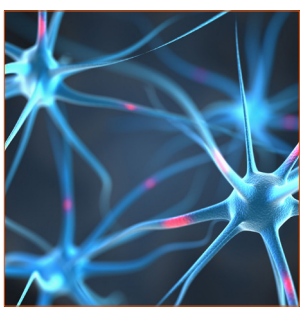
Poster

Brain Disorder 2019




6th International Conference on

Brain Disorders and Therapeutics

July 01-02, 2019 | Paris, France

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Evaluating the anti-hypoxic and anti-ischemic effects of some GABA-receptor mimetics in Brains of mice and rats

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Introduction: Cerebrovascular Accident (CVA) in which Cerebral hypoxia and Ischemia happen is one of the most important causes of disability and mortality in adults, however, it is not treated properly yet. Since the main reason of neural death in this disease is the release of excitatory substances like glutamate, inhibition of neurons with GABA receptor mimetics may reverse the excitotoxicity. In this study, we investigated the anti-hypoxic and anti-ischemic effects of diazepam and phenobarbital (GABA-A allosterics) and baclofen (GABA-B agonist) in comparison to phenytoin (sodium channel blocker and positive control) and normal saline (negative control).

Materials and Methods: The mentioned medicines were injected intra-peritoneally to mice in different doses before the hypoxia. For inducing hypoxia, we put mice individually in a sealed glass container in presence of soda lime and recorded their survival time. In order to create ischemic stress in rats for histopathological evaluation of the hippocampus, we used four-vessel occlusion method. 15 minutes after the ischemic period, 0.6-1cc normal saline, phenytoin 50mg/kg, diazepam 10mg/kg and phenobarbital 40mg/kg were then administered into the rats' peritoneums.

Results: There was a significant increase in the survival time of mice receiving 2mg/kg (PV< 0.01), 5mg/kg, 10mg/kg, 15mg/kg (PV< 0.001) of diazepam, 40mg/kg (PV< 0.01) and 60mg/kg (PV< 0.001) of phenobarbital, and 10mg/kg, 20mg/kg, 30mg/kg and 40mg/kg of baclofen (PV< 0.001) compared to the negative control group (23.03 ± 0.78 minutes), while, the figure for phenytoin 100mg/kg (positive control) was 55.3 ± 3.21 minutes (PV< 0.001). Based on histopathologic examinations, diazepam had no noticeable anti-ischemic effect, however, the preventive effects of phenytoin and phenobarbital was prominent in comparison to the control group.

Conclusion: This study reveals that these compounds may be of great benefit in treating hypoxic-ischemic diseases of CNS.

Speaker Biography

Faezeh Nemati Karimooy has been graduated as an MD from Mashhad University of Medical Sciences, Iran. After graduation she immediately started to work as a GP and the Head of a general health center in Taybad city. Along with her GP career, she was engaged in neuroscience researches. She has also written a book in Persian- translation and complition- named "Sleep and Its Disorders" which is going to be published soon. As an MD, she is also interested in emergencies and collaborated in writing a book in Persian on procedures in emergency medicine.

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Notes:

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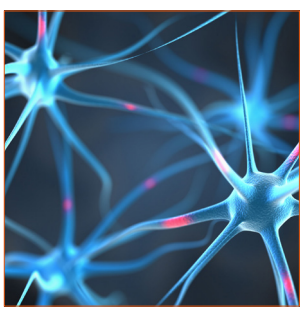


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The Role of APOE in microglia regulation in Neurodegeneration

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Alzheimer's disease (AD) is the most prevalent senile dementia affecting 4.5 million Americans. Neuroinflammatory changes are prominent and may significantly contribute to the pathologic process. Mononuclear phagocytes (brain resident microglia and recruited peripheral monocytes) accumulate around amyloid plaque in AD brains. However, their exact cellular identity, molecular and functional phenotypes, and their protective or destructive roles in AD are not well understood. This stems in part from the lack of a specific molecular signatures for mononuclear phagocytes, cell type-specific antibodies, and analytic tools for in situ characterization. We recently identified a unique TGFβ-dependent molecular signature of homeostatic (M0)- and APOE-dependent neurodegenerative (MGnD)-microglia in neurodegenerative mouse models including APP-PS1 mice and human AD. Mechanistically, the TREM2-APOE pathway mediates a switch from M0- to MGnD-microglia phenotype after phagocytosis of apoptotic neurons in a cell-autonomous manner. TREM2 induces APOE signaling which is a negative regulator of the transcription program in M0-microglia.Transcription regulatory network analysis identified direct effect of APOE on suppression of major microglial homeostatic regulators including TGFβ signaling and induction of disease-associated molecules which are essential for pathogenicity in neuroinflammation. Specific genetic ablation of Apoe and/orTrem2 in microglia restored their homeostatic phenotype and genetic ablation of Apoe or Trem2 in TAU (P301S) mice arrested neurodegeneration and brain atrophy. Therefore, APOE plays an important role in microglia phenotype regulation in neurodegenerative conditions, and restoration of the homeostatic microglia by targeting the APOE-signaling in microglia represents a novel immunotherapeutic approach. Taken together, our work identifies the TREM2-APOE pathway as a major regulator of microglial functional phenotype in neurodegenerative diseases and serves as a novel target to restore homeostatic microglia. These advances have major implications not only for understanding normal CNS function, but have opened up new avenues to understand the role of microglia in disease and most importantly have created the opportunity for consideration of ways in which microglial may be imaged and targeted for the treatment of disease. Since APOE ε4 is the major risk factor of the disease, we study the role of APOE ε4 in microglia regulation by employing novel tools including new mouse models and techniques to specifically target APOE in order to restore microglia-mediated protein clearance and brain function in animal models of tauopathies and AD. I will present recent advances in understanding the new molecular signature of homeostatic microglia, disease associated microglia and how microglia are regulated in health and disease.

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Eosinophilic Granuloma/Langerhans Cell Histiocytosis: Pediatric Neurosurgery

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We report a case of Langerhans cell histocytosis in a 20 months old child which was first misdiagnosed as a case of infective etiology and received several courses of antibiotics with no avail. Eventually he was referred to Neurosurgery department and CT showed a soft tissue mass eroding the temporal bone which was managed surgically and histopathology reported as Langerhands cell histocytosis.

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A dawn of a new era in Stroke care

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Over the past year, landmark clinical trials have transformed stroke care for patients with large vessel occlusion, extending the therapeutic window for mechanical thrombectomy for up to 24 hours from symptom onset. Since, many patients who may have not been eligible for treatment who have favorable clinical and radiographic mismatch profiles with salvageable brain tissue determined by advanced imaging are been treated with excellent outcomes. In addition, our understanding of the pathophysiology of cerebral ischemia has been enhanced by these findings. Clinical guidelines recommend adoption of these best practices and the implications this has had on stroke systems of care are many from EMS to destination therapy. This talk will go over the advances in diagnosis and treatment of large vessel occlusion stroke, current experience and best practices.

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The composition of visual memory and the impact of emotions: The color of memory

Nydia J Gutierrez
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Art therapy creates an implicit memory that helps to reduce stress and anticipates emotional cognitive changes and behavior. It creates a regular function between peripheral nervous system, autonomic nervous system, and the somatic nervous system. This model is represented as a neural algorithm that defines brain functions and models. The model represents a brain relation, behavior, and correlation to human thought and the human mind. In the peripheral nervous system we find dividend between the autonomic nervous system and the somatic nervous system.

The model design and created in this research presents daily functioning cognitive variables that serve as a unity to reduce stress and negative emotional experiences. There is an increase of variation and stimulation that carries effects and voluntary messages to the brain. We induce into a calm cognitive effect and hierarchy that helps to regulate our inner unconscious, subconscious, and thoughts. This also helps defines a healthy synapses and dendrites that intercommunicates with the brain.

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Optoacoustics: An emerging, noninvasive theranostic modality for Brain Disorders

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We proposed to use optoacoustics for biomedical applications and for more than 25 years have been working on it starting from idea to clinical studies. At present, optoacoustics is an emerging theranostic modality. This novel technique is based on detection and analysis of optoacoustic waves generated in tissues by short optical pulses. We proposed many important diagnostic, therapeutic, and theranostic applications of this technology; developed and built optoacoustic systems; and performed animal and clinical studies. Our diagnostic applications include noninvasive transcranial mapping, monitoring, and imaging for management of patients with intracranial hematomas, stroke, neurodegenerative disorders, and other neurological conditions. Here we present an overview of our optoacoustic works from ideas and basic science research to studies in tissues in vitro and to animal and clinical studies. We developed and built medical grade optoacoustic systems for early detection of intracranial hematomas, mapping of cerebral blood oxygenation, and detection of cerebral hypoxia. We tested them in small and large animals (rats and sheep) and then in humans: 1) healthy volunteers; 2) patients with traumatic brain injury (TBI); 3) in neonates (both term and premature); and 4) in fetuses during labor. Recently, we proposed to use optoacoustics for therapy of brain disorders and successfully tested it in rats with TBI. The obtained animal and clinical data indicate that the optoacoustic technique can be used for early diagnostics, therapy, and theranostics of brain disorders. Research support: multiple NIH grants including grants from NINDS, NIBIB, and NICHD.

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Obsessive-Compulsive Disorder as a part of Prodromal Schizophrenia

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Prodromal schizophrenia presents with a wide variety of psychiatric symptoms including obsessive-compulsive disorder (OCD) or obsessive-compulsive symptoms (OCS). However, this differentiation between a sole diagnosis of OCD and prodromal schizophrenia seems challenging in some settings. We present a sixteen-year-old male with six-months history of recurrent intrusive images and fearfulness, in addition to decreased socialization. He was managed as a case of prodromal schizophrenia and was treated with antipsychotics. His obsessions decreased but he continued to exhibit negative schizophrenia within two years of follow-up. Acknowledging the diversity of prodromal schizophrenia presentations rather than treating symptoms as a cross-sectional diagnosis (especially in high-risk population for psychosis) is crucial for a better management.

Discussion: This case illustrates the complexity of the diagnosis of an officially established disorder that is OCD with well-defined criteria and controversial labeling prodromal schizophrenia with several presentations including OCD. The impact of OCD/OCS among prodromal schizophrenia or at-risk people for psychosis was revealed in some studies by having a higher clinical impairment, more depressive symptoms and suicidality.

Conclusion: Our patient was managed as a case of prodromal schizophrenia rather than solely OCD based on the associated features (aloofness, progressive social and academic decline, slowed psychomotor functions and dysprosody). Positive family history of schizophrenia in addition to praecox feeling further confirmed the patient's condition. The following two years of the patient's course revealed the necessity of considering the full detailed presentation of prodromal schizophrenia rather than the spot diagnosis of OCD to benefit from early intervention psychosis services and minimize the clinical deterioration.

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