (AD) is characterized by disruptions in gamma oscillations (>30 Hz), which contribute to cognitive decline and neuropathological changes. Recently, non-invasive techniques like Gamma Entrainment Using Sensory Stimulation (GENUS) have emerged to target these gamma oscillations at 40 Hz, offering potential benefits for AD pathology. Animal studies have provided compelling evidence supporting the efficacy of GENUS in mitigating AD-related neuropathology, including reductions in amyloid plaques, neurofibrillary tangles, and neuronal loss.

Findings: The animal studies offer convincing support for the effectiveness of GENUS in addressing AD-related pathology. By inducing gamma oscillations through sensory stimulation at 40 Hz, GENUS has shown promise in ameliorating cognitive decline and neuropathological changes in various AD mouse models. Furthermore, human studies, including Phase 1 feasibility trials and Phase 2A pilot studies, have demonstrated the safety, feasibility, and promising clinical outcomes of GENUS in AD patients. These studies indicate that GENUS can improve memory function, reduce cerebral atrophy, and modulate cholinergic transmission, suggesting its potential as a therapeutic intervention for AD. Additionally, researchers have explored transcranial alternating current stimulation (tACS) at gamma frequency targeting the precuneus to enhance episodic memory and cholinergic transmission in early AD. Results from randomized, double-blind, sham-controlled crossover studies have shown significant improvements in episodic memory and cholinergic transmission following gamma-tACS, particularly in patients with specific genetic factors and disease stages. These findings underscore the potential of tACS as a targeted intervention for early AD.

Conclusion: In summary, the evidence from both animal and human studies underscores the potential of Gamma Entrainment Using Sensory Stimulation (GENUS) as a promising intervention for Alzheimer's disease. Animal studies provide robust evidence of GENUS's efficacy in mitigating AD-related neuropathology, while human studies demonstrate its safety, feasibility, and promising clinical outcomes in AD patients. Further research is necessary to optimize parameters, elucidate underlying mechanisms, and develop GENUS as a disease-modifying therapy for Alzheimer's disease.

Keywords: Alzheimer's disease (AD), Gamma entrainment, Gamma-tACS, GENUS's efficacy

Abstract ID:1058

Exploring the Clinical Impact of Augmented Reality in Neuro-oncology

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ABSTRACT

Introduction: Augmented reality (AR) is a new technology with emerging significance in neurosurgery allowing for guiding the surgeon and maximizing brain tumor resection accurately. It projects CT or MRI images directly onto the surgical field—superimposed on it or projected from above allowing surgeons better visualization of difficult-to-see brain structures. It also improves the ability of the surgery to be at gaze with three-dimensional virtual objects in real-time surgical environment making neuronavigation intuitive and effective. The utilization of AR in surgical procedures provides support for complicated anatomical structure navigation, creating a safer experience and better association of the patient anatomy. There is room for AR technology to take even more significant strides forward with neurosurgery as AR evolves itself. **Methodology:** A literature review was conducted from year 2018 to 2024 with a targeted approach towards keywords such as "augmented reality" "neurooncology'' and "neurosurgery" through platforms such as Pubmed Central, Google scholar and Science direct on applications of Augmented Reality in Neuro-Oncology focusing on clinical studies, case series and systematic reviews. **Results:** The use of augmented reality (AR) in neurosurgery has resulted in improved precision in neuronavigation with real-time CT or MRI images superimposed on the surgical field. It means that with very specific operations, for example, tumor resection or tractography, the surgeon can operate without changing line of vision from the operative site. AR also helps distinguishing blood vessels and neural tracts to prevent injury. Chromadepth rendering and color coding are really useful in those hard cases, such as arteriovenous malformations (AVMs). Furthermore, it encourages the safe and immersive procedural training of neurosurgery. But inherent limitations of system latency, tissue movement and image drift is not fully overcome, especially in deep brain surgeries. Nonetheless, AR presents a useful tool for superficial lesions and promises in worldwide neurosurgical education programs with future prospective studies required to evaluate the clinical use of these technologies. **Conclusion:** The use of Augmented reality (AR) is paving the way in neurosurgery by improving precision and visualization. It facilitates highly precise procedures like tumor resections and mapping of neural pathways, by superimposing live CT or MRI images onto the surgical field using neuronavigation through AR. This technology also helps in reducing the threats to imperative structures like blood vessels, and neural tracts. Though it is advanced, it still faces typical issues such as; Image latency, misalignment particularly in deep brain surgeries. But AR has huge potential for surgical uses as well as training. While the implementation of this technology progresses, more clinical and adoptive studies are necessary to completely analyze its clinical effects and extend its integration into neurosurgery.

Keywords: Augmented reality (AR), AVMs, CT, MRI, Neuro-oncology

Abstract ID:1059

Robotic Stereotactic Assistance (ROSA) for Epilepsy Treatment

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ABSTRACT

Epilepsy is one of the most common and disabling neurological conditions. Excessive, hyper synchronous discharge of neurons in the brain cause seizures. Epileptic seizures vary greatly from person to person. Some last few seconds or go unnoticed, some affect one arm or leg and others affect the whole body. Medication with or without therapy can help to prevent seizures. Unfortunately, it doesn't always help. A major challenge confronted by neurosurgeons in the 21st century is the age group of population that needs care. Many elderly and infants experience seizures and ROSA is here to assist. Robotic Stereotactic Assistance (ROSA) is one of the most recent and significant breakthroughs in Epileptic Surgery offering a wide range of benefits. It reduces blood loss, pain improving precision and accuracy. It provides an alternate treatment option for patients who are not suitable for open surgery. ROSA is a computer-controlled metal arm with an integrated platform that combines image guided neurological planning software with robotic navigation to assist neurosurgeons. It works best during Stereotactic Encephalography (SEEG), an intercranial monitoring procedure in which 10-20 thin electrodes are inserted into the brain to precisely map the locations from which the seizures emanate. Based on CT and MRI scanning, it is programmed into ROSA where each electrode should be inserted after which ROSA's robotic arm pivots to the precise positions for each insertion. The electrodes remain in the patient's brain for a week or two, mapping the seizures. The resulting data will determine the best treatment. This includes Laser Assisted Ablation or Resection in the region of the brain causing the seizures. ROSA, a Minimally Invasive Procedure requires less time than traditional surgical procedures and reduces the risk of infection. it improves perioperative results for patients with intractable epilepsy. Since it does not involve large craniotomies, complications are reduced. It is also a safer option for patients with comorbidities. ROSA boasts a success rate of over 90% and seizure free life.

Keywords: Seizures; Robotic Stereotactic Assistance; Stereotactic Encephalography; Seizure Mapping; Ablation.

Abstract ID:1060

The effects of Aticaprant, a κ-opioid receptor antagonist, in stress-induced deficits in mood and cognition.

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ABSTRACT

Kappa opioid receptors (κ-ORs) are extensively investigated for their emerging role in anxiety and depression, as κ-OR blockade impedes the effects of stress in animal studies (1). Aticaprant, a selective κ-OR antagonist is currently in phase III clinical trials and is used for treatment of depression (2). In the present study, mice were subjected to unpredictable chronic stress, followed by administration of Aticaprant and behavioral assessments were performed, including elevated plus maze, open field, novel object recognition, Y-maze and forced swim test. Our results demonstrate that Aticaprant produced an anxiolytic and antidepressant effect, reversed stress-induced impairments in long-term memory, but was ineffective on short-term memory deficits. Recent studies from our laboratory have demonstrated that κ-OR induces autophagy (3), a homeostatic mechanism that degrades dysfunctional proteins to modulate the morphology of neuronal cells and alter synaptic plasticity (4). We have shown that activation of κ-OR by its selective agonist U50,488H mediates the autophagic machinery via a Gαi/o-ERK1,2-CREB pathway resulting in the decrease of hippocampal synaptic proteins, under acute stress conditions (3). In this respect, herein, we demonstrate that the levels of the hippocampal synaptic proteins spinophilin, PSD95 and SNAP25 in stressed animals were restored in Aticaprant-treated animals. By performing neuronal reconstruction analysis in Golgi-cox-stained neurons we show that Aticaprant alleviated stress-induced neuronal and spine loss in stressed animals, supporting its protective role in synaptic plasticity. Moreover, Aticaprant altered the levels of the autophagic markers in chronic stressed animals compared to naïve ones, with a concomitant alteration of the MAPK and Akt/mTOR signaling pathways. Our data provide evidence for the mechanism via which Aticaprant exerts its therapeutic effects as a putative novel drug to alleviate stress-related disorders.

Keywords: Kappa opioid receptor; antagonist; depression; autophagy; synaptic plasticity

Abstract ID: 1061

Executive Function Disorders in Substance Psychosis and Schizophrenic Patients

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ABSTRACT

Aim: In this study, we aimed to test the hypothesis that different areas of the executive functions deteriorate in patients with schizophrenia (SCH), and substance-induced psychosis (SIP) psychosis due to substance abuse particularly. In schizophrenia we suggest that executive function disorder is primarily caused by the frontal cortex, whereas high cognitive problems in substance-induced psychosis develop secondary to the disorder in the subcortical regions.

Material and Method: In this study, the files of 30 male inpatients diagnosis with SIP and 30 male inpatients diagnosed with SCH according to DSM-5 criteria were retrospectively reviewed. The patients were selected by matching their age and educational status, and the routinely performed neuropsychological tests especially related with executive functions. Stroop tests, WMS-R (Wechsler Memory Scale Revised), Digit Span Test, WMS 3 (Wechsler Memory Scale), Mental Control tests were applied to patients with substance-induced psychosis and schizophrenia patients. The results were compared.

Result: Stroop 2 values were found to be higher in SCH group than SIP group. Stroop Spontaneous Correction and Stroop Error numbers were higher in SIP group than SCH group. There was no significant difference between the two groups in the other tests.

Conclusion: This result indicates that a higher level of deterioration in the SCH group compared to the SIP group in the process of changing the perception target. In the parallel processing of attention-directed stimuli and non-attention stimuli, a higher level of deterioration was observed in the SIP group compared to the SCH group.

Keywords: Substance-Induced psychosis, schizophrenia, Stroop tests, executive function, Digit Span Test

Abstract ID: 1062

The effect of hepatic encephalopathy on cognitive functions of patients after living donor liver transplantation

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ABSTRACT

Aim; Hepatic encephalopathy (H.E.), a major complication of severe acute and chronic liver failure, is a common neuropsychiatric syndrome leading to impairments in cognitive and motor activity.
There is no adequate and comparative study in the literature on the postoperative cognitive function of patients with hepatic encephalopathy before transplant surgery and those without hepatic encephalopathy. For the reasons mentioned above, this descriptive and correlational study was conducted to investigate the effect of hepatic encephalopathy on cognitive functions of patients after living donor liver transplantation.
Material and Method: The study was conducted at Inonu University Turgut Özal Medical Center Liver Transplant Institute between July 2022 and September 2024. With the power analysis, it was determined that the sample size should be at least 70 patients with a 95% confidence interval, 0.5% bias level and 0.90 representative power. Considering possible losses, at least 5 more patients were included in each group. The study was completed with 93 patients (45 with hepatic encephalopathy and 48 without hepatic encephalopathy). Patient Information Form, Montreal Cognitive Assessment (MOCA) and Liver Function Tests Record Form were used for data collection.
Results: The majority of the patients who participated in the study were male, married, graduated from primary school and had income equal to expenditure. While there was a difference between the groups in some preoperative blood values (WBC, albumin, ALP, LDH, INR, ammonia and lactate), no statistical difference was found between these values postoperatively. While the MOCA score of individuals who had postoperative hepatic encephalopathy was 15.33, the MOCA score of those who did not have H.E. was 19.85 and the difference was found to be statistically significant (p=0.000*).
Conclusion: As can be understood from the results of the study, the cognitive scores of liver transplant (L.T.) patients who had hepatic encephalopathy were low. Therefore, it may be useful to routinely examine the cognitive scores of all patients who will undergo liver transplantation, whether they have a history of H.E. or not, and to provide cognitive therapies in necessary patients after surgery.

Keywords: Hepatic Encephalopathy, Montreal Cognitive Assessment (MOCA), Liver Transplantation

Abstract ID: 1063

New attitudes for correction of neurodevelopmental disorders symptoms

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ABSTRACT

Introduction: Neurodevelopmental disorders are mostly characterized by inattention, hyperactivity and impulsivity. Those symptoms are induced by impaired brain network activity. The Neurofeedback seems to be a promising method for correction of aberrant cortical processing. After the Inflow Frequency (IL-F) individually chosen training protocol was demonstrated the improvement speed of response and consistency of response in children with attention deficit and hyperactivity syndrome (ADHD) and autism spectrum disorders (ASD).
Methods: 12 children diagnosed with ADHD and ASD aged 7-14 participated in the study after obtaining informed consent. The electrode placement and training frequency were chosen according to the guidelines for the symptom tracking, neuropsychological diagnostic and QIK-Test results.
Results: QIK-Test IL-F training session revealed improvement in speed of response and consistency of response.
Conclusions: The symptoms accompanied by neurodevelopmental disorders are induced by impaired brain cortex network activity. The results obtained from our study show that proper regulation of cortical connectivity ameliorates ADHD as well as ASD symptoms.

Keywords: ADHD, neurodevelopmental disorders symptoms, Neurofeedback

Abstract ID: 1064

Vortioxetine modulates the response to dexamethasone of serotonergic system genes in the Dorsal Raphe Nucleus of mice with social defeat stress experience

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ABSTRACT

This study assessed the effects of vortioxetine on GC-responsive gene expression in mice exposed to chronic social defeat stress. We analyzed transcriptomic changes in the dorsal raphe nuclei (DRN) of the midbrain, which contain serotonin neuron nuclei and are involved in the development of depression. Male C57BL/6 mice underwent 30 days of social defeat stress, followed by 3 weeks of vortioxetine or saline treatment. Dexamethasone or saline was administered 6 hours before tissue collection. RNA from DRN was used for library preparation and sequenced (5 samples per group) using DNBSEQ platform.

In this work, we focused on analyzing the expression of serotonin-related and intracellular signaling-related genes. The expression of Tph2 (encoding the rate-limiting enzyme of serotonin synthesis) and Slc6a4 (the serotonin transporter) was not affected by stress or vortioxetine treatment. Htr1a (somatodendritic autoreceptors) and Htr1b (presynaptic autoreceptors) also showed no significant changes, except for a minor reduction in non-stressed mice treated with dexamethasone. This reduction was attenuated in stressed mice or those treated with vortioxetine, indicating that vortioxetine buffered dexamethasone's effects while stress decreased dexamethasone sensitivity. A significant interaction between stress and vortioxetine was observed in the expression of Htr1b and Htr2c (serotonin receptor regulating excitatory neurotransmission), Maoa (the enzyme that degrades serotonin), and Slc18a2 (the transporter involved in serotonin storage), vortioxetine decreased their expression in non-stressed mice, yet significantly increased it in stressed mice, suggesting a restoration of serotonin receptor function. Vortioxetine altered the dexamethasone response in Htr2c, Maoa, Slc18a2, and Gch1 (the enzyme involved in serotonin synthesis), but only in non-stressed conditions. This implies that vortioxetine modulates serotonin receptor dynamics, though its effects could not be fully restored under stress. In the control group, dexamethasone had no effect on Plcb2 and Plcb3 (enzymes that mediate serotonin-related calcium signaling pathways) or on Gng13 and Gng2 (G-protein-coupled receptor subunits that modulate serotonin receptor signaling). Vortioxetine maintained this lack of response, while normalizing Itpr3 expression (a receptor for serotonin-induced calcium release), in response to dexamethasone. Stress altered dexamethasone response in Gng13, Plcb2, and Gng2, but this effect was absent in stressed mice treated with vortioxetine, suggesting that stress-induced dexamethasone effects were normalized by vortioxetine.

Keywords: Stress experience, Dorsal raphe nuclei (DRN), C57BL/6 mice, Vortioxetine

Abstract ID: 1065

An Improving intervention program for the symptoms related to Mourn and burnout in caregivers of Alzheimer's patients

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ABSTRACT

This study seeks to adapt the mourn intervention program to family caregivers of dementia patients and to evaluate its effectiveness in improving bereavement symptoms and other health-related variables in the grieving process. A total of 52 caregivers from the families of people with dementia participated. They were evaluated by using a series of self-report measures. The results indicate that this program improves the mourn symptoms of the caregivers and reduces the grief of the family. The result was effective. Therefore, due to the lack of programs that provide effective solutions for the mental and physical health of caregivers, and due to the human, social and economic costs of ignoring this issue, creating and implementing interventions targeting vague feelings of sadness and grief and helping Improving grief tolerance in caregivers is clear and obligatory.

Keywords: Sadness, Vague, Dementia, Care, Grief, Program, Intervention

Abstract ID: 1054

Unmasking the Unexpected: Postoperative Venous Pseudoaneurysm Following Torcular Meningioma Resection

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ABSTRACT

Introduction: Owing to their complex anatomical placement at the meeting point of the major venous sinuses, torcula meningiomas present significant challenges in neurosurgical management. Postoperative complications, such as venous pseudoaneurysms, are exceedingly rare but may pose considerable diagnostic and therapeutic dilemmas. We present a unique case of a middle-aged woman who developed a venous pseudoaneurysm following a meticulously executed resection of a torcula meningioma. This case underscores the indispensable role of surgical precision and diagnostic insight in preventing unnecessary invasive interventions. **Case Presentation:** A middle-aged female patient presented with progressive hydrocephalus and imaging revealed a posterior fossa mass, consistent with a torcula meningioma. The patient underwent an occipital midline craniotomy with microsurgical tumor resection. During surgery, a defect in the transverse sinus wall was identified and successfully repaired using autologous muscle fascia and fibrin glue. The patient experienced an uneventful recovery with no postoperative complications. At her two-month postoperative follow-up, magnetic resonance imaging (MRI) revealed a mass at the site of the tumor resection. While the imaging initially raised concerns for recurrence, the lack of mass effect and the surgeon's meticulous intraoperative technique cast doubt on the likelihood of the tumor returning. Considering the possibility of a rare venous pseudoaneurysm, the surgeon made a judgment call to avoid unnecessary interventions. Further follow-up validated this cautious approach, preventing invasive procedures that may have been unwarranted. **Discussion:** This case demonstrates the significance of clinical expertise in navigating complex postoperative findings, particularly in the context of rare complications. Venous pseudoaneurysms, though uncommon, must be considered in patients presenting with postoperative masses when mass effect is absent. The surgeon's rigorous intraoperative approach, combined with a deep understanding of venous anatomy and the rare presentation of a pseudoaneurysm, was pivotal in avoiding additional interventions that may have led to morbidity. **Conclusion:** This case report offers critical insights into the early detection and management of venous pseudoaneurysms following cranial surgery. In rare cases like these, where a postoperative mass may mimic tumor recurrence, the surgeon's judgment and thorough intraoperative technique were paramount in preventing unnecessary invasive procedures. This case goes beyond highlighting the potential for venous pseudoaneurysm formation after sinus wall repair and illuminates the broader implications for postoperative care in complex neurosurgical patients. The seamless integration of surgical precision, imaging, and clinical reasoning sets a benchmark for best practices in neurosurgery, emphasizing the value of individualized patient management in rare and challenging scenarios.

Keywords: Case Presentation, Venous pseudoaneurysms, Torcular Meningioma Resection

Abstract ID: 1067

Prospects and limitations of non-contact recording of brain activity with an infrared camera

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ABSTRACT

Relevance: Humanity rightly considers the human brain as the most complex structurally and functionally organized organ and tries not only to study it more deeply, but also to visualize the work of the brain. Some methods allow to visualize active areas of the brain (EEG, QEEG, Direct Corticography, PET-CT, f-MRI, fNIRS), but each of them has its own limitations. The development of new, more informative and more accurate methods for studying the brain function do not stop. The aim is to study the possibility of recording thermal radiation from the brain with infrared cameras and trying to find out the possibility of deciphering the information received. **Methods:** A cell phone CATS62Pro, with an infrared camera FLIR, was chosen. First, photographs were taken, and then infrared videos were taken from four surfaces of the head. Several people had brain radiation recorded and then a multichannel electroencephalogram was performed. During forensic autopsies of 20 corpses, three-dimensional simultaneous intracranial and extracranial measurements were taken, to specify the projections of some intracerebral formations on different surfaces of the head. Material: Registration of infrared radiation was carried out on people of both sexes, different ages (2-86 years), in a state of wakefulness, with relative physical, psycho-emotional, intellectual and mental peace. **Results:** The presented method, along with the fact that it is inferior in its some capabilities to known similar methods, also has a number of advantages. This is the possibility of conducting direct, not mediated registration, low-cost research. The video frame rate of the CATS62Pro made it possible to shoot high-quality videos of the very fast thermal changes of brain. Moderate focal dynamic activity of infrared rays was found in all lobes of the brain in the waking state with relative physical, emotional-intellectual and mental peace. Small movements of the fingers, hands and the head were accompanied (brain activity preceded these mechanical movements) by rapid jumps of active foci on the parietal and frontal lobes projections, with changes in both the shape and size. During video filming, short thermal "flashes" were visible on the camera screen. To exclude the technogenic cause of the outbreaks, we shot videos from heated surfaces of inanimate objects. No flashes appeared during video recording of artificial heated surfaces, but this issue needs further study. **Conclusion:** Although this study is still at the data accumulation stage, the preliminary results convincingly showed the promise of this method. This method will not only be a successful addition to existing methods for imaging the structure and mostly of function of the brain, but also is able to raise new paradigmatic questions and successfully answer them.

Key words: brain; infrared radiation; brain function visualization.

Abstract ID: 1068

Dynamics of somato-sensory cortex temperature and local blood flow velocity differ under cortical neuron activity modulation and systemic blood flow activation in anesthetized mice brains

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ABSTRACT

Brain temperature (T) is the most important but least studied biophysical parameter determined by the balance between metabolic heat production and hemodynamics. The brain is characterized by high metabolic activity, which leads to intense heat production (Siesjö, 1978). According to theoretical calculations, the thermogenicity of the brain is ~0.66 J/min/g, which in the absence of heat release would result in a temperature increase of 0.16 °C/min (Falk, 1990). The primary mechanism for heat dissipation from the brain is blood flow (Falk, 1990). Available evidence, although limited in quantity, suggests that changes in T brain are a consequence of functional neuronal activation that increases local blood flow through neurovascular coupling (Drew, 2022). However, very little is known about the role of T in brain metabolism and function. In the present study, we performed for the first time long-term simultaneous recording of blood flow velocity and T of deep layers of somato-sensory cortex in the brains of anesthetized mice using microdiamond thermometry and laser speckle contrast imaging techniques. Two approaches were used to analyze the coupling between T and blood flow velocity - enhancement of total blood flow by i.p. injection of adrenaline and enhancement of neuronal activity by application of KCl solution to the brain surface. Adrenaline injection resulted in simultaneous rapid increases in both blood flow (by 52±18%) and T (1.17±0.24°C). However, the duration of the effects of adrenaline on blood flow and T were different: the increase in blood flow lasted 9.75±3.13 minutes in all animals, whereas the increase in T was longer and varied individually among animals. KCl application led to a prolonged increase in blood flow velocity in all animals by an average of 47%, but to multidirectional changes in T in different animals - both its increase (by 0.7°C) and decrease (by 0.84°C) were observed. Our findings of heterogeneous T dynamics in nervous tissue under KCl action are consistent with electrophysiological data that spreading cortical depression is a heterogeneous process in both space and time (Kaufmann et al., 2017). Studies of T dynamics in neural tissue during spreading cortical depression, conducted for the first time in the present work, can make a significant contribution to the understanding of the mechanisms of this phenomenon. The data obtained indicate that the T of neural tissue has more complex dynamics compared to blood flow, which is apparently associated with direct or mediated activation of both individual neurons and neuronal ensembles. Studies of local T dynamics in the brain in different functional states can make a significant contribution to understanding the mechanisms of neurovascular coupling.

Keywords: somatosensory cortex temperature, rectal temperature, dynamics, heating up body, microdiamond thermometer.

Abstract ID: 1069

Therapeutic Potential of the Microbiota-Gut-Brain Axis in Neurodegenerative Diseases: A Comprehensive Review

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ABSTRACT

In Symbiotic bacteria within the human digestive tract play a critical role in maintaining host homeostasis, with profound implications for both overall health and central nervous system (CNS) diseases. These microorganisms form a "gut-brain axis" (GBA), a bidirectional communication pathway that links peripheral intestinal activities to the cognitive and emotional centers of the brain. Emerging research reveals that alterations in the gut microbiota can drive specific responses in the CNS through the neuroendocrine pathway. This intricate signaling involves a combination of hormonal, immune, and neurological interactions, integrating the gut and brain's regulatory functions and affecting human health. The brain continuously monitors energy fluctuations to maintain systemic balance and adapt to metabolic and behavioral changes. Disruptions in the gut microbiota have been shown to impact these processes, leading to neuroendocrine changes that may contribute to various CNS disorders. The gut microbiota's influence extends to neurodegenerative diseases such as Alzheimer's, Parkinson's, autism spectrum disorder (ASD), and multiple sclerosis (MS). Studies suggest that these conditions may be linked to imbalances in the gut microbiome, which in turn affect neuroinflammatory pathways, protein aggregation, and neuronal health. This review delves into the regulatory mechanisms of the gut microbiota, with a focus on nanomedicines as a promising therapeutic approach for neurodegenerative diseases. Nanomedicines, with their targeted delivery systems, offer a new frontier for modulating the gut-brain axis to alleviate neurological conditions. By delivering drugs directly to specific brain regions or influencing microbiota composition, these therapies hold the potential to reverse or slow disease progression. This interdisciplinary approach, integrating gastroenterology and neuroscience, underscores the significance of a holistic understanding of gut-brain interactions in developing effective treatments for neurodegenerative diseases.

Keywords: Microbiota, Gut-Brain Axis, Neurodegenerative Diseases, Nanomedicines

Abstract ID: 1070

Long term memory impairment associated with elevated microglia activation in hippocampal subfields in a rat model of chronic hepatic encephalopathy

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ABSTRACT

Hepatic encephalopathy (HE) refers to a wide range of neurocognitive and neuropsychiatric abnormalities resulted from impaired liver function. Under cirrhosis conditions more than 60% of patients can develop HE. Learning and memory impairment is among the complaints of HE patients under cirrhosis. Several pathophysiological mechanisms are thought to underlie memory deterioration in HE; however, the exact mechanisms remain not fully understood. Excessive microglia activation and the subsequent neuroinflammation might be responsible for memory abnormalities observed in HE patients. The aims of the present investigation were to assess long term memory (LTM) and identify the hippocampal microglial changes in a chronic model of HE. The study was carried out in 5-months male Wistar rats with chronic liver failure induced by thioacetamide (TAA. 100mg/kg. b.w) administration. LTM function was assessed by Morris water maze test, together with an immunofluorescence study of the microglia activation marker, ionized calcium-binding adapter molecule 1 (Iba-1) within the hippocampal subfields; Cornu ammonis 1 (CA1) and CA3. Our data showed impaired LTM in our TAA-treated rats with significant elevation of microglia activation in the CA1 and CA3 regions of the hippocampus which might be responsible for such cognitive impairment. Hence, microglia activation and the subsequent neuroinflammation might underlie memory deterioration in HE patient.

Keywords: Hepatic encephalopathy, long term memory, memory impairment, hippocampus, Morris water maze, microglia activation.

Abstract ID: 1071

Stress and Sleep: What's the link? Neurobiology, Neurocircuitry, and Neuroinflammation of stress-sleep interactions

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ABSTRACT

Stress and sleep are ancient evolutionary behavioural alterations that have allowed multicellular organisms to survive the hardships of external environments. Sleep allows for the homeostatic reset of the brain after times of arousal and is involved in memory consolidation and emotional regulation. Stress is a reactive response to external stimuli that threaten an organism's survival and responds to both chronic psychological and acute physical stressors. Observing patients with stress conditions, a major pathological symptom and risk factor is sleep disturbances. And in the other direction, conditions of sleep dysfunction are associated with increased irritability, emotional dysregulation, and elevated levels of stress hormones in the blood. How both of these systems interact has been researched for decades, but novel breakthroughs are still being made in this field of neuroscience. The objective of this review chapter is to navigate through the nexus between stress and sleep, their mechanisms and interactions, then expands outwards on how dysregulation of these behavioural states emerge as pathology in mammals. Initially, we delve into the neurobiology of the stress-wake system, circadian regulation, and the stress response (Both the hypothalamic-pituitary-adrenal axis and the sympathoadrenal-medullary system). All these systems involve multiple neural centres, as both sleep and stress act as global regulators of neural activity and behavioural responses. We then discuss current understanding on neurocircuitry interactions behind stress and sleep interactions. Vital neural circuits connecting stress and sleep are examined with the attention of the ventral tegmental area GABA-somatostatin neurons and the locus Coeruleus-dorsomedial hypothalamus in sleep regulation in response to stress. Neuroinflammatory markers that are present in cases of stress and disease, such as cytokines interleukin-1β (IL-1β) and tumour necrosis factor alpha (TNF-α), have been shown to be involved in the induction of sleep. Alterations in their concentrations cause significant changes in sleep and arousal. Neuroinflammation, mediated by microglia, is currently being investigated as a major modulator of sleep in normal body function. We also discuss how IL-1β and TNF-α are involved in sleep disturbances caused by stress in the case of psychiatric diseases, such as post-traumatic stress disorder, major depressive disorder anxiety disorders, and chronic stress. It concludes by summarising the implications of neuroinflammatory modulation in stress-related psychopathologies, emphasising the opening this provides for interventions that target this inflammation, helping to lighten sleep disorder. A deeper and more thorough understanding of this complex interaction between the neurocircuitry of stress, sleep, and neuroinflammation may lead to novel therapeutics targeting specific mechanism that may lead to sleep disturbances in patients.

Keywords: Hypothalamic-Pituitary-Adrenal axis, Sleep, Ventral tegmental area, Post-traumatic stress disorder, Retinohypothalamic tract, Corticotropin-releasing hormone

Abstract ID: 1072

A Novel Benzodiazepine Derivative: Exploring Anxiolytic and Analgesic Properties.

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ABSTRACT

Benzodiazepines (BZDs) are widely prescribed for the treatment of conditions such as anxiety, depression, insomnia, and seizures. However, their clinical use remains controversial due to concerns about dependence and withdrawal symptoms and cognitive impairments such as memory deficits, which limit their long-term therapeutic applications. The aim of the current study was to investigate a novel semi-synthetic benzodiazepine derivative that demonstrates promising anxiolytic and analgesic properties while potentially minimizing the adverse effects associated with traditional BZDs. We explored the pharmacological profile of this new molecule through various neurobehavioral assessments in mice. Our results show significant reductions in anxiety-like behaviors, along with effective analgesia in pain models. Importantly, no signs of motor dysfunction or cognitive impairment were observed. In summary, the acute administration of our benzodiazepine derivative demonstrated both anxiolytic and analgesic effects. These observations indicate that the compound could be effective in reducing anxiety and pain, supporting its potential for further therapeutic development.

Keywords: BZD, adverse effects, withdrawal, dependence, Analgesic, Anxiolytic.

Abstract ID: 1073

Neuro AI: Leveraging Deep Learning to Map Neurodegenerative Disease Progression and Therapeutic Response

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ABSTRACT

The ability to predict and track the progression of neurodegenerative diseases, such as Alzheimer's and Parkinson's, has long been limited by the complexity of neurobiological data. This research presents an innovative deep learning framework designed to analyze longitudinal neuroimaging and biomarker datasets, identifying subtle patterns that precede clinical symptomatology. Unlike traditional diagnostic methods, this AI-driven model provides an unprecedented level of precision in predicting disease progression and therapeutic response. Furthermore, we integrate AI with neuroplasticity-driven rehabilitation protocols, tailoring interventions in real-time based on individual patient data. Early results demonstrate improved predictive accuracy and therapeutic outcomes, with significant implications for the future of personalized neurology. This study pioneers the use of AI not only for prediction but also for shaping the therapeutic landscape, enabling real-time adjustments in treatment. The findings offer a revolutionary approach to managing neurodegenerative conditions, opening doors to both clinical and technological advancements.

Keywords: Neuro AI, neurodegeneration, deep learning, personalized, treatment, neuroplasticity

Abstract ID: 1074

Stem cell therapy in degenerative disk disease

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ABSTRACT

Chronic pain affecting millions of people worldwide often leading to diminished quality of life and significant health care burdens. Traditional management are pharmacological treatments and physical therapies. Artificial intelligence is increasingly being integrated into the management of chronic pain, offering innovative solutions to improve patient outcomes and streamline care. In this lecture, we aim to explore the potential of artificial intelligence in enhancing chronic pain management. 1. Patient monitoring. Artificial intelligence can monitor physiological markers like heart rate, skin conductance and facial expression. It can also track a patient lifestyle and keep accurate records. Wearable devices equipped with artificial intelligence can continuously monitor patient physiological parameters provide real-time feedback. This allows timely interventions and adjustment to treatment plans based on the patient's current condition. 2. Medication adherence. Artificial intelligence can send reminders to take medications at specific times, and can adjust for potential drug interactions. 3. Personalized treatment plans. Artificial intelligence can analyze vast amounts of patient data, including medical Histories, genetic information and lifestyle factors to develop personalized treatment plans. Machine learning algorithms can identify patterns that help to predict which treatments may be most effective for individual patients. Machine learning algorithms can be used to predict the likelihood of a patient experiencing chronic pain based on demographic and clinical characteristic or other characteristics as defined by the physical providers' data. 4. Improving diagnosis. Artificial intelligence algorithms can assist healthcare providers in diagnosis chronic pain conditions more accurately. By analyzing imaging studies, lab results and clinical notes artificial intelligence can highlight potential underlying causes of pain that maybe overlooked. 5. Predictive Analytics. Artificial intelligence can help predict pain flare-ups by analyzing data from wearable devices, electronic health records and patient reports. 6. Optimizing medication regiments Artificial intelligence can assist prescribing better medications by analyzing patient responses to different drugs, predicting potential side effects or interaction. This issue is particularly important in chronic pain management because polypharmacy is common. 7. The role of A.I in research. Artificial intelligence facilitates data analyses for research projects. It can help identify trends and correlations in chronic pain management. 8. Personalized education. Artificial intelligence creates personalized educational content including chat-bots and virtual assistants. Artificial intelligence can help patients understanding the physiology and biology of pain through visualization. 9. Cognitive Behavioral Therapy. Artificial intelligence can help with cognitive behavioral therapy and psychological support through remote psychological consultations. 10. Tele Medicine. Artificial intelligence powered chat-bots and virtual assistants can provide patience with immediate support and information about managing their pain. This technology enhances accessibility especially for those in remote areas or with mobility issues. Conclusion. Artificial intelligence holds significant promises for improving outcomes of chronic pain management. While artificial intelligence can be a positive force in patient care, it is important to be aware of the potential risks. Artificial intelligence can make mistakes and it is important to have safeguards in place to protect patient privacy.

Keywords: disc herniation treatment; stem cell therapy; regenerated medicine; degenerative disc

Abstract ID: 1075

Addressing critical gaps in nanofiber scaffold research: long-term efficacy, mechanistic insights, and clinical translation for neural regeneration.

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ABSTRACT

Nanofiber scaffolds are gaining attention for their potential to enhance neural regeneration in neurodegenerative diseases and peripheral nerve injuries by mimicking the natural extracellular matrix (ECM). These scaffolds promote cell adhesion and guide axonal growth. However, several critical research gaps must be addressed before they can be effectively utilized in clinical settings. Key areas needing attention include long-term efficacy studies, understanding cellular interactions, standardizing fabrication techniques, and overcoming challenges related to clinical translation (Shi, Ou et al. 2023). To address these issues, we propose a comprehensive research plan centered on specific solutions. Initially, we recommend conducting long-term efficacy and safety studies using longitudinal animal models to evaluate functional recovery and tissue integration over extended periods (6-12 months) (Gold, Su et al. 2013). Advanced imaging techniques like MRI and histological analysis will assess how well the scaffolds integrate with host tissues and support neuronal growth. Additionally, molecular biology approaches such as RNA sequencing and proteomics will provide insights into the interactions between nanofibers and neuronal cells, identifying key growth factors and cytokines that influence neuronal differentiation. Standardization of fabrication techniques is essential for ensuring reproducibility across studies. Developing consensus protocols for electrospinning parameters—like polymer concentration, voltage, and collector distance will help produce nanofibers with consistent properties. Furthermore, exploring the integration of stem cell therapies could improve regenerative outcomes by evaluating how co-cultured neural stem cells interact with nanofiber scaffolds to support differentiation and integration into damaged neural tissues. Thorough assessments of biocompatibility and neurotoxicity are crucial to ensure the safe use of these scaffolds in clinical applications. Rigorous testing protocols should include chronic exposure studies to evaluate inflammatory responses and long-term cellular viability in relevant animal models. Preliminary findings indicate that enhancing functionalization strategies for controlled release of bioactive molecules can significantly improve scaffold performance. Understanding how environmental factors like inflammation or mechanical stress affect scaffold performance is also vital. Early collaboration with regulatory bodies will help establish safety guidelines and address scalability issues in manufacturing nanofiber scaffolds. Expanding research beyond peripheral nerve regeneration to central nervous system injuries and other neurodegenerative conditions is essential. Utilizing advanced characterization techniques like atomic force microscopy (AFM) will further aid in understanding the structural properties of nanofibers. Through these initiatives, the goal is to advance neural regeneration using nanotechnology, ultimately improving therapeutic strategies for affected patients.

Keywords: Nanofiber Scaffolds; Neural Regeneration; Clinical Translation

Abstract ID: 1076

The role of artificial intelligence in management of chronic pain

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ABSTRACT

Disc degeneration is a major cause of low back pain and is characterized by changes in the matrix and inflammation. This problem significantly affects quality of life often leading to acute or chronic pain and functional limitations. Stem cell therapy has gained attention in the field of regenerative medicine due to its potential to restore damaged tissue. Current treatment options for degenerative disc disease focus on symptom management and providing temporary relief rather than reversing the underlying processes. These approaches include pain medications, physical therapy, steroid injections, and surgical procedures such as microdiskectomy and artificial disk replacement. Also in severe cases, laminectomy and fusion may be considered. Although these methods offer some advantages, they come with operative and post-operative risks, potential complications and limited successes rates. Moreover, these options do not address underlying pathophysiological mechanisms such as inflammation, disturbed matrix balance and the loss of functional native cells with the intervertebral disc. Surgical intervention often result functional impairments, reduced mobility and altered spinal biomechanics, which can lead to generative changes in adjacent disc and vertebre and the eventual recurrence of pain. Disc degeneration begins with a reduction in the population of large notorchordal in the nucleus pulposus. This initiates generative cascade by the loss of normal matrix, increased activity of matrix metalloproteinase, a shift from type 2 to type one collagen, and a decrease in proteoglycan content. Consequently, these changes reduce the binding of water molecules to intervertebral components leading to disk dehydration and subsidence. Contributes critical pressure this process disturbs maintaining the mechanical integrity of the spine, which in turn results local instability and mechanical trauma. Recently stem cell therapy has generated significant attention of regenerated medicine. It seems the method that has the potential of restoring degenerative process of disc can be more successful in treating this problem. The primary objective of stem cell is to replace or restore damaged cells and tissues facilitating the differentiation of implemented cells into the native cells of the target tissue. This type of therapy shows promise for the treatment of the generated disc disease in spite of challenges associated with sell therapy, such as optimal cell sourcing, delivering techniques, and long-term outcome the lack of enough experiences in humans. In conclusion, this lecture aims to provide a comprehensive overview of the current advancements in stem cell therapy for degenerative disk disease.

Keywords: Chronic pain; artificial intelligence; machine learning; pain management; personalized treatment

Abstract ID: 1077

Revolutionizing Skull Base Tumour Resection: The Role of Pulsed Laser-Induced Liquid Jet in enhancing Neurovascular Preservation

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ABSTRACT

Introduction and Purpose: The surgical management of skull-base and soft tissue tumours constitutes considerable challenges due to the need of preserving vital neurovascular structures; however, advancements in minimally invasive techniques, such as the transsphenoidal approach for ventral skull base lesions (e.g., pituitary tumours), have significantly enhanced outcome. These procedures continue to present dangers, including vascular damage resulting from thermal or mechanical trauma during tissue debulking. Conventional technologies such as Doppler probes and micro-hook blades are still prevalent, while recent findings report the effectiveness of water jet systems in accurate tissue resection and preservation of neurovascular structures. This study is focused on investigating the potential benefits of a pulsed laser-induced jet system (LIJS) for skull base tumour procedures, prioritising intraoperative safety and improved postoperative results relative to traditional dissection instruments. **Methods:** This systematic review assesses randomised clinical trials, multi-institutional studies, and experimental investigations conducted in the last decade. Data was gathered utilising online databases, including PubMed, Cochrane and Google Scholar, with applicable search phrases. Eight relevant papers, selected using the PRISMA-P technique (refer to Figure 1), were reviewed to evaluate the utilisation of LIJS in the surgical management of skull base tumours. **Results:** Numerous institutional studies have demonstrated that utilising pulsed LIJS for the excision of skull base tumours, especially pituitary adenomas, yields accurate tissue resection and tumour de-bulking. Our study revealed the preservation of intratumoral arteries in over 84% of cases among 46 patients. LIJS demonstrated excellent devascularization while securely preserving adjacent tissues, such as the cavernous sinus. No complications developed in any of the 46 procedures. Furthermore, its utilisation in complex tumours like meningiomas, assisted in the preservation of the optic chiasm and the associated neurovascular structures. **Conclusion:** The pulsed LIJS has demonstrated the ability to safeguard essential neurovascular systems during tumour excision, preventing heat injury or mechanical trauma usually associated with conventional micro-incisional instruments. The accurate tissue debulking it provides could substitute traditional techniques in skull base procedures. Concerns have been expressed about the system's capacity to produce internal stress in brain tissues, which may impact the manoeuvrability of the jet axis. Future modifications to catheter design could enhance its efficacy in minimally invasive neurological surgeries.

Keywords: Pulsed laser-induced jet system (LIJS), Invasive neurological surgeries, Skull Base Tumour

Abstract ID: 1079

Brain-derived neurotrophic factor in central nervous system myelination: a novel approach to enhance myelin adaptation and restoration

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ABSTRACT

Introduction: The neurotrophin brain-derived neurotrophic factor (BDNF) regulates CNS myelination, neural development, and plasticity in health and disease (1). Myelin injuries resulting from disruption of electrical impulses can cause serious neurological problems. BDNF upon its signaling with oligodendrocytes, can promote myelin repair. This study digs into the influence of BDNF on the myelinating process during development and injury. **Methods:** This literature review involved a comprehensive search through databases such as PubMed and Google Scholar using timelines from 2011-2024 using key terms like myelination, neutrophils, oligodendrocytes, TrkB, BDNF, and central nervous system. The study included mice that don't produce BDNF (BDNF knockout or KO mice) and BDNF heterozygous (HET) mice that produce 40% less BDNF (2). **Results:** BDNF KO mice showed significant decreases in the expression of a key myelin protein, myelin basic protein (MBP), and fewer genes that make MBP and proteolipid protein (PLP). Studies in these indicated that BDNF is specific to myelination rather than neuronal development but is limited in adulthood. These findings in CNS were confirmed in the case of BDNF heterozygous (HET) mice. (2) BDNF works through two types of receptors: p75 NTR and TrkB, in which TrkB is the main receptor for BDNF's pro-myelinating effects. BDNF-TrkB has mitogen-activated protein kinase (MAPK)/Erk pathways that promote oligodendrocyte differentiation and myelination by helping in the maturation of Oligodendrocyte Progenitor Cells. Delivering BDNF through Direct infusion or cell-based gene therapy helps in stroke and spinal cord injury, because of the increased OPC numbers and expression of myelin proteins MBP and PLP. TDP6 and DHF are two TrkB agonists that mimic BDNF. works efficiently in myelin repair, and thickness by activating TrkB without involving other receptors, thus making it effective in the treatment for promoting remyelination in diseases like MS (3). Studies have shown that the multiplication of OPC from TrKB in oligodendrocytes, can also be influenced by neurons and astrocytes. **Conclusion:** BDNF has been proven to promote CNS myelination during development and to enhance remyelination following myelin injury. This makes it effective in therapeutic strategy for neurologic conditions such as MS, stroke, and traumatic injury where myelin damage is the main underlying cause of clinical dysfunction (2). However, the role of TrkB receptors on neurons and astrocytes for myelination is still undiscovered. The outcomes of these studies will ultimately lead us to identify new therapeutic approaches that effectively and specifically enhance myelin repair.

Keywords: Oligodendrocytes; BDNF; myelination; TrkB

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Incorporating robotics in neurosurgical stereotactic procedures: are we missing the point or getting closer to it?

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ABSTRACT

Introduction: Robotic-assisted surgical procedures have become an integral part in modern healthcare. The first surgical procedure executed with the help of a robotic system was a brain biopsy. However, there exist some concerns about the practicality and usability of robotic systems for stereotactic neurosurgery, despite their potential for more accurate targeting of the end-point. The goal of this work is to evaluate the clinical results of robotic-assisted stereotactic DBS procedures reported in the literature, to assess whether the benefits of its implementation justify making a point of facing the financial and logistic drawbacks associated with it. **Methods:** A systematic literature review was performed using the MEDLINE search engine and applying the PRISMA guidelines for systematic reviews. We used the following keywords: "robotic", "stereotactic neurosurgery", "functional neurosurgery", "deep brain stimulation", and "DBS". Only articles comparing the results from robotic-assisted and frame-based stereotactic DBS were included in the review. Original research articles with available abstracts, written in English were taken into account. 42 results were screened, 12 were selected for full read, 10 were retrieved. After careful review, 9 were selected for final analysis. **Results:** The total number of patients who have been reported in all the papers we reviewed and who have undergone robot-assisted stereotactic placement of leads for deep brain stimulation was 472. The following metrics were selected as the outcomes, on which the comparison of the robotic-assisted stereotactic procedure to the conventional frame-based stereotactic procedure was based: mean radial error of the lead placement, mean duration of surgery and rate of complications. In all the papers we reviewed the mean radial error was significantly lower in the robot-assisted group. In only one of the papers, the mean operative time was longer in the robot-assisted group. Some of the reported complications, not related to the proper positioning and function of the leads, include wound dehiscence, hardware infections, postoperative subdural hemorrhage. There was no data within the reviewed papers that points out a significant difference in the rate of the aforementioned complications for the frame-based and robotic-assisted procedures. **Conclusions:** Based on the systematic review that was perfomed, it can be concluded that robotic-assisted stereotactic lead placement for DBS can be safely utilized in clinical practice, while also providing increased accuracy for targetting the preoperatively planned position of the electrodes within the brain.

Keywords: Robotic-assisted surgical procedures, Deep brain stimulation, DBS

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Liquid Biopsies for Early Detection of Brain Tumors

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ABSTRACT

Introduction: Liquid biopsy is a non-invasive method used for cancer detection, specifically in the use of brain tumours. It works by detecting circulating tumour cells (CTCs) and circulating tumour DNA (ctDNA) in the body. This results in giving insights into tumour progression and location. Liquid biopsy when combined with imaging such as magnetic resonance imaging (MRI) and positron emission tomography (PET) scans lead to enhanced detection of brain tumours, track tumour growth, provide personalised treatment approaches and lead to earlier detection. Recent studies have shown that ctDNA holds promise as a prognostic biomarker, with improved sensitivity and accuracy in diagnostics (Khalili et al., 2023; Wen et al., 2022). **Methods:** This review combined from 10 recent studies on liquid biopsy in cancer detection. Khalili et al. (2023) and Wen et al. (2022) focused on combining liquid biopsy with imaging for brain tumor diagnosis. Connal et al. (2023) explored LB's potential in early cancer detection. Capuozzo et al. (2023) discussed the predictive value of CTCs in solid tumors, and Galarza Fortuna and Dvir (2020) provided an overview of ctDNA's role in monitoring tumor progression. The selection was based on studies that highlighted the utility of liquid biopsy in both research and clinical practice. **Results:** Khalili et al. (2023) found that imaging improves ctDNA-based diagnostics for brain tumors, enhancing accuracy. Connal et al. (2023) noted that LB could revolutionize early cancer detection but called for standardized methods. Capuozzo et al. (2023) showed CTCs' prognostic value in solid tumors, aiding clinical decisions. Wen et al. (2022) emphasized ctDNA's role as a novel biomarker, citing advancements in detection sensitivity. Galarza Fortuna and Dvir (2020) confirmed ctDNA's potential in tracking tumor progression, especially when conventional biopsies are challenging. **Conclusion:** Liquid biopsy when integrated with imaging as mentioned previously, especially for brain tumors, forms a significant advancement in oncology. CTCs and ctDNA offer substantial potential as non-invasive biomarkers, with ctDNA showing remarkable promise in detection. Continued research is needed to refine these techniques and further solidify their clinical applications across various other cancers, positioning liquid biopy as a cornerstone of cancer diagnosis.

Keywords: Liquid biopsy, circulating tumor cells (CTCs), circulating tumor DNA (ctDNA), cancer detection, brain tumors