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Oral Presentations

O1

The Effects of Vitamin D Supplementation on the T cell Compartment in Multiple Sclerosis

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Multiple sclerosis (MS) is a complex neurological disease and its prevalence is about 2 million in the world. Neuroinflammation plays a key role in MS. Vitamins are essential nutrients that have effective role on immune system including activation of lymphocyte and differentiation of T-helper cell. Vitamin D is a micronutrient that is effective on immune function. Deficiently of Vitamin D is a risk factor for progression of MS and studies indicated that 90% of patients with MS have low level of vitamin D. studies showed that there is a relationship between regulatory T cell (Treg) function and Vitamin D status. T lymphocyte and macrophage population have vitamin D receptors especially immature immune cells of the thymus and mature CD8+ lymphocyte. Tregs can be stimulated by Vitamin D supplementation and increased the frequency of Tregs. Also transforming growth factor (TGFβ-1) interleukin 4 (IL-4) can be stimulated by vitamin D that can suppress inflammatory T cell activity. Generally function of vitamin D related to differentiation and activation status of CD4+ T cells. Therefore, attention on other vitamins can be used as an alternative treatment for immune system dysfunction. In this review the effect of vitamin D supplementation and vitamin A on T cell in multiple sclerosis were focused. Previous studies indicate that proportion IL10+ CD4+ were increased and the ratio between IFN- and IL4+ CD4+ T cell decreased by vitamin D. It has been indicated that high dose vitamin D supplementation did not effect on lymphocyte with a regulatory phonotype and the proportion of CD4+ Treg remained unaffected. According to studies, we suggested that other vitamins especially vitamin A can be effective on T cell. T cells that are instigated by Myelin Oligodendrocyte Glycoprotein (MOG) can be reduced by vitamin A.

O2

Anti-Inflammatory Approach to Epilepsy Treatment

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Epilepsy is one of the most common neurologic diseases around the world and more significantly in Iran (0.4-1 % worldwide and 5% in Iran). Almost one-third of these patients suffer from treatment-resistant epilepsy, which reduces their quality of life by recurring epileptic onsets. There are different approaches for the treatment of both treatment-resistant and treatment-nonresistant epilepsy, including drug therapy (Carbamazepine, Diazepam, Eslicarbazepine, Felbamate…), surgery and diet therapy (ketogenic diet), most of which focusing on symptomatic or palliative treatments. Yet the main pathways leading to epilepsy attacks remaining intact. In recent year's evidence have been found suggesting inflammatory mediators might be involved in epileptogenesis. Dr.Vezzani have proved in epilepsy, inflammatory cytokines such as IL-1b, HMGB1 or S100beta are overexpressed in diseased tissues and IL-1b and HMGB1 act as pro-convulsant factors in various seizure models by decreasing the threshold. (Epilepsy and Inflammation in the Brain: Overview and Pathophysiology). In another study she states after a brain seizure, pro-inflammatory cytokines including IL-1β, TNF and IL-6 are over-expressed in micro-glia and astrocytes decreasing excitability threshold afterward. Walker and Sills also add Toll-like receptor signaling pathways as a key mediator resulting in epilepsy. In light of these new findings, a new approach for curing treatment-resistant epilepsy is making its way among other approaches. Inhibitory drug VX-765, an interleukin

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converting enzyme inhibits the formation of IL-1b, the cytokine involved in epileptogenesis. In a randomized, double-blind, placebo-controlled study enrolling 60 adults with treatment-resistant partial onset epilepsy, the end result showed a statistically insignificant difference. However, a 9-13% reduction in seizure rates were observed. To conclude, anti-inflammatory treatment approach to epilepsy and using inflammatory mediator affecting drugs seems to be a promising area, aiming to treat the main causes of epilepsy.

O3

Therapeutic Potential of a Novel NMDA Receptor Subunit 2B Antagonist in a Mouse Model of Autoimmune Neuroinflammation

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Glutamate-mediated excitotoxicity and neurodegeneration have been shown as pathophysiological hallmarks of multiple sclerosis (MS) and other autoimmune inflammatory CNS disorders. NMethylDAspartate (NMDA) receptors play a pivotal role in the mediation of neuronal glutamate excitotoxicity leading to cellular damage and apoptotic cell death. Current treatment approaches targeting glutamate excitotoxicity are unspecific and associated with severe adverse events due to the broad and important functions of NMDA receptors in the CNS. Hence, the present study investigates the neuroprotective potential of a novel specific NMDA receptor 2B (GluN2B) subunit antagonist. Prophylactic and therapeutic treatment with the GluN2B antagonist WMS14-10 (WMS) significantly ameliorated the disease course in myelin oligodendrocyte glycoprotein-induced experimental autoimmune encephalomyelitis (MOG-EAE), a murine model of MS. At disease maximum microglia from WMS treated mice showed decreased CD86 expression indicating reduced microglial activation. In agreement, activated microglia expressed GluN2B. Under restimulation with MOG splenocytes from WMS treated mice demonstrated decreased secretion of TNFα, INFγ and IL-17. In vitro WMS showed no significant effects on the function of T cells and macrophages/monocytes. However, incubation with WMS reduced lipopolysaccharide (LPS)-mediated secretion of cytokines like GM-CSF, IL-1α and TNFα by microglia. In conclusion, our results indicate that specific inhibition of GluN2B in microglia cells displays a newly identified pathway in neuroinflammatory degeneration. Ongoing studies aim at dissecting the underlying mechanisms and a putative additional effect on neuronal glutamate excitotoxicity.

O4

Kynurenine Impairs MbMEC Function in Vitro Through Arylhydrocarbon Receptor Activation

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In the development of neuroinflammatory diseases, alterations of the blood brain barrier (BBB) represent key events. The integrity of the BBB is partially maintained by endothelia cells (ECs), since they actively limit the transmigration of immune cells. However, the factors that cause endothelial cells to develop an immune cell-permissive phenotype are poorly understood. In general, it has been shown that vascular dysfunction can be caused by kynurenine pathway (KP) metabolites. In the initial step of the KP, the bioactive intermediate synthesized is kynurenine (Kyn). It is known to activate the arylhydrocarbon receptor (AhR), a ligand binding transcription factor that mediates immune responses. To examine if this pathway has an effect on the BBB, we investigated the effects of Kyn-mediated AhR activation in primary isolated murine brain microvascular endothelial cells (MbMECs) *in vitro*. First, we confirmed AhR expression in MbMECs at RNA and protein levels. Transendothelial electrical resistance (TEER) of MBMEC monolayers was unaffected by Kyn treatment. However, treatment with Kyn did cause an increased migration of T-cells. Addition of MNF, an AhR specific inhibitor, reversed this effect. These findings were further confirmed by an increase in the intracellular adhesion protein 1 (ICAM-1) expression in KYN-treated MBMECs. These results suggest a role of KYN in MBMEC dysfunction via AHR activation.

O5

Coagulation Factors in Multiple Sclerosis may Represent Diagnostic and Therapeutic Strategies

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Multiple sclerosis (MS) is a neuroinflammatory

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autoimmune disease which mediated by various molecular and cellular immune components However Recent reports have shown that coagulation factors that traditionally separate from the immune system might also be involved in MS development and progression. studies on experimental autoimmune encephalomyelitis (EAE) and human MS patients reports alterations of some factors of the coagulation cascade such as fibrin, thrombin, prothrombin, factor X and FXII to confirm that coagulation factors have an important role in pathogenesis of autoimmune inflammatory disorders, recent studies report that Genetic deficiency or pharmacologic blockade of FXII significantly protected from EAE and also fibrin depletion, either genetically or using anticoagulants, significantly reduces neurolinflammation, and axonal damage in EAE. Another important study shows that increase thrombin activity is an early event and increases with progression of neuroinflammatory disease, with noted microglial activation and axonal damage. In this review we aim to evaluate elevated coagulation factors of tissue or blood as a new therapeutic strategy for the treatment of MS or other neuroinflammatoy disorders. As we described some coagulation factors such as fibrin and thrombin are significantly increased in MS and blockade of this factors in EAE improve neurolinflammation, and axonal damage so maybe using anticoagulants in Clinical trials develop treatment of MS.

O6

Kininogen Deficiency Ameliorates Neuroinflammation by Reducing Immune Cell Trafficking

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Enhanced immune cell trafficking into the central nervous system (CNS) and disruption of the blood brain barrier are pathophysiological hallmarks of neuroinflammatory disorders like multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE). However, recent studies suggest that the coagulation and the contact-kinin system might also be involved in MS development. For instance, it was shown that the coagulation factor XII modulates immune cell function and bradykinin influences the integrity of the blood-brain barrier (BBB). High molecular weight kininogen (HMWK) is a central constituent of the contact-kinin system. Here, we identify HMWK as a critical player in neuroinflammation. Deficiency of HMWK renders mice less susceptible to EAE and was accompanied by decreased numbers of infiltrated lymphocytes into the CNS, whereas the distribution of immune cells was unaltered as determined by flow cytometry analysis. Preliminary *in vitro* migration experiments showed that HMWK leads to an enhanced immune cell trafficking through an endothelial cell layer. Altogether, our study indicates that HMWK inhibition reduces cell invasion during autoimmune CNS disease and may offer a novel strategy to combat MS.

O7

KCNK2 Regulates the Nanoscale Formation of Immune Docking Structures on Brain Endothelial Cells Under Autoinflammatory Conditions

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KCNK2 was previously shown to regulate immune-cell trafficking into the central nervous system (CNS). *Kcnk2*$^{-/-}$ mice demonstrated a more severe disease course in experimental autoimmune encephalomyelitis, an animal model of multiple sclerosis, due to an increased immune-cell migration into the CNS. An upregulation of the cellular adhesion molecules ICAM1 and VCAM1 on brain endothelial cells in *Kcnk2*$^{-/-}$ was proposed as underlying mechanism. The exact molecular pathways involved are currently unknown. By using super resolution microscopy methods, we were able to identify an altered surface morphology of brain endothelial cells upon inflammation indicated by 200-300 nm high membrane protrusions. Analysis of atomic force microscopy (AFM) images of *Kcnk2*$^{-/-}$ mouse brain endothelial cells showed a significant increase in number and volume of membrane protrusions. Confocal imaging identified these membrane protrusions as ICAM1- and VCAM1-containing immune-cell docking structures. *Kcnk2*$^{-/-}$ cells showed alterations of the actin cytoskeleton and an increase of stress fibers already under basal conditions, indicating a regulation of cytokine rearrangement by KCNK2 channels. KCNK2 regulates the nanoscale formation of adhesion molecule-containing immune docking structures on brain endothelial cells under autoinflammatory conditions, thereby regulating leukocyte adhesion and migration into the CNS.

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O8

Tenasin-C as a New Target for Multiple Sclerosis Treatment

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Multiple Sclerosis (MS) is an autoimmune disease, which is characterized by demyelination and neuroinflammation. Extracellular Matrix (ECM) have important role in the central nervous system (CNS). Alterations are happening to the ECM after the CNS disorder like MS, Alzheimer and other neural injury. Tenasin-C (TnC) is a glycoprotein that is highly expressed in inflammatory conditions of the CNS and expression of this protein is up regulated in tissues and organs that are affected by inflammation. Currently cell therapy is one of the main hopes for MS treatment. Unfortunately there is no powerful study to examine the correlation between the Cell Therapy and expression of TnC as a dependent variable for investigates of the improvement percent. Transplantation of mesenchymal stem cells to the experimental autoimmune encephalomyelitis (EAE) model and investigate quantity of the TnC before and after cell transplantation. Evaluation of TnC in the experimental autoimmune encephalomyelitis (EAE) model after mesenchymal stem cells transplantation may be useful for MS treatment.

O9

Immunomodulatory Effects of Neural Stem Cell on Multiple Sclerosis: A Systematic Review

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Multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE), are chronic inflammatory demyelinating disorders of central nervous system (CNS). While the cause is unclear, the fundamental mechanism is thought to be destruction of myelin sheaths of neurons through immune system. One of the approaches being proposed in EAE therapy is neural stem cells (NSCs) transplantation. Several studies have been conducted, investigating immunomodulatory effects of neural stem cells (NSCs) in order to assess their efficacy in the animal model of MS, but still controversies have remained. Our study aim was to systematically review the existing papers in the field of immunomodulatory Effects of Neural Stem Cell on Multiple Sclerosis. The systematic review was conducted according to the preferred reporting items for systematic reviews guidelines. We searched PubMed and Scopus databases based on the relevant medical subject headings (MeSH) of Immunomodulation, neural stem cell, and multiple sclerosis and all articles before January 2017 were included. The included studies had accurate data for immune mechanisms assessment and almost all reported neurologic clinical score assessment. Totally, 30 articles were eligible to be included in our systematic review out of 233 articles found at initial search. Studies showed exert immune modulation when neural stem cells (NSCs) are transplanted in the animal model of MS, experimental autoimmune encephalomyelitis (EAE). Regarding the potent immunomodulatory effects of neural stem cells (NSCs) and their beneficial effects in experimental autoimmune encephalomyelitis (EAE), including their capacity for neuroprotection and Immunomodulation it seems that NSCs may be a new therapeutic method in MS therapy.

O10

Multiple Sclerosis: General Aspects of Pathophysiology, Symptoms and Therapeutic Options

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Multiple Sclerosis (MS) is an autoimmune disease of the central nervous system associated to myelin loss and neurodegeneration. Clinically patients suffer from diverse symptoms and face the risk to become wheelchair-bound. At the moment MS is incurable, thus there is an unmet need for therapeutic options.

O11

Diagnosis and Management of Neuromyelitis Optica

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Typical NMO is characterized by simultaneous or sequential acute transverse myelitis and optic neuritis. Spinal cord lesions extending over 3 or more vertebral segments and normal brain imaging are the typical MRI findings in NMO. In typical cases with positive NMO antibody the diagnosis is easy but in seronegative and atypical cases with different clinical manifestations

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and MRI features the diagnosis will be challenging. Brain stem syndromes such as ataxia, intractable vomiting and cranial nerve palsies are less common initial manifestations of NMO. Shorter spinal cord lesions in MRI can be found very early during relapse or in residual atrophic stages. Some of the lesions may appear hypointense on corresponding T1-weighted images which reflects sever inflammation with necrosis. T1-weighted hypointensities may help in differentiating NMO lesions from MS in spinal cord. Brain MRI is initially normal in most patients with NMO but serial imaging may depict lesions in up to 85% of the patients at later stages. Brain lesions are usually nonspecific but may fulfill the Barkhof criteria for dissemination in space. Acute, large, edematous callosal lesions with a heterogeneous intensity occasionally develop in patients with NMO (marbled pattern). In contrast, in MS, callosal lesions are small, non-edematous, and the intensity was homogeneous in the acute phase and located at the lower margin of the corpus callosum. Multiple patchy enhancements with blurred margins (cloudlike enhancement) have been reported to be typical for NMO. Pencil-like ependymal enhancement is another type of enhancement around the anterior horns of lateral ventricles. Extensive hemispheric lesions, periependymal lesions surrounding the aqueduct, the third and fourth ventricles and brain stem lesions should raise the diagnosis of NMO. Actually NMO should be considered in any atypical lesion for MS in appropriate clinical settings. Other auto anti bodies such as anti MOG have extended the spectrum of this entity and should be evaluated in special cases who are seronegative for anti-aquaporin 4. Treatment of NMO consists of acute relapse management with steroids and/or plasma exchange and prevention of exacerbations with cytoxic agents or Rituximab.

O12

The Pathophysiological Hallmarks of MS Beyond the Blood Brain Barrier: Myelination and Neuronal Network Interactions

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Multiple Sclerosis (MS) is a complex disease resulting from the occurrence of intermingled episodes of neuro-inflammation and degeneration. The temporal and spatial patterns in which these events occur are not well understood as well as the molecular substrates underlying it. Myelin loss and gain, as well as axonal damage are considered crucial events influencing the course of the disease but their cause/effect dependency remains unclear. Numerous recent evidence showed impaired cognitive behaviors both in MS patients and animal models, which would support a profound involvement of neurons in mediating such effects, along to the long-known MS hallmarks like locomotor deficits and slow axonal conductance, attributed mainly to myelin loss. In order to investigate the role of neurons and their functionality in the pathophysiology on MS we took advantage of different animal models of neuro-inflammation and general de- and remyelination, namely the experimental autoimmune encephalitis (EAE) and the cuprizone model, respectively. For both animals we could observe the occurrence of different cognitive impairments including loss of short and long term memory and of high functional cortical abilities. In both animals models we could associate these symptoms to an altered neuronal network excitability and therefore, we pursued pharmacological modulation in vitro and in vivo in order to verify this finding by identifying potential molecular players. Therefore, by using different novel or established compounds we tried to identify new drugable targets and new therapeutic time windows for intervention.

O13

Ion Channels in Autoimmune Neurodegeneration

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Multiple sclerosis (MS) is a chronic inflammatory disease of the central nervous system characterized by widespread inflammation, focal demyelination and a variable degree of axonal and neuronal loss. Ionic conductances regulate T cell activation as well as neuronal function and thus have been found to play a crucial role in MS pathogenesis. Since present therapeutical approaches are only partially effective so far, ion channel modulation as a future strategy was brought into focus. Here, we review the status quo concerning recent findings from ion channel research in MS and its animal model, experimental autoimmune encephalomyelitis.

O14

Differentiating Demyelinating Disorders of the Central Nervous System – a Focus on Multiple Sclerosis and Neuromyelitis-Optica Spectrum Disorders

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Significant advances have been made in diagnosis and therapy of demyelinating disorders of the central nervous system. The most common entities of this disorders in adults – multiple sclerosis and neuromyelitis optica were initially thought to be different phenotypes of more or less the same disease. During the last ten years, this view was subsequently changed and the term neuromyelitis optica-spectrum disorder (NMOSD) was established. He encompasses a variety of clinical presentations that can mimic MS and other disorders. NMOSD are characterized by certain hallmarks in pathophysiology, especially the presence of different antibodies with high specifity. In clinical routine, a clear differentiation between MS and NMOSD is essential due to significant differences in therapy. Treatment of NMOSD with most substances approved for MS can lead to devastating relapses and even death. Furthermore, the clinical course of NMOSD often is severe and itself leads to massive impairment and high mortality. Neurologists – especially those with a scope on neuroimmunology – need to be familiar with diagnosis and at least acute treatment of this disease. This lecture will give an introduction into epidemiology and pathophysiology of the disorder and will highlight its differences to MS. The new criteria for diagnosis of NMOSD will be presented and discussed including the new category of MOG-spectrum disorders. I also will give insights into current strategies for therapy and review future strategies for therapy.

O15

Limbic Encephalitis: General Aspects of Pathophysiology, Symptoms and Therapeutic Options

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Patients with limbic encephalitis usually present with new onset mesial temporal lobe seizures, progressive memory disturbance, and a variety of other behavioral, emotional, and cognitive changes. Autoimmune inflammation of the limbic gray matter structures of the human brain has recently been identified as major cause of mesial temporal lobe epilepsy with interictal temporal epileptiform activity and slowing of the electroencephalogram, progressive memory disturbances, as well as a variety of other behavioral, emotional, and cognitive changes. Magnetic resonance imaging exhibits volume and signal changes of the amygdala and hippocampus, and specific anti-neuronal antibodies binding to either intracellular or plasma membrane neuronal antigens can be detected in serum and cerebrospinal fluid. Therefore, a deeper understanding of the underlying pathophysiological mechanisms is critically required to develop targeted therapies.

O16

Challenge in Diagnosis and Management of Autoimmune Encephalitis

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The first description of an autoimmune encephalitis dates back to 1888, when Hermann Oppenheim described a patient with neurological symptoms but no underlying brain pathology (Oppenheim, 1888). The field of autoimmune encephalitides associated with antibodies targeting cell-surface antigens is rapidly expanding and new antibodies are discovered frequently. Epidemiological studies suggest that anti-NMDA receptor encephalitis may be the most common cause of autoimmune encephalitis after acute demyelinating encephalitis. Autoimmune encephalitis is a difficult clinical diagnosis due to the similarities in the clinical, imaging and laboratory findings of many forms of autoimmune and infectious encephalitis. Patients generally have impaired memory and cognition over a period of days or weeks. There may be clues to specific causes on history of physical examination, but often these specific signs are absent.

Autoimmune Encephalitis Involves Several Types

The first group includes the classic paraneoplastic disorders associated with antibodies to intracellular antigens, such as anti-Hu. of diseases with different pathophysiology. The second group involves autoantibodies to extracellular epitopes of ion channels, receptors and other associated proteins, such as the NMDA receptor. Occupying an intermediate position are diseases with autoantibodies to intracellular synaptic proteins such as GAD65. It is unclear whether this group involves T-cell responses and/or functional effects of antibodies. final group includes other forms of autoimmune encephalitis in which precise antigens are less clearly established, such as lupus cerebritis or ADEM. The classical presentation of encephalitis consists of subacute (days to a few weeks) progressive decrease in the level of consciousness, often with fluctuations, and altered cognition. Memory, especially retention of new information, may be impaired early in the clinical course. Patients may progress to coma. Psychiatric manifestations are common early in the course of autoimmune encephalitis. Although

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this presentation is well known for anti-NMDAR encephalitis, anti-AMPAR and anti-GABA-B-R both may have prominent early psychiatric manifestations (Overall, anti-NMDAR encephalitis is more common and should be suspected first, especially in young adults and children, but they could each cause this presentation across a wide range of ages. Abnormal movements may be the presenting symptom in several types of autoimmune encephalitis. These may resemble dystonia or chorea, with writhing and fixed abnormal postures of the limbs. In adults with anti-NMDAR encephalitis, writhing movements of the face and limbs may be most prominent in the comatose phases of the illness. GAD65 and GlyR autoimmunity may present with stiff person syndrome (SPS) or progressive encephalomyelitis with rigidity and myoclonus A striking feature of PERM with GlyR antibodies is a pathologically exaggerated startle response Seizures are common in autoimmune encephalitis and may be a presenting symptom. Fasciobrachial dystonic seizures (FBDS) are brief seizures consisting of rapid jerks of the face and/or ipsilateral arm and shoulder.10 Seizures may be partial or associated with temporary disruptions in consciousness and may be multifocal and variable on EEG. FBDS are characteristic of LGI1 autoimmunity and may precede other symptoms of the disease by weeks or months. Patients may have hundreds of these seizures per day. These seizures may have only limited response to seizure medications but respond well to immune therapies. Cerebellitis is a distinct syndrome of ataxia of gait, limb movements, eye movements, voice, and/or swallowing. The precise mixture of symptoms varies from patient to patient. Vertigo and nystagmus are common. Cerebellitis may occur with infectious causes, but the presentation of a subacute cerebellar syndrome portends a good probability a specific autoimmune etiology and also a significant risk of tumors. Certain types of autoimmune encephalitis may precede or follow neuromuscular manifestations, particularly acquired neuromyotonia (Isaacs syndrome). Isaacs syndrome presents with muscle spasms, cramps and fasciculations due to peripheral nerve hyper-excitability. Morvan syndrome (Morvan's fibrilllary chorea) consists of peripheral nerve hyper-excitability with encephalitis and severe insomnia.

Diagnostic Approaches

Antibody Testing

Autoantibody testing is extremely important for the proper diagnosis of autoimmune encephalitis. However, the tests have complexities that require consideration, and taking certain test results as conclusive evidence of autoimmune encephalitis can be a mistake.

Imaging

Brain MRI in patients with NMDAR, AMPAR, LGI1, Caspr2, and GABA-B antibodies may be normal or show increased T2 signal, especially in the medial temporal lobes. As mentioned above, imaging abnormalities in routine MRI and FDG PET are not specific and clinical MR imaging can also return completely normal results, e.g. in NMDAR, DPPX or GlyR encephalitis. Advanced imaging techniques, e.g. automated volumetric analyses, quantification of signal alterations, or the application of new sequences or imaging methods, e.g. resting-state functional MRI will help to bridge the gap between clinical and radiological findings.

EEG

EEG is useful in patients with autoimmune or infectious encephalitis for excluding subclinical seizures, for prognosis, and sometimes for suggesting particular diagnoses. In patients with HSV encephalitis, EEG may predict prognosis in addition to helping exclude non-convulsive seizures; normal EEG correlates with good outcomes independent of other prognostic factors.48

Biopsy

Brain biopsy generally is not used in the diagnosis of encephalitis for several reasons.

Cancer Screening

Paraneoplastic disorders are, in general, autoimmune disorders that are triggered by tumors.

Treatment Approaches

Treatment for suspected autoimmune encephalitis is often given empirically prior to specific antibody test results. This may include steroids and/or IVIG. If a cell-surface/synaptic antibody disorder is diagnosed, initial treatments may include IVIG, plasmapheresis, and/or steroids. Steroids may be beneficial in a range of autoimmune disorders but could potentially create problems with the diagnosis of certain disorders such as CNS lymphoma. IVIG offers an important advantage of being unlikely to make infectious encephalitis worse. Plasmapheresis is also unlikely to significantly worsen infectious encephalitis. The proper diagnosis and management of autoimmune encephalitis requires an organized approach. Evaluation should begin with a detailed history and physical examination to detect clues to specific causes. A diverse range of infections should be considered, and appropriate testing should be done to exclude relevant pathogens.

O17

Childhood Anti-NMDA Receptor Encephalitis

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Anti N-methyl-D-aspartate receptor (NMDR) encephalitis has been recognized as the most frequent autoimmune encephalitis in children after acute demyelinating encephalomyelitis (ADEM).However due to the variable the variable clinical presentation, the paucity of specific finding on standard laboratory and radiological investigation remains under recognized. First discribed in 2005, most commonly affected are children and young adults; 80% patients are females. It has characteristic evolution in several stages, in prodromal phase appears fever, malaise, nausea, vomiting, diarrhea, or upper respiratory tract symptoms in about 70%, then gradually manifest psychotic symptoms (delusions, hallucination and mania) that in children can be present as temper tantrum, hyperactivity, irritability, neurologic presentation as seizure, status epilepticus and dystonia. NMDA receptors are ligand cation channels involved in synaptic glutaminergic transmission that plays a key role in functions such as memory, learning, behavior and cognition. Anti-NMDA encephalitis is associated antibodies against the NRI1 subunit of NMDA receptors. MRI changes are non-specific; EEG changes are slowing and background activity; CSF abnormalities are common, mild lymphocytic pleocytosis, elevated protein and positive oligoclonal bands in 60% of patients. Definitive diagnosis is based on demonstration of anti-NMDA antibodies in CSF or serum. Management include symptomatic therapy, definitive immunotherapy, and tumor surveillance. Early diagnosis and aggressive immunetherapy are important. In this presentation, I introduce a patient with anti-NMDA encephalitis.

O18

Autoimmune Dementia

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Dementia is defined as significant acquired cognitive impairment in one or more cognitive domains that represents a significant decline from previous baseline and interferes with independence in daily activities. Autoimmune dementia is a term that has been used to describe a steroid-responsive autoimmune disorder characterized by a rapidly progressive dementia with a fluctuating course. Even compelling evidence suggests that the immune system plays a critical role in the pathophysiology of Alzheimer disease, the most common type of neurodegenerative dementia. According to literature, autoimmune dementia can be due to these disorders: Paraneoplastic and autoimmune limbic encephalitis, encephalopathy associated with systemic autoimmune, and multiple sclerosis. Paraneoplastic limbic encephalitis is characterized by acute or subacute mood and behavioral changes, short-term memory problems, complex-partial seizures, and cognitive dysfunction. The subacute memory loss is a hallmark of the disorder but it can be overlooked easily because of the presence of other symptoms. Hashimoto's encephalitis or encephalopathy (HE) is a rare autoimmune disease often under diagnosed. It can present as rapidly progressive dementia which is treatable with high dose steroids. Early diagnosis and prompt initiation of steroid therapy are associated with good prognosis. Progressive cognitive impairment has been also described in association with systemic lupus erythematosis, Sjogren syndrome, and Behcet disease. These conditions typically produce nonvasculitic encephalitis. In summary, a high clinical suspicious is essential to make an appropriate diagnosis of autoimmune dementia.

O19

Treatment and Porognosis of Autoimmune Encephalitis

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AE is a serious medical condition in which the immune system attacks the brain, impairing function. With rapid diagnosis and appropriate treatment, many patients recover most or all functions. However, not all patients experience full recovery; with approximately 6% mortality and other patients who never regain significant brain and/or bodily functions. Autoimmune encephalitis can produce a wide range of neuro-psychiatric symptoms. While the term "autoimmune encephalitis" appears in the medical literature in the 1970's and 1980's, the first specific AE antibody was identified in 2005 when Dr. Josep Dalmua described the anti-NMDA-receptor encephalitis type. The disease occurs in men, women and children of all ages. AE is a multi-disciplinary disease. Diagnosis and treatment often requires the combined efforts of multiple specialists including: psychiatrists, neurologists, rheumatologists, and immunologists. As soon as a patient is diagnosed with AE, they should receive one or more of the four (4) first-line treatments.

- removal of a teratoma (if present) that could be triggering the autoimmune response
- steroids to reduce immune response and inflammation
- plasmapheresis to remove harmful antibodies from blood
- intravenous immunoglobulin (IVIG), which is believed to occupy the binding sites where harmful antibodies attach to brain cells.

Second line treatments—immunosuppressant drugs—should be started promptly if first-line treatments fail to improve symptoms. Finally some neurologic article reported that 12% of patients had at least one relapse within two years.

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O20

The Role of Neuroinflammation in Epilepsy: A New Target for Treatment

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Despite progress in pharmacological and surgical treatments of epilepsy, little is known about the processes that a healthy brain is rendered epileptic after seizure occurrence. Growing evidence supports the involvement of inflammatory processes, both the adaptive immunity and systemic inflammatory response, in induction of individual seizures as well as in the epileptogenesis. Clinical and experimental investigations indicated that cortical spreading depression (CSD) play a role in epileptogenesis. CSD triggered seizure-like activities in human brain and lead to chronic epilepsy. Poly I:C (Toll-like receptor antagonist) attenuated CSD-induced production of cytokines in the brain and the spleen. In addition, application of poly I:C modulated CSD-induced expression of GABAAα, GABAAβ as well as Hsp70 and GAD65 in the entorhinal cortex. CSD-induced reactive astrocytosis, were paralleled by an increased expression of protein markers indicative of astrocytes and neuroinflammation in ex vivo brain tissues. Cultured astrocytes also showed an enhanced expression of the pro-inflammatory markers in CSD-treated brain. Targeting neuroinflammation with approved and available immunomodulatory treatments may thus represent a strategy to combat or ameliorate CSD-related disorders, such as epilepsy.

O21

A Case of Anti-NMDA Receptor Encephalitis Who Presented with Status Epilepticus

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Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is is most common form ofimmune-mediated encephalitis with prominent neurologic and psychiatric features at disease onset. We describe a 16 years old patient who presented with fever and status epilepticus. His MRI sequences was normal. Anti-NMDAR antibodies were detected in the CSF. MRI, CT and other studies didn,t show any tumor.

O22

Reactive Oxygen Species and Epilepsy

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Seizure activity has been proposed to result in the generation of reactive oxygen species (ROS), which then contribute to seizure-induced neuronal damage and eventually cell death. Although the mechanisms of seizure-induced ROS generation are unclear, mitochondria and cellular calcium overload have been proposed to have a crucial role. We aim to determine the sources of seizure-induced ROS and their contribution to seizure-induced cell death. Live cell imaging techniques in glio-neuronal cultures and in ex vivo epileptic brain tissue. We show that prolonged seizure-like activity increases ROS production in an NMDA receptor-dependent manner. Unexpectedly, however, mitochondria did not contribute to ROS production during seizure-like activity. ROS were generated primarily by NADPH oxidase and later by xanthine oxidase (XO) activity in a calcium-independent manner. Inhibition of NADPH or XO markedly reduced seizure-like activity-induced neuronal apoptosis. In addition, ROS were upregulated in chronic epilepsy in ex vivo brain slices. Inhibition of ROS production in vivo by AEBSF, a NADPH oxidase inhibitor, markedly reduced seizure-induced cell death. These findings demonstrate a critical role for ROS, generated by NADPH oxidase, contributing to seizure-induced cell death. These findings point to NADPH oxidase inhibition as a novel treatment strategy to prevent brain injury in seizures, status epilepticus and chronic epilepsy.

O23

Anti-Inflammatory Treatment in Children with Refractory Seizure

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Epilepsy is a neurological disease of the central nervous system. It is estimated that about 50-70 million people worldwide suffer from this chronic disorder and 20 to 30% are resistant to conventional anti-epileptic drugs. In the epilepsy therapeutic arena, there is real need for developing novel antiepileptogenesis treatments that offer a way to prevent the onset or the progression of the disease.

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Such treatments are still lacking. Numerous experimental and clinical findings demonstrate that brain inflammation plays a key role in the generation of seizures and the pathogenesis of epilepsy. Some conventional corticosteroid therapies are used for seizures in infantile spasm, lafora disease and Rasmussen syndrome. There are some herbal drugs that have anti-inflammatory effects but small side effects, like curcumin. Curcumin is the active component of turmeric which is used in every day cooking. Molecular investigations reveal that curcumin has anti-inflammatory, antimicrobial, anti-hepatotoxic and anti-hyperlipidemic affects. Curcumin has recently been reported to have anticonvulsant effects in several animal models of epilepsy and in our investigation, has effect on refractory myoclonic seizures in children with no significant side effect.

O24

Functional Role of The K2p Potassium Channel Task-3 in A Syngeneic Murine Glioma Model

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To investigate the effects of the two-pore-domain potassium (K2P) channel TASK-3 in a syngeneic murine model for malignant glioma. Malignant or high-grade glioma (WHO grade III and IV) are the most common and most aggressive primary brain tumors in adults. Despite aggressive multimodal therapy, the outcome of patients with malignant glioma remains poor. However, recent phase I and II trials have shown promising results for immunotherapies. The potassium-selective K2P ion channel TASK-3 plays a key role in modulating T cell effector functions. TASK-3 has been shown to functionally impact survival in human glioma cells in vivo and in vitro. We used a syngeneic murine glioma model based on the GL261 glioma cell line. Intracranial injection of GL261 cells leads to development of brain tumors in C57BL/6 wildtype and TASK-3$^{-/-}$ mice. On day 29 after tumor cell implantation eight asymptomatic animals (n = 4 C57BL/6 and n = 4 TASK-3$^{-/-}$ mice) were taken for MRI investigation. Symptom-free survival of GL261 tumor-bearing TASK-3$^{-/-}$ mice was significantly longer as compared to C57BL/6 mice (31 days (29-33 days, 95% confidence interval) versus 23 days (20-26 days, 95% confidence interval); Log-rank test $P < 0.001$). On MRI, tumors of TASK-3$^{-/-}$ mice showed a clear tendency to have smaller volumes as compared to C57BL/6 wildtype mice (5,25 µm³ vs. 15,48 µm³, n.s.). The K2P ion channel TASK-3 plays an important role in local tumor control, as knockout mice exhibit a delayed onset of symptoms and a longer survival after tumor cell implantation. The mechanisms of these effects and the role of other K2P channels are currently under investigation.

O25

Immunotherapy for Brain Tumor

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In 1890, Coley observed that cancer patients who developed infections had smaller tumors. From this, he developed Coley's toxin and treated tumors with injections of infectious materials. In 1960s, Mahaley used monoclonal antibodies to treat central nervous system(CNS) tumors, that research and clinical investigations in brain tumor immunotherapy became a serious undertaking.
There are several different approaches to immunotherapy, include the following:

- Antibody-directed therapies
- Cytokine-mediated therapies
- Cellular therapies
- Vaccines

High-grade gliomas are notable for marked cellular heterogeneity, which accounts for the failure of therapy. Immune therapeutics had previously been underused in patients with low-grade gliomas. These patients are usually less immune suppressed and are less likely to respond to chemotherapy and radiation because their tumor is less rapidly dividing than a malignant glioma. Given the underlying biology, combination therapy will ultimately become the mainstay of therapy. Recent strides have been made in improving survival in these patients with both chemotherapy and immunotherapy . This may be the case when chemotherapy is delivered during the effector phases. However, these two forms of therapy may actually be synergistic in nature when appropriately administered. The proper combination of both chemotherapeutic agent and time of administration will maximize eficacy of treatment and offer an additional regimen for patients, especially those with heterogeneous types of tumors such as glioblastoma multiform, who are not likely to respond to single-agent modality treatments. immunotherapies for gliomas appear promising, but the unique characteristics of the CNS tumors need to be considered for rational design of therapeutics. Novel approaches to antitumor immunotherapy need to be independently and thoroughly evaluated against tumors within the CNS despite successes with these techniques outside the CNS. Identification of tumor-associated antigens provide selective targeting to the gliomas with a theoretical decreased risk for induction of autoimmunity. Many of the previous attempts to

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treat glioma patients with immunotherapies, such as lymphocyte transfer, vaccination with glioma cells, and the use of some cytokines, have not met with significant success. Emerging concepts within the field of immunology, advances in molecular technique, and a greater understanding of the interaction between the CNS and the immune system provide background for more rational and hope- fully more efficacious treatments.

O26

Treatment of Traumatic Brain Injury in Adult Rats with Injection of Human Epileptic Neural Stem Cells and Nano-Scaffold

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Traumatic brain injury (TBI) is described by a disruption in the normal function of the brain due to an injury following a trauma, which can potentially cause severe physical, cognitive, and emotional impairment. The use of human stem cells and self-assembling peptide scaffolds suggest huge potential for application in the treatment of TBI. In the present study, we surveyed the beneficial effects of human adipose-derived stem cells (hADSCs), human epileptic neural stem cells (hENSCs), and PuraMatrix hydrogel (PM) in an acute brain injury model of mild TBI using 3 months rats. hADSCs and hENSCs were transplanted with or without scaffolds into rats brain and the electroencephalography were compared. Modified neurologic severity score (mNSS) tests were performed to measure behavioral outcomes. PM scaffold increased the retention of hENSCs in the lesion site and limited its distribution at the transplanted region. Significantly more hENSCs were detected in the brain when transplanted with PM scaffold. The results showed PM scaffold also efficiently improved cell survival in vivo, resulting in better neural functional recovery. We hypothesized that PM scaffold would improve early engraftment and support the survival of grafted cells and functional recovery post-transplantation.

O27

Traumatic Brain Injury and Inflammation

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Traumatic brain injury (TBI) is a significant public health concern in our country, because of placing in top three most common causes of death and substantial direct and indirect costs to society. The incidence of TBI in our country is 1.7 times of international incidence. Traumatic brain injury induced by primary and secondary mechanisms that give rise to death and neurologic morbidity in patients. Understanding the Pathogenesis and interacting mechanisms of secondary insult may decrease mortality and morbidity of injury. Inflammation is one of the strong theories and understanding the inflammatory mechanism of TBI may conduct us to better prevention and treatment of secondary insults. Neuroinflammation after TBI can have both detrimental and beneficial effects. These effects probably differ in acute and chronic phases. Minimizing the detrimental effect of neuroinflammation may be the key point in the neuroprotective treatment after TBI in the future. We are going to discuss about all of these effects and how we can use these mechanism in clinic and improve the mortality and morbidity of TBI.

O28

Autoimmune Myositis: General Aspects of Pathophysiology, Symptoms and Therapeutic Options

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Idiopathic inflammatory myopathies are a heterogeneous group of muscle disorders characterized by chronic muscle inflammation and progressive muscle weakness. Polymyositis (PM), dermatomyositis (DM) and inclusion body myositis (IBM) are the three major subsets based on distinct clinical and histopathological features. Since the pathogenesis remains unclear, therapeutic approaches actually comprise unspecific immunosuppressive strategies with limited success and frequent side effects. Therefore, a deeper understanding of the underlying pathophysiological mechanisms is critically required to develop targeted therapies.

O29

Autoimmune Myasthenia Gravis Introduction, Immunopathogenesis and Classification

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Autoimmune Myasthenia Gravis (MG) is a unique disease among all autoimmune disorders in two ways. First, there are a wide range of sub-specialties involved in the diagnosis and management of MG; secondly MG is an autoimmune disorder whose autoimmunity is well established. In this paper I will cover these topics: history and epidemiology of MG followed by a brief overview on physiology of neuromuscular junction (NMJ). Immunopathogenesis of MG is probably the most important goal of this article which describe the rule of immune dysregulation and NMJ inflammation in pathogenesis of MG. Finally, I am going to conclude the clinical aspects and serological classification of MG. The incidence rate of MG is estimated to be somewhere between 9 and 30 per 1000000 and the prevalence rate is estimated to be somewhere between 100 and 14 per 1000000. During last two decades both incidence and prevalence rates have been increased significantly. NMJ aperture has a very complicated structure which the action potential passes from motor nerve into the muscle membrane. From immunopathogenesis point of view, MG is actually characterized by reduction of total number of active Acetyl Choline Receptors (AChR) which leads to reduction in end plate potentials (EPPs) necessary for action potential generation of muscle. The reduction of AchR is caused by Abs which target AchRs primarily or other type of receptors which affect AchR consequentially (like MuSK, discovered in 2000 and LRP4 in 2011). Serological classification of MG is probably the most useful approach which helps the clinician to set up management and treatment strategies.

O30

Peripheral Nervous System Diseases and Inflammation

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Inflammatory neuropathies may be due to infection (with a specific casual agent identified) including Lyme disease, HIV, Leprosy, Herpes Zoster, Hepatitis B & C. The other group of are Autoimmune or possibly infectious (but with no specific causal infectious agent identified) including sarcoidosis, Guillain-Barre syndrome/ acute inflammatory demyelinating polyneuropathy (AIDP), chronic inflammatory demyelinating polyneuropathy (CIDP) and neuropathies due to vasculitis such as polyarteritis nodosa (PAN), rheumatoid arteritis, systemic lupus erythematosus (SLE) and Sjogren syndrome. Autoimmune neuropathies also including neuropathy in Celiac disease, multifocal motor neuropathy (MNN), and peripheral neuropathy associated with protein abnormalities such as monoclonal gammopathy, amyloidosis, cryoglobulinemia and POEMS.

O31

HTLV-1 and Inflammation

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Human T-lymphotrophic virus type 1 (HTLV-1) is a retrovirus that is thought to infect approximately 10-20 million people worldwide. The majority of infected individuals remain asymptomatic carriers lifelong. HTLV-1 is thought to cause both local and systemic inflammatory diseases. The most common inflammatory condition associated with HTLV-1 is HTLV-1 associated myelopathy/Tropical spastic paraparesis (HAM/TSP), which occur in 0.25% to 5% of infected individuals. Migration of HTLV-1 infected lymphocytes from periphery into CNS induce a strong immune response and subsequent development of HAM/TSP, which characterized by progressive spastic paraparesis, bladder dysfunction and mild sensory disturbance. HTLV-1 is also associated with other inflammatory conditions such as myositis, uveitis, dermatitis, Alveolitis/ bronchiectasis, arthritis and nephritis.

Poster Presentations

P1

Effect of Clavulanic Acid on Trimethyltin-Induced Cytotoxicity in PC12

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Introduction: Trimethyltin (TMT) is a short-chain trialkyltin used as a stabilizer of plastics. It is also a known neurotoxic agent in both human and animals. Clavulanic acid is previously known as a non-competitive inhibitor of β-lactamase. Oral bioavailability, low antibiotic activity and good CNS penetration are important properties of CA which nominated this compound for evaluation neuroprotective effects. **Materials and Methods:** PC12 cells were exposed to different concentrations of CA for 24 h. Then, TMT at final concentration 20 µM was added. After 24 h exposure, cell viability was determined

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using MTT test. For evaluation reactive oxygen species production, 2, 7-dichlorofluorescein diacetate (DCFH-DA) method was used. Additionally the levels of Bax, Bcl-2 and Caspase 3 proteins were evaluated using western blot analysis. **Results:** Exposure to TMT significantly increased ROS production, Bax/Bcl-2 and Caspase 3 protein levels while decreased cell viability. Pretreatment of cells with CA inhibited ROS production and increased viability. Also, CA could decrease apoptosis through modulation of proteins involved in apoptotic pathway. **Conclusion:** The oxidative stress and apoptosis pathway have important roles in toxicity of TMT in PC12 cells. Clavulanic acid exhibited protective effects through inhibition of oxidative stress and apoptosis pathway.

P2

Proliferation of Neural Stem Cells Promotes in Presence of Feijoa Methanolic Extract in the Oxidative Stress Condition

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Introduction: Now days, several studies have indicated the central nervous system has capacity for endogenous repair. But, the proliferation of endogenous neural stem cells (NSCs) is insufficient for treatment of neurodegenerative diseases. So, it sound that stimulation of endogenous NSC proliferation is essential for neuroregeneration. The *Acca sellowiana (Feijoa)* extract as herbal extract is used as antioxidant agent in antient medicine. Its fruits are rich in vitamin C, polyphenols, terpenes, tannins, steroidal saponins, flavonoids hydrocarbons, minerals, iodine and both methyl and ethyl benzoate. The aim of this study was to examine the self-proliferation and antioxidant properties of Feijoa extract on neonatal rat hippocampus-derived neural stem cells (NSCs). **Materials and Methods:** The NSCs were isolated and cultured. The expression of neural-specific marker, nestin was examined by immunocytochemistry. At first, the cells were in presence of hydrogen peroxide with 50µm concentration in order to oxidative stress induction in vitro and toxicity percentage of hydrogen peroxide was examined. Then, NSCs were exposed to various concentrations (25, 50, 100 and 200 µg/ml) of Acca sellowiana extract for 24 hrs. Thereafter, cell proliferation rate was assessed using MTT colorimetry assay. **Results:** NSCs expressed neural marker (Nestin). Proliferation rate of NSCs was increased in treated groups in comparison with control group. In addition, the results demonstrated that 100µg/ml concentration was the best group for self-proliferation of NSCs. ($P<0.05$). **Conclusion:** These finding shows that the methanolic extract of Acca sellowiana is an antioxidant compound and can promote self-proliferation and survival of NCSs in vitro, suggesting its potential benefits on neuroregeneration.

P3

The Study about MRI Images of Encephalitis and Diagnosis by Using the Software Ways

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Introduction: Encephalitis is inflammation of the brain. Viral infections are the most common cause of the condition .Encephalitis can cause flu-like symptoms, such as a fever or severe headache. It can also cause confused thinking, seizures, or problems with senses or movement. However, many cases of encephalitis result in only mild flu-like symptoms or even no symptoms. It's important to get a timely diagnosis and treatment. A CT-scan may be useful in detecting changes in brain structure. It can also rule out other causes, such as stroke, an aneurysm, or a tumor. However, an MRI is the best imaging option for encephalitis; it can identify the classic brain changes that suggest encephalitis .The purpose of this study was to design and introduce a diagnostic software for encephalitis lesions in MRI images. **Materials and Methods**: This research was a software designing study that many MRI images that used in the past articles were analyzed with the software designer. The designed software was in MATLAB. In this study, we used image processing techniques such as; noise removing, edge denotation, separate of area with high density and contrast increasing for analysis. Based on the evidences from this analysis, radiologist could have the best diagnosis of the lesions. The results of all lesion diagnostics were analyzed and compared in the pathologist's report. **Results**: Designed software enables the present MRI images analyzes them pixel by pixel. This software in addition evaluates the areas of lesions and shown them without viewer diagnosis completely. Final results of diagnostic software analysis showed high

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sensitivity. **Conclusion**: Contemporary assessments of morphologic and physiologic traits of lesions by a computer aided diagnostic software can improve the radiologist's precision and decrease reading time of bulk images of MRI. Using this software to increase the accuracy of the lesions detection is suggested.

P4

The Hypothesis Detect Multiple Sclerosis in Early Stage with Saliva Testing

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Introduction: Recent studies point to the clinical and research efficacy of saliva as a respected diagnostic aid for observing Multiple Sclerosis. The objectives of this Hypothesis are to identify novel biomarkers recognized to Multiple Sclerosis in early stage in saliva and to determine if the levels of these markers correlate with level of these Cerebrospinal fluid and blood assays and urine of diagnostic in multiple sclerosis. **Materials and Methods:** In total, 200 MS patients (100 women) will recruit (in early and late level). Paired samples of saliva, cerebrospinal fluid (CSF), blood serum and urine will be collected to detect osteopontin, Melatonin, Uric acid (UA), malonic dialdehyde (MDA) and oligoclonal IgG an using multiplex proteomic immunoassays. **Results:** we hope to changes of osteopontin, Melatonin, Uric acid (UA), malonic dialdehyde (MDA) and oligoclonal IgG in saliva testing. **Conclusion:** If these parameters change in secretion of salivary gland we can design Microchip to diagnose MS in early stage with saliva testing.

P5

The Effect of Previous Endurance Exercise in Traumatic Brain Injury

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Introduction: It has been suggested physical exercise exerts neuroprotection in traumatic brain injury (TBI). However little information is available about the effect of endurance exercise on brain edema, inflammation and oxidant activity in diffuse TBI. Therefore, we investigated the prophylaxis effect of endurance training against oxidative damage, inflammation and brain edema associated to neurologic outcome in diffuse TBI. **Materials and Methods:** A number of adult male rats of study sustained 8 weeks of treadmill training before TBI induced by Marmarou method. The brain edema (determined by brain water content), inflammation (evaluated by IL-1β level) and oxidative damage (determined by lipid peroxidation) were evaluated in all animals at ٢٤ hours after TBI. Outcome neurologic was determined ١-, and ٤ ,١ and ٢٤ h post-TBI. **Results:** Animals with previous exercise developed less brain edema than animals without exercise following TBI. A reduction in IL-1β level was shown in group with exercise compared to group without exercise. A defect of neurologic outcome was observed following TBI in all times evaluated. Whereas this defect was not observed in exercised animals in any times. The level of lipid peroxidation was no different between and exercise and no exercise groups. **Conclusion:** The results of current study indicate the athletes probably have better neurologic outcome than non- athletes following diffuse TBI maybe part because of less development of brain edema and inflammation.

P6

Effect of Voluntary Exercise on Learning and Memory Impairments in Sleep Deprived Female Rats

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Introduction: Sleep disorders are common problems in modern societies affecting different aspects of individuals' lives. Many studies have reported that sleep deprivation (SD) leads to impairments in hippocampus-dependent learning and memory formations. Physical exercise has been shown to improve learning and memory. The objective of the current study was to investigate the effects of Voluntary exercise on cognitive functions of female rats following sleep deprivation. **Materials and Methods:** Intact female Wistar rats were used in the present study. The exercise protocol

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was 4 weeks of voluntary wheel running. The multiple platform method was applied for the induction of 72 h sleep deprivation and the cognitive function was evaluated using Morris water maze (MWM). ANOVA and repeated measures were used to analyze the data and $P < 0.05$ was considered statistically significant. **Results**: Throughout the investigation, significant learning and memory impairment was observed in sleep-deprived rats compared to the control group. Voluntary exercise alleviated the SD-induced learning and memory impairment. **Conclusion:** The results of our study confirmed the negative effects of SD on cognitive functions in female rats and voluntary exercise seems to protect rats from these effects.

P7

The Effect of Curcumin and Melittin on an Animal Model of MS

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Introduction: Multiple sclerosis (MS) is a progressive and autoimmune neurodegenerative disease of the central nervous system (CNS). This disease is renowned through symptoms like inflammation, demyelination and the damage of neurological actions. Melittin is one of components of bee venom and has anti-neuroinflammatory effects. Curcumin also, a dietary spice from turmeric, has outstanding anti-inflammation and neuroprotective effects. **Materials and Methods:** Experimental allergic encephalomyelitis (EAE) is a widely accepted animal model for MS. EAE is created in animals by injecting the tissue of myelin basic protein (MBP), CNS, or myelin oligodendrocyte glycoprotein (MOG) along with the adjuvant. EAE and MS are similar diseases. EAE was induced in 40 rats randomly placed in four groups of 10: Group 1: Named E-S received normal saline (0.2 mL) every day. Group 2: Named E-mel, received 10 mg/Kg melittin every day. Group 3: Named E-cur, received 100 mg/Kg curcumin every day and Group 4: Named E-cur+mel .The treatments started from the first day of post immunization through GPSH-CFA and lasted until the tenth day. The ELISA and the high performance liquid chromatography (HPLC) were used for the assessment of tumor necrosis factor alpha (TNF-α) and nitrate in rats serum. **Results:** In this study, we indicated that the treatment of EAE with melittin and curcumin decreased the symptoms of clinical disorder, level of serum TNF-α and the serum nitrates in rat EAE. **Conclusion:** This activity of melittin and curcumin may be caused by the anti-inflammatory effects and the immuno-modulatory and antioxidant effects of these.

P8

Affective Factors in the Event Time of Neuropathy in Diabetic Patients (Type II)

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Introduction: Neuropathy is a common complication of diabetes that can cause disability in diabetic patients. The aim of this study was to determine of affective factors in the Event Time of Neuropathy in type 2 diabetes using Cox proportional hazards model. **Materials and Methods:** This study included 371 patients with type II diabetes without neuropathy who were registered at Fereydunshahr diabetes clinic. They were followed up (at least every 4 months) for diagnosis of neuropathy and other complications of diabetes since 2006 until March 2016. To investigate of affective factors in the Event Time of Neuropathy, we used Cox proportional hazards model. All computing was analyzed by R software (ver. 3.2.3). **Results:** At the end of 10 years of study, the cumulative incidence and prevalence of neuropathy were 30.7% and 41.6%, respectively. By Kaplan-Meier method, survival time of neuropathy was 76.6 (±5) months after the first diagnosis of diabetes, in men and women 83.8 (±8) and 72.7 (±6) months, respectively. Among the patients 22% were diagnosed with neuropathy in less than four years after the first diagnosis of diabetes. By The Semi-parametric Cox regression model, Disease-Free survival of one year, two years, five years and eight years were 0.867, 0.819, 0.647 and 0.527, respectively. Also, four variables; length of diabetes period, gender, familial history of diabetes, and HbA1c in Semi-Parametric model (COX), recognized as strong risk factors for event time of neuropathy ($P<0.05$). **Conclusion:** Neuropathy is a common complication in Iranian type 2 diabetic patients. It's related to the duration of diabetes, sex, familial history of diabetes and HbA1c. Optimal glycaemia control and regular evaluation of legs in elderly patients especially in women with positive family history, decrease the

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occurrence and progression of neuropathy and improve the quality of life in diabetic patients.

P9

Neuoprotective Effect of Cannabinoid CB1 Receptor Antagonists Rimonabant and AM251 on Hypoxic Mouse Model of Brain Oxidative Stress

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Introduction: The hypoxic state, in which experimental animals were subjected to an atmosphere of 5% O2 and %95 N2, has been used to screen agents for possible cerebral protection by measuring their ability to prolong survival time in mice exposed to hypoxia. Researchers showed that rimonabant and AM251 allosteric potentiate all but the β1 subunit containing GABAA receptors at nM concentrations. We also showed the potentiating of GABA receptors prolonged the survival time of mice subjected to hypoxia and brain ischemia model. Oxidative stress is caused by excessive production of reactive oxygen species such as hydroxyl radical, superoxide anion radical and hydrogen peroxide. In this study we aimed to test potential protective effects of negative manipulation of cannabinoid receptors. **Materials and Methods:** Male mice weigh 25 to35 gram was separated into 4 groups of 10. The first group give 0.5 cc normal saline IP. Group2: 100 mg/kg phenytoin as standard neuroprotective agent. group3: 10 mg/kg Rimonabant and Group 4:1 mg/kg AM251 injected 30 minutes prior to hypoxia started. Hypoxic anoxia state was performed in closed glass box with N2 pumped and survival time measured by a chronometer. In all sessions the survival time from closing the door of box to stopping the animal breath was measured and recorded. The data were under appropriate statistical tests in SPSS and Prism 6 software. **Results:** In phenytoin group as a standard protective in hypoxic investigations, the survival time increased distinctly in all groups as expected. AM251 showed significant anti hypoxic effect than control group and comparable to phenytoin. Also Rimonabant have same increased in survival time in contrast to control. **Conclusion:** According to the result of this study, antagonizing the cannabinoid receptor, may have protective role in hypoxic trauma situation of nervous system. Although it is required to further investigation. We suggested to test these compound in ischemic situation with histological confirmations study.

P10

Safranal Attenuates Quinolinate-Induced Oxidative OLN-93 Cells Death

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Introduction: Quinolinic acid (QA) is a product of tryptophan degradation and its pathologic accumulation has been found to induce neuroinflammatory and demyelinating diseases such as multiple sclerosis via excessive free radicals generation. Recent studies showed Safranal which is the main component of essential oil of saffron, has several pharmacological effects such as antioxidant, anti-inflammatory and neuroprotective properties. The aim of this study was evaluation of the protective effect of Safranal on oxidative OLN-93 cells death induced by QA. **Materials and Methods:** Cells were pretreated with Safranal (1-800 □M) for 2 h and then were subjected to QA (8 mM) for 24 h in which the same treatments were applied. Cell viability and the parameters of redox status including the levels of intracellular reactive oxygen species (ROS), and lipid peroxidation were measured using 2-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium (MTT), 2,7-dicholorofluorecin diacetate (DCF-DA) and thiobarbituric acid assays, respectively. **Results:** safranal at concentration ranges of 1-800 μM had no toxic effect on cell viability ($p>0.05$). Treatment with Safranal significantly increased cell viability following QA insult at concentrations 1-800 □M ($p<0.001$). Cytoprotective potential of Safranal also ameliorated ROS accumulation and lipid peroxidation induced by QA. **Conclusion:** These data suggest that Safranal exhibits oligoprotection potential by means of alleviating oxidative stress parameters.

P11

Terminalia Chebula Attenuates Quinolinate-Induced Oxidative PC12 and OLN-93 Cells Death

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Introduction: Quinolinic acid (QA) is a product of tryptophan degradation and its pathologic accumulation has been found to induce neuroinflammatory and demyelinating diseases such as multiple sclerosis via excessive free radicals generation. Recent studies showed Terminalia chebula has several pharmacological effects such as antioxidant, anti-inflammatory and neuroprotective properties. The aim of this study was evaluation of the protective effect of T. chebula alcoholic extract (TCAE) on oxidative PC12 and OLN-93 cells death induced by QA**. Materials and Methods:** The cells were pretreated with TCAE (6.25-50 □g/mL) for 2 h and then were subjected to QA (8 mM) for 24 h in which the same treatments were applied. Cell viability and the parameters of redox status including the levels of intracellular reactive oxygen species (ROS), lipid peroxidation and oxidative DNA damage were measured using 2-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium (MTT), 2,7-dicholorofluorecin diacetate (DCF-DA), thiobarbituric acid and comet assays, respectively. **Results:** Based on Folin-Ciocalteu method, the total phenolic compounds in TCAE were estimated about 1.18%. TCAE at concentration ranges of 6.25-50 µg/mL had no toxic effect on cell viability ($p>0.05$). Treatment with TCAE significantly increased cell viability following QA insult at concentrations above 25 □g/mL ($p<0.01$). Cytoprotective potential of TCAE also ameliorated ROS accumulation, lipid peroxidation and DNA damage induced by QA. **Conclusion:** These data suggest that TCAE exhibits neuroprotection and oligoprotection potential by means of alleviating oxidative stress parameters.

P12

Study of the Association between Serum Level of Cystatin C and Behavioral Symptoms of 6-Hydroxydopamine – Induced Parkinsonism in Rat

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Introduction: Parkinson's disease (PD) is the second most neurodegenerative disorder which is characterized by a progressive loss of dopaminergic neurons in the substantia nigra pars compacta. Clinical symptoms do not appear until approximately 70% of dopaminergic neurons and 80% of the striatal dopaminergic terminals have been lost. Thus, detecting nonclinical factors such as detecting biomarker for PD is necessary. In this study, we evaluate the serum level of Cystatin C as a possible biomarker of PD in 6-hydroxydopamine (6-OHDA)-induced Parkinsonism in rat. **Materials and Methods:** Rats were divided into two groups: Parkinson and Control. 6-OHDA was administered by stereotaxic surgery into forebrain bundle. Severity of the Parkinsonism was evaluated by Apomorphine (APO)-induced rotational test at the third and sixth week's post-surgery. Also, serum level of Cystatin C was measured before surgery and at the third and sixth weeks post-surgery. **Results:** Although rats of control group didn't show a significant response to APO, rats of parkinson group showed significant rotations. The rotations at the sixth week's post-surgery were significantly more than the rotations at the third week's post-surgery. However, there was no significant difference between serum level of Cystatin C in rats of control and parkinson group. Also, there was no difference between serum level of Cystatin C in rats of parkinson group before and after the surgery. There was no difference between serum level of Cystatin C and severity of symptoms in rats of parkinson group. **Conclusion:** Our data show that in 6-OHDA animal model of PD, serum level of Cystatin C cannot predict onset or progress of PD and therefore ~~this compound cannot consider as a biomarker for PD.~~

P13

Correlation Between Alanine Aminotransferase Serum (ALT) Level and Behavioral Symptoms of 6-Hydroxydopamine (6-OHDA)-Induced Parkinsonism in Rats

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Introduction: Parkinson disease (PD) is the second frequent human neurodegenerative disease following Alzheimer. This illness is caused by degeneration of dopaminergic neurons in the compact part of substantia nigra (SNpc). Clinical demonstrations are observable when 70% of dopaminergic neurons in SNpc and 80% of dopaminergic terminals in striatum have been degenerated. Characterization of non-clinical bioindicators are therefore of great importance in diagnosis of the disease. This work aimed to assess the serum level of ALT, as a bioindicator of PD, following induction of the disease by 6-OHDA in an animal model. **Materials and Methods:** Twenty-eight Wistar rats were

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classified in two equal control and PD-induced groups. The 6-OHDA toxin was injected into the medial forebrain bundle of rats in PD-induced group through stereotaxic surgery. Animals in control group received no such treatment. Severity and extent of Parkinsonism in both animal groups were assessed by apomorphine-induced rotational test. Blood specimens were consequently collected from all animals and the serum ALT level was determined. **Results:** In the PD-induced rats, ALT level was detected at much lower amounts compared to the control group ($P<0.05$). The severity of PD, however, was not similar between animals as severe, mild and even no-detectable clinical symptoms were observed in examined rats. The ALT level in severely and mildly affected animals was clearly lower compared to control group ($P<0.01$, $P<0.05$). Besides, no definitive difference was detected between animals with no symptoms and control group. **Conclusion:** Since the severity of rotations in the apomorphine-induced rotational test is in harmony with extent of degeneration of dopaminergic neurons in SNpc, the serum level of ALT is likely to be an important bioindicator of Parkinson disease.

P14

CSF NGF/IL-6 Ratio: a Useful Marker for the Evaluation of Progesterone Efficacy in Traumatic Brain Injury

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Introduction: Following our previous studies on the neuroprotective effects of progesterone and cytokines such as IL-6 (IL-6) is involved in the inflammatory response, to examine whether changes in IL-6 and Nerve grows factor(NGF) concentrations in CSF can responsible for the neuroprotective effects of progesterone after traumatic brain injury(TBI). **Materials and Methods:** Female ovariectomized rats were divided into 9 groups: intact (pro estrous and non-proestrous), sham, TBI and 4 groups treated by vehicle or different doses of progesterone, including: vehicle, LP (low dose of progesterone, 1.7 mg/kg), HP (high dose of progesterone, 8 mg/kg), IVC (implant vehicle capsules, 10-20 ng/ml) and IPC (Implant low dose of progesterone capsules, 10-20 ng/ml). In groups receiving hormone or vehicle, treatment was administered as a single dose of intraperitoneally 30 minutes or implant capsules 6 hours following a diffuse TBI that was induced by Marmarou's method. The levels of biomarkers in CSF were measured at 48 h after the TBI. **Results:** Both doses of progesterone reduced CSF levels of IL-6 compared with vehicle group ($p<0.05$, $p<0.001$, respectively), but the difference between CSF levels of NGF in progesterone and vehicle was not significant. After trauma, although the ratio of NGF to IL-6 significantly higher in the progesterone groups than in the vehicle group ($p<0.05$). The CSF level of IL-6 was reduced in IPC group, compared with IVC group ($p<0.05$), but the CSF level of NGF is increased ($p<0.05$). The NGF/IL-6 ratio in IPC group was 4.13 higher following administration of vehicle (1.16, $p<0.001$) levels, and the highest maximum NGF/IL-6 ratio is in the same group. **Conclusion:** Based on our findings, we conclude that individual measure these two indicators to evaluate the effects of drugs may be not useful, but CSF NGF/IL-6 ratio might be a better marker for determine the effectiveness of drugs.

P15

Forced Exercise Attenuates Neuropathic Pain in Chronic Constriction Injury Male Rat: An Approach to Oxidative Stress and Inflammation

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Introduction: Peripheral and central nerve injuries cause chronic neuropathic pain in many injured people besides motor disability. Exercise, as a behavioral and non-pharmacological treatment, has beneficial effects on people's general health both for healthy and sick people. Therefore, this study was conducted to examine the effects of exercise on neuropathic pain induced by chronic constriction injury (CCI) of the sciatic nerve. **Materials and Methods:** Wistar male rats weighing 200 ± 20 g were randomly divided into five groups (normal, sham, CCI, pre-CCI exercise, and post-CCI exercise group). Once the rats were anesthetized, their sciatic nerve was legated to induce CCI, and they were then housed in separate cages. The rats ran on treadmill at a moderate speed for 3 weeks. Mechanical allodynia and thermal hyperalgesia were determined using von Frey Filament and plantar test, respectively. TNF-α, malondialdehyde, and total antioxidant capacity were measured using Western blot test, thiobarbituric acid, and ferric reducing

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ability of plasma (FRAP) respectively. **Results:** The mechanical allodynia ($P<0.05$) and thermal hyperalgesia ($P<0.01$) in the CCI group were significantly higher than those in the sham group. Exercise after CCI significantly reduced ($P<0.01$) mechanical allodynia and thermal hyperalgesia ($P<0.05$) against those in the CCI group. Moreover, the level of FRAP in the CCI group was significantly ($P<0.01$) lower than that in the sham group, and the level of FRAP in the post-CCI group increased significantly ($P<0.05$) against that in the CCI group. The level of MDA did not differ significantly between groups. Level of TNF-α increased significantly in the CCI group ($P<0.001$) compared to sham group and decreased significantly in the post-CCI group ($P<0.01$) against that in the CCI group. **Conclusion:** Exercise reduces mechanical allodynia and thermal hyperalgesia induced by CCI. These effects probably mediated by increasing the total antioxidant capacity and reducing the TNF-α inflammation factor.

P16

The Effect of Reducing CCL11 on Multiple Sclerosis Treatment by Using Heterochronic Parabiosis Techniques

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Introduction: Multiple sclerosis is a chronic neuroinflammatory disease that leads to distribute neurodegeneration in the grey and white matter of the brain. In MS, age-related iron accumulation, chronic oxidative injury and activation of microglia are key factors to create neurodegeneration. Also concentration of CCL11 is increased in multiple sclerosis. CCL11 can amplify glutamate mediated neurotoxicity and inhibit neurogenesis. **Materials and Methods:** A model of Heterochronic Parabiosis is created with joining a sick animal to healthy young animal. **Results:** It is expected by reducing CCl11, healthy young blood could be effective in slowing and improving disease. **Conclusion:** Other studies suggest that multiple sclerosis treatment should be based on a combination of anti-inflammatory, regenerative, and neuroprotective strategies. By using heterochronic parabiosis has been shown that, young blood can rejuvenate and improve the regenerative capacity of peripheral tissues and central nervous system in aged animals. GDF11 is a systemic 'pro-youthful' factor in young parabiont, that promote neurogenesis and rejuvenate regeneration capacity of CNS and promote tissue regeneration. So it seems high concentration of GDF11 and low concentration of ccl11 in healthy young parabiont may be effective on regenerative capacity of central nervous system in MS animals.

P17

Electrophysiological Effects of Cannabinoid Receptor Antagonist AM251 on Harmaline Toxicity in Rat's Cerebellar Vermis Slices

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Introduction: The Cannabinoid receptors (CBR) densities are high within the cerebellum. Cannabinoid receptors manipulations have been reported to cause altering the cerebellar functions. *harmaline* have immune-modulatory effects in several studies. i.e., significant anti-inflammatory effect via the inhibition of prostaglandin E2 (PGE2) and tumor necrosis factor alpha (TNF-α). Endocannabinoid system has some therapeutic effects in the nervous system. In the present work, the electrophysiological effects of central cannabinoid receptors modulation, particularly in the cerebellum, upon harmaline neurotoxicity were studied using whole cell patch clamp recording in the current clamp mode. **Materials and Methods:** In this study whole cell recording done from soma of purkinje cells in cerebellar vermis slices. The changes in voltage and the active and passive properties of membrane in Current clamp configuration recorded. Rats aged 4 weeks selected and grouped in 3, the control, harmaline and the AM251 as cannabinoid antagonist. Whole cell patch clamp performed on purkinje cells. All data records and reported by appropriate graphs. **Results:** Data from spontaneous activity showed despite some significant change seen in comparison to control but no effect on deteriorations caused by harmaline. In positive charge protocols there are significant decrease in number of action potential in 500 ms positive charge. Also significant decreased in number of rebound action potential in response to applying negative charge. Number of action potential event during 500 ms positive charge (0.1 to 0.5 nA) by 0.1 nA and 0.2 nA

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significantly decreased in comparison to control and harmaline groups but not in higher charges. **Conclusion:** The result showed some degree of protective effects of cannabinoid antagonist against deterioration caused by harmaline. It may be suggest that this parameter will be best test by other cannabinoids and different doses of these compounds.

P18

Alterations of Electrophysiological Activity of Cerebellar Pukinje Cells of Rats Under Harmaline Toxicity

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Introduction: Beta-carboline alkaloids of *P. harmala* are shown to have immune-modulatory effects in several studies. Extracts of this plant have significant anti-inflammatory effect via the inhibition of some inflammatory mediators including PGE۲ and TNF-α. In postmortem studies, structural alterations to the cerebellum have been recognized, including Purkinje cell loss being reported in some studies suggesting that Purkinje cell deterioration may be dominant to pathogenesis of this toxicity. In this study, we aimed to test the effects of this agent on electrophysiological properties of purkinje cells by whole cell patch clamp recording method. **Materials and Methods:** To test the effects of the harmaline, rats aged 4 weeks selected and electrophysiological test was performed on cerebellar slices by whole cell patch-clamp recording. Experimental groups including, control (saline 0.5 ml) and harmaline (30mg/kg). For investigating cells electrophysiological properties, we designed three sets of experiments including spontaneous activity, positive and negative evoked charges. After the animals deeply anesthetized, the Vermises of their cerebellum were dissected and sliced, then under video microscopy, the whole cell patch-clamp was performed and spontaneous activity as well as positive and negative charges applied to system, recorded. All activities analyzed in Pclamp software and transferred to Prism 6 and reported as appropriate graphs. **Results:** Harmaline exposure induced sever alterations in the spontaneous and evoked firing behavior of purkinje neurons in purkinje cells as evidenced by a significant decrease in the mean number of spikes, half width and instantaneous frequency. Also reduced AHP, action potential amplitude were seen. **Conclusion:** These results suggest that harmaline induced sever alterations in electrophysiological properties of purkinje neurons that may use to explain some behavioral effects of its toxicity like Tremor seen in dose dependent manner reported in some studies. We suggested that use of other member of this compound like Harman and also their effects in behavioral characteristics for confirmation.

P19

The Effects of Aloe Vera Extract on Brain Edema and Blood-Brain Barrier Permeability after Traumatic Brain Injury

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Introduction: Recent studies have reported that the Aloe vera (Aloe barbadensis miller) plant has anti-inflammatory and antioxidant effects. This study evaluated the neuroprotective effects of different doses of Aloe vera extract after traumatic brain injury (TBI) in male rats. **Materials and Methods:** In this study, 70 male rats were divided into 2 groups; each group consists of 5 of sub-groups as following: sham, TBI, TBI + vehicle, TBI + Aloe vera extract (low dose, 200mg/kg) and TBI + Aloe vera extract (high dose, 400mg/kg) groups. TBI was induced by the Marmarou method, and Aloe vera extract was administrated intra peritoneal (ip) 30 min after TBI. Brain edema was evaluated by measuring brain water content 24 h after the TBI and blood-brain barrier (BBB) permeability was determined by measuring Evans blue dye content 5 h after the TBI. **Results:** Our results showed that brain water contents was no significant difference in TBI group compared to TBI + vehicle group (P<0.687). But Aloe vera extract administration after TBI in different doses (200, 400 mg/kg) significantly reduced brain water content in TBI group compared to TBI + vehicle group (P<0.005). Also blood-brain barrier permeability significantly increased after TBI compared to vehicle group (P<0.001).In addition there was no significant difference in TBI group compared to TBI + vehicle group (P<0.742). But Aloe vera extract administration after TBI in different doses

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(200, 400 mg/kg) significantly reduced blood-brain barrier permeability in TBI group compared to TBI + vehicle group (P<0.001). Of course, high doses of aloe vera could more effectively reduced the brain blood barrier permeability compared to low dose of aloe vera. **Conclusion:** The current study, show that the Aloe vera extract may be had neuroprotetection effects after TBI. However, the mechanism(s) for this effect have not yet been elucidated.

P20

Neutrophil to Lymphocyte Ratio as a Prognostic Marker in Glioblastoma Multiforme: a Systematic Review and Meta-Analysis

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Introduction: Glioblastoma multiforem (GBM) is the most common primary malignant brain tumor in adults and it is important to identify biomarkers that can predict its prognosis. The aim of this study was to systematically review the prognostic value of neutrophil-to-lymphocyte ratio (NLR) in patients with GBM. **Materials and Methods:** PubMed, Scopus and EMBASE databases were searched until February 2016 using the following search strategy: neutrophil* AND lymphocyte* AND (glioma OR glioblastoma OR astrocytoma). Two authors independently screened the retrieved articles to find all the studies that evaluated the prognostic value of NLR in GBM patients. Data extraction and quality assessment for the included studies was performed independently by two authors. Studies using Cox proportional hazards model to compare overall survival (OS) in patients with low and high values of NLR were included in the meta-analysis. **Results:** Six studies and 827 patients were included in the systematic review. Progression-free survival (PFS) was the primary outcome in two studies. One study identified lower values of NLR as a significant predictor of better PFS, but the other one showed the opposite effect. Performing a meta-analysis was not possible on these two studies. The primary outcome in six studies was OS, four of which reported NLR as a significant prognostic marker. Pooled univariate hazard ratios (HRs) of two studies for predicting OS was 1.903 (95% CI: 1.420-2.551) and pooled multivariate HRs of four studies for predicting OS was 1.564 (95% CI: 1.208-2.024). Negligible heterogeneity was observed between studies. **Conclusion:** Overall survival of GBM patients can be predicted using NLR, but its application as a predictive marker of PFS is uncertain.

P21

Epidemiology of Multiple Sclerosis in Khorasan Razavi Province

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Introduction: Multiple Sclerosis is an autoimmune disease which demyelinates neurons. It is the leading cause of non-traumatic disability in young adults in many countries. According to the atlas of MS, the number of patients with MS has increased from 2.1 million in 2008, to 2.3 million in 2013. Although several studies have been done worldwide to clarify the epidemiological patterns of the disease, researchers have not yet been able to ascertain the accurate geographical distribution, or the precise prevalence and incidence of MS. Previously, Iran has been classified as areas of low MS prevalence but this rate has significantly increased in the recent years. The latest statistics of MS patients in Khoarasan Razavi province belongs to 2013 [Ghandehari et al.] which the prevalence of MS patients was 36 in 100000. According to the increased prevalence of MS in Iran, updating these statistics is needed. The aim of this study is to investigate the epidemiological features of MS in Khorasan Razavi province in 2017. **Materials and Methods:** The demographic information of MS patients will be derived from Mashhad, Gonabad, Sabzevar, Torbat Heidarieh and Torbat jam's medical universities. **Results:** Now we are collecting our data. They will be published as soon as data collection and analysis finish. **Conclusion:** In 2013, et al. showed that Iran has the highest MS prevalence in the Middle East and Asia. Updating the present data is necessary for the exact incidence of MS.

P22

Antihypoxic Effect of Saffron Extract in Mouse Model of Hypoxic Ischemia

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Introduction: Stroke causes high rate of mortality in different societies. The medicinal uses of saffron (Crocus sativus) have a long history. Some metabolites derived from saffron stigmas exert numerous therapeutic effects due to hypolipidemic, antitussive, antioxidant, antidiabetic activities and many

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others. **Materials and Methods:** Experiment performed with 120 mice (25-35g) keeping in standard condition to accessing water, food and laboratory temperature. Mice were randomly divided into 4 groups of ten, each for 3 separate experiments. For control group administered 0/5cc normal saline. Second group administered phenytoin 100mg/kg as potent neuroprotective agent. Third group administered 80 mg/kg aqueous extract of saffron providing from Ghaenat area of southern Khorasan IP and fourth group administered phenobarbital 20mg/kg. In 3 designed experiments, mice situated in a tight glass container in hypoxic condition. In first experiment hypoxic condition done with closed container, second experiment added soda lime as CO2 absorbent and third experiment done with substitution of N2 for O2. Survival time calculated from closing the door until stopping of respiration. Data analyzed by prism 6 software. As the case of normal distribution, results reported as Mean±SE and for abnormal distribution as Median±IQR. **Results:** Results of experiment 1 (without soda lime) showed that phenytoin and saffron have significant antihypoxic effect. In experiment 2 (+ soda lime), although the saffron extract indicated significant effect in contrast to control group but its effect not as more as phenytoin. Experiment 3 (N2), saffron group indicated antihypoxic effects like experiment 1. **Conclusion:** According to mentioned experiments, saffron extract probably has neuroprotective effect but it is necessary to do supplement experiment. We suggest to study neuroprotective effects of saffron with hypoxic and ischemia intervention for different compounds of this extract.

P23

Apoptosis Following Cortical Spreading Depression in Juvenile Rats

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Introduction: Repetitive cortical spreading depression (CSD) can lead to cell death in immature brain tissue. Caspases are involved in neuronal cell death in several CSD-related neurological disorders. Yet, whether repetitive CSD itself can induce caspase activation in adult or juvenile tissue remains unknown. Inducing repetitive CSD in somatosensory cortices of juvenile and adult rats *in vivo*, we thus aimed to investigate the effect of repetitive CSD on the expression of caspases 3, 8, 9, and 12 in different brain regions. **Materials and Methods:** Toluidine Blue staining and TUNEL assay were done to measure the mean number of dark neurons formation and the mean number of TUNEL positive cells in CSD rat models. Neuronal apoptosis assay was performed by immunohistochemistry and western blotting assay. **Results:** Higher numbers of dark neurons and TUNEL-positive cells were observed in the hippocampal CA1 and CA3 regions as well as in the entorhinal and somatosensory cortices after CSD in juvenile rats. This was accompanied by higher expression of caspases 3, 8, and 9. Caspase-12 levels remained unchanged after CSD, suggesting that endoplasmic reticulum stress is not involved in CSD-triggered apoptosis. Changes in caspase expression were paralleled by a decrease of procaspase-3, -8 and -9 in juvenile rat brain tissue subjected to CSD. In contrast, repetitive CSD in adult rats did not result in the upregulation of caspase signaling. **Conclusion:** Our data points to a maturation-dependent vulnerability of brain tissue to repetitive CSD with a higher degree of apoptotic damage and caspase upregulation observed in juvenile tissue. Findings suggest a key role of caspase signaling in CSD-induced cell death in the immature brain. This implies that anti-apoptotic treatment may prevent CSD-related functional deficits in the immature brain.

P24

Evaluation of the Voxel Based Morphometry in Quantitative Analysis of Brain MRI Images

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Introduction: Voxel based morphometry is a type of statistical parametric mapping that can be used to investigate the effect of diseases such as epilepsy, Alzheimer's disease and Parkinson's disease or other agent such as skills on brain structure (white matter, gray matter and cerebrospinal fluid). The aim of this study is evaluate the effectiveness of this method in detection of differences of the two groups. **Materials and Methods:** In this study the statistical distribution of gray matter with and without applying the modulation compared. Twenty healthy anatomical model images segmented, then modulated and unmodulated gray matter extracted and it evaluated which gray matter mode have less dispersion. Also two groups with controlled differences

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in a specific region created to evaluate the efficiency of voxel based morphometry against region of interest analysis. **Results:** Explore in modulation effect led that use of unmodulated image, statistically can reveal smaller changes in gray matter of whole brain in same sample conditions. In study of local changes in desired region that created in this study, by minimizing the region size the significant difference between the two groups can reveal. This difference can show with voxel based morphometry with different methods. **Conclusion:** voxel-based morphology is appropriate in determining brain differences which are not detect visually and hardly detect by methods such as region of interest.

P25

The Facilitatory Action of Snake Venom Phospholipase A2 Neurotoxins by Which Increase the Release of Acetylcholine, May Improve Alzheimer's Disease Symptoms

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Introduction: In a serious brain disorder like Alzheimer's disease, the levels of acetylcholine (Ach) drop significantly. The gradual death of cholinergic brain cells leads to a profound loss of memory and learning ability. Acetylcholine is the chemical messenger that sends messages from one neuron to another in the area of the brain used for memory. Many of the current medications act to enhance the low levels of ACh in the patient's brain. For example; those that inhibit cholinesterase and prevent the normal breakdown of ACh. The presynaptic phospholipase A2 (PLA2) neurotoxins from snake venom including: Bungarotoxin, taipoxin, crotoxin, and ammodytoxin are primarily characterized by their ability to affect ACh release from motor nerve terminals on the indirectly stimulated twitch in vitro and respiratory failure in vivo. **Materials and Methods:** Lyophilized PLA2 neurotoxins, twitch tension recording and whole cell patch clamp recording. **Results:** All these PLA2 neurotoxins exhibit a triphasic modulation of ACh release at the neuromuscular junction on isolated mammalian nerve-muscle preparations. The first phase is a transient initial reduction in the amount of ACh release in response to an action potential, is followed by the second phase or facilitatory phase which is a period of time when ACh release is facilitated and quantal content is increased. Finally, the third phase is a progressive decline, leading to complete block of transmitter release. Although the mechanism of this facilitatory effect is unknown, but several hypotheses such as blockade of some types of K+ channels etc, have been suggested. With patch clamp experiments we found evidence that facilitation is not due to direct block of nerve terminal K+ channels. **Conclusion:** Despite elusive mechanism, understanding the mechanism by which PLA2 neurotoxins increase the release of ACh in phase II, may facilitate the development of novel therapeutic agents to improve Alzheimer's disease symptoms.

P26

The Protective Effect of Nano-Hesperetin on Memory Disorder Induced by Streptozotocinin Male Rat in Alzheimer Models

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Introduction: Intracerebroventricular (i.c.v.) injection of streptozotocin (STZ) to rodents has been reported as an appropriate model for sporadic dementia of Alzheimer's type (SDAT), characterized by a progressive impairment of memory. However, very little or nothing is known about non-cognitive behavioral effects in the STZ model. This study was carried out in order to show the protective effects of nano-hesperetin on memory disorder in the brains of Alzheimer's animal models. **Materials and Methods:** 49 male rats were divided into 6 groups: control, sham, disease group (rats were injected with STZ), 2 treatment groups receiving 10 and 20 mg/kg/day of Nano-hesperetin. Then 3 µgr/rat of (STZ) was injected to the cerebroventricular of rats of all groups except the control and sham groups. The control and sham and toxin groups received distilled water orally. The two treatment groups were gavaged by, respectively10, 20 mg/Kg of nano-hesperetin 30 days. Then, three successive weeks, recognition memory was examined by shutl box test. **Results:** The results showed that injection of STZ increases memory disorders ($p \leq 0.001$) and treatment of Nano-hesperetin effectively decrease memory disorders ($p<0.001$) and increases duration that spends in light area of Shuttle box ($p<0.01$) compared with disease group. **Conclusion:** the treatment with Nano-hesperetin cause the protect cholinergic neurons against memory disorder in the Alzheimer rat

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model.

P27

The Beneficial Effect of Aminoguanidine on Lipopolysaccharide -Induced Memory Impairment and Neuro-Inflammation in Rats

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Introduction: In the present study, the effect of an inducible NO synthase (iNOS) inhibitor, aminoguanidine (AG) on lipopolysaccharide (LPS)-induced memory impairment and oxidative stress and inflammation parameters was evaluated. **Materials and Methods:** The rats were divided into 5 groups and treated: 1) Control (Saline), 2) LPS (1 mg/kg), 3-5) AG 50, 100 and 150mg/kg 30 min before LPS injection. The treatment was started 5 weeks before the behavioral experiments and continued during the behavioral tests (LPS injection two h before each behavioral experiment). Finally, brain tissue was removed for biochemical measurements. **Results:** the escape latency in Morris water maze (MWM) test and the latency to enter the dark compartment in Passive avoidance (PA) test in LPS group were significantly higher than in control ($P<0.001$) whereas, in AG100-LPS and AG150-LPS groups they were shorter than LPS group ($P<0.001$). Malondialdehyde (MDA) concentration in the hippocampus of LPS group were higher than control group ($P<0.001$) while, in AG100- LPS and AG150-LPS groups it was lower than LPS group ($P<0.001$). The thiol content in the hippocampus of LPS group reduced compared to control group ($P<0.001$) while, in AG100 - LPS and AG150-LPS groups it enhanced compared to LPS ($P< 0.01$). **Conclusion:** It is suggested that LPS induced neuroinflammation, brain tissues oxidative damage and learning and memory impairments are preventable by aminoguanidine as an iNOS inhibitor.

P28

Bone Marrow-Derived Mesenchymal Stem Cells Reduces Neuroinflammation and Splenic Cytolytic CD8 + T Cells in Mice with Experimental Autoimmune Encephalomyelitis

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Introduction: Multiple sclerosis (MS) has been recognized as a common neurodegenerative disease that occurs after an Auto reactive T cells against myelin antigens. Demyelination and inflammation are the main features of this disease. The anti-inflammatory and neuroprotective roles of bone marrow-derived mesenchymal stem cells (BM-MSCs) have been considered as a suitable treatment against autoimmune diseases. Previous studies have shown that treatment with BM-MSCs may regulate immune responses and improve the symptoms in experimental autoimmune encephalomyelitis (EAE) mice, an animal model of multiple sclerosis. Therefore, the present study was designed to evaluate immunomodulatory effects of BM-MSCs in the treatment of myelin oligodendrocyte glycoprotein (MOG) 35-55-induced EAE in C57BL/6 mice. **Materials and Methods:** MSCs were obtained from the bone marrow of C57BL mice, cultured with DMEM/F12, and characterized with flow cytometer for the presence of cell-surface markers for BM-MSCs. Following three passages, BM-MSCs were injected intraperitoneally into EAE mice. Immunological responses of the transplantation were evaluated. **Results**: The results demonstrated that BM-MSCs transplantation in EAE mice significantly reduced inflammation infiltration and demyelination, enhanced the immunomodulatory functions, and inhibited progress of neurological impairments compared to control groups. **Conclusion**: This study suggests the potential of BM-MSCs to induce immunomodulatory and anti-inflammatory roles in the treatment of neuroinflammatory disorders.

P29

Effects of Different Doses of Some GABAergic Agents in Mouse Brain Under Hypoxic State: Possible Role in Neuro-Inflammation

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Introduction: Pro-inflammatory chemokines and

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cytokines such as MCP-1 and IL6 can activate microglial cells that has found in some neuro-inflammatory disorders. Hypoxia activates cerebral endothelial cells to release these pro-inflammatory mediators. We aimed to investigate the anti-hypoxic effects of different doses of some GABAergic agents. **Materials and Methods:** We randomly divided 150 mice into 3 drug groups (n=10) and tested them by three discrete experiments. We investigate the effects of phenobarbital, diazepam and baclofen in comparison with phenytoin as standard neuro-protective agent and normal saline as a control group. Mice in each group were undergone acute hypoxic conditions, including: 1. Closed empty chamber 2. Closed chamber with soda lime as CO2 absorbent 3. Closed chamber with the substitution of N2 with O2. The survival time (the interval time between closing the chamber cap to stop the rat's breath) was measured. The data in each group were analyzed by prism 6. **Results:** In diazepam treated groups, despite no effect seen at lower doses, a significant effect was observed at the dose of 10mg. In soda lime group phenobarbital was most effective at a dose of 40mg although it was less than phenytoin. In the other two phenobarbital groups highest effect was observed at the dose of 30mg. The most effective dose of baclofen was 20 mg in N2-hypoxic group, 30mg in the one without soda-lime and 40 mg in the group with soda-lime however it was not as effective as phenytoin in the last group. **Conclusion:** With respect of the significant and direct correlation of survival time to the dose of diazepam, it is suggested to use this drug, to alleviate the Neuro-inflammatory complications as a result of hypoxia.

P30

Effect of Fasudil on Acrylamide-Induced Cytotoxicity in PC12 Cells Through Evaluation of ROS and MTT Test

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Introduction: Acrylamide (ACR) known as a neurotoxic agent in human and animals. Previous studies have been shown; fasudil improves neurological deficit and neuronal damage. In this study, the effect of fasudil, potent Rho-kinase inhibitor, on ACR-induced cytotoxicity was evaluated using PC12 cells as a suitable in vitro model. **Materials and Methods:** PC12 cells were exposed to different concentrations of fasudil 50 ,25 ,5,10) and 100 µM) for 24 h. Then, ACR 6 mM was added. After 24 h exposure with ACR, cell viability was determined using MTT test. For evaluation reactive oxygen species production, 2, 7-dichlorofluorescein diacetate (DCFH-DA) method was used. In addition the levels of Bax and Bcl-2 proteins were evaluated using western blot analysis. **Results:** ACR Exposure increased ROS production and Bax/Bcl-2 ratio while decreased cell viability. Pretreatment with fasudil50) µM) for 24 h inhibited ROS production (*** p< 0.001) and increased viability (*** p< 0.001). Also, fasudil could decrease Bax/Bcl-2 ratio but was not significantly. **Conclusion:** The oxidative stress and apoptosis pathway play important roles in ACR toxicity on PC12 cells. Fasudil exhibited protective effects on ACR toxicity through inhibition of oxidative stress and apoptosis pathway.

P31

Reduce Inflammation and Hypothalamic Disorders in Patients with Multiple Sclerosis

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Introduction: Multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE), is a chronic inflammatory diseases and degenerative myelin in the CNS. With respect to being the main cause of this disease is unknown; the most common treatments for the disease (MS) with the aim of suppressing harmful inflammatory responses are used. Neuro transmitters having immunosuppressive properties and neuro protection, is a good candidate for the treatment of inflammatories diseases and neurodegenerative. On the other hand, can be improved it whit composing to other anti-inflammatoris. The aim of this study is the effects of noradrenalin and serotonin and vitamin B-12 in reduction of hypothalamic disorders in MS. **Materials and Methods:** After infusion of EAE in rats with intraperitoneal injection of tedium bromide and create MS-like symptoms in rats and dividing them into groups, control group and experimental group 1: Receiving combination of nor adrenaline (0.1µic/L) By injection into hippocampus by stereo taxi and injection of estrogen on the back of the neck 0.1(mg Dissolved in peanut oil) and receiving vit B12 (0.2mg), and experimental 2: nor adrenaline with dose 0.51µic/L and vit B12 (0.5 mg) For the same amount received estrogen .and experimental 3: Receiving interferon for treatment of common comparable to the proposed treatment. Then the rats were tested after treatment period of were evaluated microscopically and macroscopically and functional tests and tissue culture to reduce inflammation and lesions. **Results:** The results showed that the combination of estrogen and vitamin B-12 and noradrenaline meaningful decrease for the number of multiple sclerosis hypothalamic disorders .and reduction of hypothalamus inflammatory (By studying the histological slides). **Conclusion:** This method can be

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used as an effective treatment in reducing inflammation of the CNS and hypothalamic disorders in these patients and as a treatment protocol can be available to medicine society.

P32

The Role of CB2 Activation in Rats Under Harmaline Toxicity

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Introduction: β-carbolines are shown to have significant anti-inflammatory effect via the inhibition of some inflammatory mediators including TNF-α and PGE2. In previous studies Purkinje cell deterioration have been proposed the dominant pathogenesis of harmaline toxicity. WIN55, 212-2 is a non-selective cannabinoid CB_1 and CB_2 receptor agonist. Combination of WIN55, 212-2 and AM 251(CB1-Selective Cannabinoid Receptor blocker) can give us activation of CB2 receptor. In this study we aim to evaluate the possible protective effect of this combination against harmaline toxicity. **Materials and Methods:** 30 rats (4 weeks aged) were kept in separate cages. They were randomly distributed into 3 groups. 1) Control, 2) harmaline (30 mg/kg according to our previous pilot study) and 3) WIN55, 212-2 (1mg/kg) +AM 251(1mg/kg) as cannabinoid receptor modulation. Agents were injected i.p. Open field test was used for evaluation of rat's behavior including: mobility, Total distance movement (TDM), velocity, rearing and grooming. Also data collected from rotarod test to evaluate balance motor and balance performances and wire grip test (hanging) to asses muscle strength and balance. **Results**: Harmaline in this dose reliably affect a significant alteration in all body parts as severe tremor in which in the open field test severely decreases all parameters mentioned. On the other hand treatment with WIN55, 212-2 +AM251 increase both mobility ($p<0.0027$) and total distance movement ($p< 0.0001$) in contrast to harmaline. No significant differences were found in the velocity and rearing of harmaline group and WIN55, 212-2 +AM251. Also despite severe alteration in muscle strength and balance, there is no significant decrease in rod and hanging in rats treated by WIN1+AM251 compared to harmaline group. **Conclusion:** These results allow us to propose that treatment by WIN55,212-2 +AM251 in rats under harmaline toxicity have some positive effects on some aspects of movement by activation CB2 receptors but it needs further investigation on selective modulators and supplementary tests.

P33

Effect of Alcoholic Extract of Rosmarinus officinalis L. and Rosmarinic Acid on Inflammation Induced by Chronic Constriction Injury (CCI) Model of Neuropathic Pain in Rats

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Introduction: Rosemary, *Rosmarinus (R.) officinalis* L. is a well-known plant with several useful properties such as analgesic, anti-inflammatory and anti-neurodegenerative. It has been used in folk medicine to alleviate rheumatic pain, stomachache and dysmenorrhea. Rosemary has several constituents such as rosmarinic acid which can be responsible for therapeutic properties been noted with rosemary. The aim of this study was to investigate the potential anti-inflammatory effects of *R. officinalis* and rosmarinic acid in a rat model of sciatic nerve chronic constriction injury (CCI)-induced neuropathic pain to verify usage of rosemary in folk medicine. **Materials and Methods:** Rats underwent CCI, were treated with either normal saline, ethanolic extract of aerial parts of *R. officinalis* (400 mg/kg, i.p.) or rosmarinic acid (40 mg/kg, i.p.) from the day of surgery (day 0) for 14 days. The anti-inflammatory effects of *R. officinalis* extract and rosmarinic acid were evaluated by assessing the levels of some spinal inflammatory markers including cyclooxygenase-2 (COX2), prostaglandin E2 (PGE-2), interleukin 1 beta (IL-1β), matrix metallopeptidase 2 (MMP2) through western blotting and nitric oxide (NO) production via Griess reaction on days 7 and 14 post-surgery. **Results**: CCI rats exhibited a marked expression in the levels of inflammatory markers (COX2, PGE-2, IL-1β, MMP2 and NO) on both days 7 ($p < 0.001$) and 14 ($p < 0.001$). Rosmarinic acid and ethanolic extract of *R. officinalis* were able to decrease amounts of mentioned inflammatory markers on both days 7 ($p < 0.001$) and 14 ($p<0.001$). **Conclusion:** Our data support the traditional use of *R. officinalis* as an effective remedy for pain

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relief and inflammatory disorders. It also suggests that the ethanolic extract of *R. officinalis* and rosmarinic acid through modulating neuro-inflammation might be potential candidates in treating neuropathic pain and different neurological disorders associated with inflammation.

P34

Evidence of Panax Ginseng in Multiple Sclerosis

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Introduction: MS disease destroys the myelin of the central nervous system. MS causing damage to the myelin sheath of the brain and the spinal cord. The mechanism is hurt by the immune system .and disruption of myelin producing cells or disconnect interconnected processes by disconnect inner white. MS is an autoimmune disease when leukocyte antigen system varies autoimmune disease like MS spread. Ginseng, the root of the panax ginseng, has been popular traditional herbal medicine in korea , japan , china for thousands of years. The major component of ginseng is ginsenoside. Divided into two spheres: American origin and Korean origin. **Materials and Methods:** The literature search was performed in Mar 2017, using three electronic databases (PubMed, Web of Science and Scopus). The reference sections of the included articles were also reviewed and a search based on the first author of the articles was carried out. The search was limited to English language articles. **Results:** MS disorders fatigue, pain, depression, sexual problems, sleep disorders. The difference between two Ginseng models are in severity index of insulin and glucose in post prandial medicinal properties include advanced functions of the brain, pain, anti-tumor effect, enhance immune function ,anti-fatigue , anti-stress , anti-diabetes , ,enhance liver function , adjustment blood pressure , anti-inflammation effective in macrophages function , effective in neuroprotective mechanism, effective in the treatment of neurodegenerative disease. And neurological disorder like MS and Parkinson ginseng regulates immune cell models like macrophages, NK cells, T cell, dendritic cells. **Conclusion:** Treatment with ginseng inhibits the secretion of inflammatory mediators like TNFa and interleukin 1B .Acidic polysaccharide of panax ginseng (APG) stimulates the reduction of the brain response during experimental autoimmune encephalomyelitis (EAE).

P35

The Mediating Role of Meta-Cognitive Beliefs on the Cognitive-Executive Functions of Brain, Sleep Disorders, Optic Neuritis in Multiple Sclerosis Patients

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Introduction: Multiple sclerosis (MS) is a chronic, autoimmune, and inflammatory nervous system disease. It leads to the loss of myelin in the white matter of brain, spinal cord and optic nerves. As a chronic disease with sudden and unpredictable Side effects and complications of the disease will lead to disability and dependency in life. Causes neurologic symptoms and sig. The purpose of the present study is to examine the mediating role of meta-cognitive beliefs on the Cognitive-executive functions of brain، sleep disorders ، optic neuritis in Multiple Sclerosis Patients. **Materials and Methods:** This is an exploratory-correlative study in which new correlations between variables will be examined. The statistical population includes patie nts suffering from Multiple Sclerosis Patients referred to khorasan razavi Ms Society 100 consecutive referrals (74 women, 26 men) were selected through purposeful sampling. All participant ts completed Perfectionism Cognitions Inventory (PCI), Dysexecutive (DEX) questionnaire. Standard questionnaires quality of sleep and severity of the insomnia) (ISI)) The patients underwent clinical tests of visual functions, including visual acuity, contrast sensitivity and color visio Data analysis was done through Pearson's correlation coefficients, two-steps regression analyses and SPSS software version 16. **Results:** meta-cognitive beliefs as well as the Cognitive-executive functions of brain had a positive relationship with sleep disorders ($P<0.001$) and the relationship between meta-cognitive beliefs and degree of optic nerve involvement. **Conclusion:** It can be concluded that the relationship between meta-cognitive beliefs and Cognitive-executive functions of brain، sleep disorders ، optic neuritis in Multiple Sclerosis Patients is not a simple linear one. This is partly mediated by meta-cognitive beliefs deficit.

P36

Protective Effects of Crocin on D-Galactose Induced Aging Model in Human Neuroblastoma Cells

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Introduction: D-galactose (D-gal) is well known as an appropriate agent to induced aging effects in the in vivo and in vitro models. In the present study, we selected crocin, the main constituent of Crocus sativus L. (Saffron), against D-gal cytotoxicity in human neuroblastoma SH-SY5Y cells. **Materials and Methods:** Cells were pretreated with crocin (25-500 µM) for 24 h and then exposed to D-galactose (25–400 mM) for 48 h. MTT assay was used for Cell viability investigation and dichlorofluorescin diacetate assay (DCF-DA) was used to evaluate the generation of reactive oxygen species,. Betagalactosidase aging marker studied in D-gal treated cells at 200 mM with or without 24 h crocin 500 µM pretreated. Also advanced glycation end products (AGEs) expression which are known as the main mechanism of age-related diseases were measured by western blot. **Results:** The finding of our study showed that treatment of cells with D-gal significantly decreased cell viability and Senescence *beta-galactosidase* (SA-Bgal) staining positive cells. Also D-gal caused increase in carboxymethyl lysine (CML) expression, is an AGE protein, and reactive oxygen species (ROS) level which are the main factors in age-related diseases. Crocin pretreatment significantly reduced D-gal neurotoxic effects. **Conclusion:** Treatment of SH-SY5Y cells with crocin before adding of D-gal dose dependency restored aging effects of D-gal. This finding indicated that crocin has potent anti-aging effects through alleviating of AGEs and ROS formation.

P37

Prescribing Pepper for Stroke Treatment

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Introduction: Black pepper (Piper nigrum L.) is one of the medicinal plants being used to prevent and manage stroke in Persian Medicine. Current findings support its anti-inflammatory, radical scavenging and neuroprotective characteristics. In this study, we aimed to evaluate role of black pepper on functional and histopathological outcomes of stroke in an animal model of MCAO. **Materials & Methods:** Black pepper powder was extracted using hydro-alcoholic solvent and drying by rotary evaporated apparatus. Black pepper suspension was then produced by suspending the powder in distilled water. Stroke was modeled by 70-80 min intraluminal filament occlusion of right middle cerebral artery in two groups of male adult Wister rats weighing 300-350 g (n:5 each group). The suspension was gavaged to one of the groups twice a day to a total dose of 200 mg (of the dry extract)/kg/d. After about 24 h, animals were tested using neurological severity and object recognition tests. Then brains were extracted and infarct volume, as well as edema, were evaluated using Triphenyl Tetrazolium Chloride staining. **Results:** Infarct volume showed significantly smaller amounts (260.8 +/- 99.28 mm^3) in pepper-treated group compared to control group (MCAO without treatment) (506.6 +/- 84.27 mm^3; $p<0.05$). Edema was also significantly less in pepper treated (51.8 +/- 29.89 mm^3) versus control group (145.7 +/- 26.40 mm^3; $p<0.05$). We didn't measure significant difference in Neurological Severity Scores and object recognition indices between two groups. **Conclusion:** Black pepper as an anti-inflammatory, antioxidant and neuroprotective plant could be considered a helpful adjuvant treatment in stroke, protecting brain from expansion of infarct size and increasing edema. Lack of significant influence on rat function by pepper might be due to the small size of the groups and limited time of follow-up after stroke. Further research with larger animal groups and longer durations of treatment is suggested.

P38

The Relationship between Personality Traits and Cortisol Levels and TGF-B in Patients with Multiple Sclerosis Have Optic Neuritis (Optic Neuritis)

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Introduction: Multiple sclerosis is a progressive neurological disease is most common in young adults. This disease has created many problems for patients and it leads to disability and dependence in life. Clinical findings indicate that mental stress of everyday life events gradually work different body systems including the immune system is affected and can weaken it. Various factors can modulate or enhance the effect. This study aimed to investigate the relationship between

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cortisol levels and components of TGF-B with five personality factors have been conducted in patients with multiple sclerosis. **Materials and Methods:** This study is a comparative study included 100 patients (27 men and 73 women) of patients with multiple sclerosis referred to the support forum of MS patients in Mashhad city that were selected available sampling. The research tools include NEO Personality Inventory short form and blood sampling to determine the levels of cortisol and TGF-B was carried out on patients. The data was used for the analysis of indicators and statistical methods including mean, standard deviation and test T. **Results:** In the five-factor personality dimension neuroticism only significant difference between male and female patients. The results also showed a significant difference between personality factors and levels of cortisol (p=0/001). There was also a significant relationship between personality factors and TGF-B (p=0/09). **Conclusion:** Psychotherapy with consideration of the traumatic personality traits can be effective in the treatment of physical symptoms in patients with multiple sclerosis.

P39

Screening of Anxiety, Depression and Quality of Life in Epileptic Patients and Their Family

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Introduction: The relationship between epilepsy and psychiatric disorders is an important issue for researchers which affect the quality of life among patients. Psychiatric comorbidities such as depression and anxiety, is associated with negative course of epilepsy, more complications, poor drug tolerance and higher mortality rates. In addition to the epileptic patients, their families are also influenced by different psychiatric, social and cognitive outcomes but there is not enough data on this significant topic. The objective of this study is to evaluate the psychiatric comorbidities among epileptic patients and their family and determine their quality of life. **Materials & Methods:** 45 epileptic patients and 45 healthy controls with their families (or caregivers) undergo the Structured Clinical Interview for DSM-V (Diagnostic and Statistical Manual of Mental Disorders) to investigate psychiatric disorders. Beck Depression Inventory (BDI) and Hamilton Rating Scale (HAM-D) are used for determination the severity of depression and anxiety. Participants also fulfill WHO quality of life BREF (WHOQOL-BREF). Type of seizure, course of seizure, age, gender, age of onset, duration of illness, family history, past medical history, type of drug was determined in another questionnaire. **Results:** The results of this study will be presented in this congress. **Conclusion:** These findings distinguish the prevalence of comorbid psychiatric conditions among epileptic patients and their families which highlight the need to intervention for improving their societal, psychological and neuropsychological aspects that affects their quality of life.

P40

Neuroprotective Effects of Saffron Extract in Rat Brain Under Ischemia Reperfusion Model

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Introduction: There are several different molecular pathways in tissue damage by Ischemic Brain Injury. The use of antioxidants and free radical scavengers are a matter of attention by some researchers. Crocus sativus (saffron) is used previously for protective effects in ischemia state by some researchers. To assess pathologic aspects of neuroprotection of saffron in some susceptible brain area, we used ischemia- reperfusion rat model. **Materials and Methods**: Adult male Wistar Rats 250-350 g were kept in constant condition (12 hr. day, night and temperature) with Food and Water ad libitum. The animals were divided into 4 groups of 8 including: Group 1, was negative control in which ischemia was done and 1 mg normal saline injected IP. Group 2, was sham operated without induction of ischemia. Group 3, positive control in which ischemia induction and 100mg /kg phenytoin injected IP as a standard neuroprotective agent. Group 4, 80 mg/kg aqueous extract of saffron was injected intraperitoneal. Brain global ischemia was done using Four Vessel Occluding (4VO) method expatiated by Pulsinelli et al (1983) with some modification. Tissue preparation: After 72 hour's brain removed and immediately fixed with formaldehyde 10%. Thin slice (2-4 Microns) of tissues stained for H&E. **Results:** Ischemic neuronal cell was seen especially in hippocampus, but some degree of necrosis seen in other area of brain such as cerebellum, basal ganglia and cortex. In normal saline injection, ischemia and necrosis of CA1, CA2 and CA3, 4 occurred. In group 2 No necrosis has seen. Phenytoin group were slightly ischemic changes only in CA1 area. In saffron injected group the necrosis in CA1 or CA3 was compatible with group 3. **Conclusion:** the result of this study showed significant anti ischemic effect that need to more investigation for other aspects of safe use of these compound and determining of effective doses.

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P41

Meningioma Stem Like Cells and Self Assembling Nanopeptide Scaffold for Treatment of Traumatic Brain Injury in Animal Model

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Introduction: Brain injury is an important cause of morbidity and mortality worldwide and so far, there has been no absolute treatment for the damaged brain tissue. Using human stem cells with self-assembling scaffolds can be a promising method for treatment of traumatic brain injury. **Materials and Methods:** Human meningioma stem cells were isolated, cultured and then expanded into in vitro condition. The rat models of TBI were divided into 5 groups as follows: sham, PBS, stem cells, scaffold and stem cell + scaffold. To evaluate movement improvement and physical activity mNSS and EEG were used and to evaluate cell differentiation and inflammation response IHC was done. **Results:** Our results showed that mNSS were significantly improved in cell group. **Conclusion:** Tissue engineering is a new therapeutic method and can be promising in treating damaged parts of brain during traumatic brain injury.

P42

Effect of Hesperetin During Pregnancy and Lactation on Locomotor Activity and Anxiety-Like Behaviors in Animal Model of Autism

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Introduction: Autism spectrum disorders (ASD) are a heterogeneous group of neurodevelopmental disorders that are defined with a wide range of behavioral impairments including social and communication deficits. Apart from these core symptoms, a significant number of ASD individuals display higher levels of anxiety. The present study is designed to understand the antioxidant efficacy of hesperetin (HET) on locomotor activity and anxiety-like behaviors in an animal model of ASD. **Materials and Methods:** In the experimental research, pregnant rats were divided into four groups including: control, disease group (injected 500 mg/kg valproic acid at gestational day 13) and treatment groups (received 10 and 20 mg/kg/day hesperetin for7 weeks during pregnancy and lactation). Locomotor activity and anxiety-like behavioral in offspring were measured in the open-field test. **Results:** The results showed that injection of valproic acid increases locomotor activity and anxiety-like behaviors ($p \leq 0.001$) and treatment of hesperetin effectively decrease locomotor activity ($p < 0.001$) and increases duration that spends in middle area of Open field ($p < 0.01$) compared with disease group. **Conclusion:** The results showed that oral administration of hesperetin improves locomotor disorder and anxiety-like behaviors in valproic acid model of autism-like.

P43

The Role of TWIK2 Channels on Immune Cells and its Impact in EAE/ MS Pathophysiology

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TWIK2 ion channels (K2P6.1, encoded by a gene named Kcnk6) belong to the family of two-pore domain potassium channels. TWIK2 is also considered as a "silent" channel because of its inability to produce measurable currents in heterologous expression systems and its intracellular retention. It is expressed in different mammalian tissues including lymphoid organs and the vascular system. However, the role that TWIK2 channels play in the immune system is far from being understood. Previous studies have already revealed that some other members of the K2P channel family have a crucial role in the pathogenesis of multiple sclerosis (MS), an autoinflammatory disorder of the central nervous system. We aim at unravelling the reasons of TWIK2 channels being silent and their role on immune cells in respect to the pathophysiology of MS and its animal model experimental autoimmune encephalomyelitis (EAE) which might offer a novel therapeutic target. Hereby, we have investigated the distribution of TWIK2 channels in different tissues and

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cells, revealing strong TWIK2-expression levels on dendritic cells, macrophages and T-lymphocytes. Future experiments will focus on the role of TWIK2 channels in vivo by inducing MOG-specific EAE in TWIK2-/- and WT animals to investigate the disease course and perform ex vivo functional assays with MOG-specific, autoreactive immune cells.

P44

The Role of HCN Channels in T Cell Function

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Ion channels play a major role in the regulation of T cell function in health and disease. In a computer-based model, established to simulate T cells' membrane potential (VM) generation, we discovered a discrepancy between the simulation and patch-clamp recordings. The predicted VM was more hyperpolarized than the measured VM, indicating that a yet unknown, depolarizing ion current might contribute to the T cells' VM. Since TASK2 and TASK3 channels are known to be expressed on T lymphocytes and the fact that the depolarizing Ih, mediated by HCN (hyperpolarization cyclic nucleotide activated) channels, was identified as a key antagonist of ITASK in previous studies, HCN channels seem to be an ideal candidate to explain the discrepancy between measured and simulated VM. HCN channels belong to the family of pore-loop ion channels and appear in four isoforms (HCN1-4) differing in activation kinetics, voltage and cAMP dependency and size. Their role for pacemaking activities in the heart and central nervous system is well known, however their expression and potential function in T cells has not been investigated so far and is subject to this study. In PCR and Western Blot experiments we could show the expression of HCN1-4 on T cells. HCN2 channels were upregulated upon stimulatory conditions both on mRNA and protein level. Quantification for HCN1 and HCN3-4 expression is currently pending. The pharmacological blockade of Ih by ivabradine (reversible) and ZD7288 (irreversible) resulted in differential, dose-dependent effects on cell viability and altered cytokine patterns as assessed by flow cytometry or ELISA respectively. Overall these findings support a regulatory role for HCN channels in T cell function and warrant further investigations.

P45

De- and Remyelination Affect Cognitive and Locomotor Abilities in Mice

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Multiple sclerosis (MS) is an autoimmune disease of the central nervous system (CNS) characterized by inflammatory and neurodegenerative processes. One of its pathophysiological hallmarks is demyelination, a consequence of oligodendroglial cell death leading supply shortfall and missing electrical insulation to axons. Demyelination induced consequences on neuronal network activity and subsequent behavior are still not fully understood. In order to characterize neuronal functionality following demyelination, we applied the cuprizone model. A diet including cuprizone leads to full CNS demyelination in five weeks and re-introduction of normal food promotes spontaneous remyelination. Therefore, we assessed a time course of the functional consequences of myelin gain and loss in mice in six experimental groups. They were tested for short and long term memory and locomotor abilities at different time points during and after cuprizone treatment. Performing the novel object recognition test by assessing the explorations of known or novel object, we evaluated short and long term memory abilities, which appeared impaired following the cuprizone administration in a time-dependent manner. Spontaneous remyelination promoted amelioration of the performance. Additionally, treated animals did not present obvious locomotor deficits but a sustained anxiety-like behavior which only partially improves upon remyelination. Demyelination of white matter fiber tracts and cortical areas associated to memory and cognition was evaluated with immunohistological staining using the proteolipid protein as a marker for adult oligodendrocytes. Indeed, cuprizone diet dramatically decreased the number of living cells while promoting astrocytosis axonal damage and activation of macrophages which physiologically remove the debris of damaged myelin. Taken together, our results show that CNS demyelination leads to impaired cognitive abilities in rodents; an effect that seems to be recover after remyelination.

P46

The Role of Kv7-Channels in the Pathophysiology of Multiple Sclerosis

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Multiple sclerosis is an autoimmune CNS-disease characterized by inflammatory neurodegenerative events occurring with de- and remyelination. Recent evidence show that demyelinated neurons are less excitable than myelinated ones while at early stages of remyelination these neurons seem to be hyperexcitable. The latter is a transitory condition that, very likely, leads to impaired neuronal network functioning also at late remyelination stages. The mechanism underlying these conditions are not clear but recent studies indicate the Kv7 channels as potential molecular players as they were shown to be altered following demyelination and, involved in regulating neuronal excitability. Therefore, we focus on assessing their role in MS pathophysiology, both in influencing the immune-system or the neuronal functionality. Using the experimental autoimmune encephalitis model (EAE) in C57Bl/6 mice, we can show that a prophylactic treatment with the specific KV7-channel opener Retigabine improves the course of the disease. We observe that Retigabine does not change the proliferation of stimulated splenocyte in culture. By quantifying the cytokines contained in their supernatant, we see no differences in which type of immune cells are proliferating. This indicates that Retigabine does not have a direct effect on the immune system. Furthermore, we investigate the neuronal contribution to the disease by using the cuprizone mouse model of general de- and remyelination. Following myelin loss, a pavlovian conditioning paradigm shows altered cognition in vivo and altered excitability in vitro. Pharmacological modulation performed during the spontaneous remyelination following cuprizone-induced myelin loss will target neurons and try to achieve neuronal protection. Taken together, our results add some interesting information about the involvement of Kv7 channel in MS pathophysiology paving the way for further studies

P47

Occupational Toxins and Neuroinflammation

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There were many occupational toxins for central nervous system and peripheral nervous system such as toxic and heavy metals. The aim of this study was introduction of occupational toxins with neurological effects and inflammatory effects specially. It is a review article. Researcher found the important data about the occupational toxins for nervous systems. Author searched in related journals, websites and texts about the subjects. There were many occupational toxins in metals and metalloid for example; lead, mercury, manganese and arsenic, organic solvents for example; n-hexane, methyl n-butyl ketone, carbon disulfide and chlorinated hydrocarbons for example; carbon tetrachloride and trichloroethylene, pesticides for example; organophosphate, carbamate and organochloride compounds, gases for example; carbon monoxide. Some of them had inflammatory effects on nervous systems. The researches demonstrated the inflammatory effects of arsenic and mercury in the nervous systems. These were used in many industries and workplaces. Guillain–Barré syndrome was showed with these exposures. These had toxicity effects for many organ systems. Control of these hazards had importance. Ways of control were important such as substitution, engineering controls; ventilations, personal protective devices, for example; respirators and gloves. Occupational examinations were done and could be useful for prevention from their effects. These were done in preplacement, periodic, fitness for work, return to work and special examinations in the workplaces. In exposure to neurotoxins, these examinations were focused on central and peripheral nervous system. There were many occupational toxins for central and peripheral nervous systems but some of them had inflammatory effects on these systems. Prevention and protection from these are important. Occupational health teams had an important role in this situation.

P48

Pioglitazone in Early Parkinson: A Review Study

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More than 10 million people worldwide are living with Parkinson's disease (PD). The ages of people who have Parkinson are variable, usually between 50 and 80 years, the average of them is 55 years old. The symptoms of PD are progressive, and within 10 to 20. It happens when dopaminergic neurons were being degenerate. Peripheral and central inflammatory and oxidative stress pathways play a complex role in PD years. Dopamine sends massages the part body of brain that controls movement in Parkinson's patient. The mount of dopamine produced in the brain decreases; due to a person unable controls movement normally who has tremor of the parts of body. Other signs: bradykinesia, rigidity, postural instability. The reason for selecting Pioglitazone is that the inflammation effect. Pioglitazone is approved by the US Food and Drug Administration (FDA) Pharmacologic category of Pioglitazone is antidiabetic agent, Thiazolidinedione; it is the peroxisome proliferator-activated receptor γ

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(PPAR-γ) agonists. Receptor γ that regulates cellular functions such as lipid metabolism, cell growth and inflammation PPAR-γ coactivator that controls mitochondrial biogenesis and oxidative stress. It might put neuroprotective effects on Parkinson's patients. The PubMed was searched for surveys published during the recent 5 years. Chronic Pioglitazone treatment in early Parkinson's disease improved mitochondrial function in various animal models and it decreased Inflammation in some nerve cells. Study epidemiological found a person took Pioglitazone who would have lower risk of PD but Biomarker test hadn't significant data one effectiveness of Pioglitazone. There are many theories about effectiveness of Pioglitazone but a clinical trial didn't observe Pioglitazone as potential neuroprotective agent. Suggesting that more clinical trials and targetable research are needed in future.

P49

Exercise Effects on Cognitive Impairments Through Altering Neuroinflammation

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Cognitive impairments describe a state of diminished or impaired mental and/or intellectual function such as Alzheimer's disease, Huntington's disease and Parkinson's disease. As these disorders are more frequent in the elderly and due to the ageing of population, serious attention must be paid to these diseases. Exercise has shown to have preventive and therapeutic effects on cognitive impairments, both in animal models and in clinical studies with the elderly. Since exercise doesn't have the possible side effects of chemical drugs and also has good effects on patients' general health, it can be considered as a second choice to the usual drug-therapy or even an alternative way of treatment. Various exercise programs used in different studies have had different effects on patients' cognitive function; which could be the result of altered release of anti-inflammatory agents. Improved cognitive function may be the result of enhanced neurogenesis, increasing synaptic plasticity by directly affecting synaptic structure and the anti-inflammatory property of neurotrophic factors induced by exercise. Although several pathways for the anti-inflammatory effect of exercise have been suggested, there is no absolute mechanism for this property. Exercise has shown to alleviate cognitive impairments through modulating neuroinflammation and enhancing neurogenesis. By trying different exercise programs, we can find the best activities for each disease, thus maximizing the positive effects for each patient. Also in these studies, the samples are usually small and the effects of exercise without usual drugs have not been checked. To achieve this goal, further studies are needed to investigate different exercise programs in larger groups.

P50

A Review on the Role and Efficacy of Vitamins in Depression

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Depression, as a mental disorder, affects more than 300 million people of all ages every year. It is said to be associated with neuroinflammation. Depression has affected women more than men. Over the world, people are disabled due to depression rather than any other condition. Neuroinflammation is the brain's reaction to injury. Apparently there is a link between neuroinflammation and depression. Vitamins have a prominent effect on brain-related diseases such as depression. This review was done to determine the relationship of neuroinflammation and depression and in order to analyze role of vitamins on it. Vitamin B_1 is said to be effective in improving depression. The role of vitamin B_2, however, is not clearly figured out, some studies showed it is effective while others show the opposite. Vitamin B_3 is proved to have a role in depression, although further investigation is needed to realize the relationship. One study showed that B_6 is no better than a placebo in treatment of depression although in another study it was observed that deficiency of pyridoxine is related to symptoms of depression. There was observed a conflict in the role of folic acid, some studies suggested its role in improving depression while others stated that it may not reduce the condition's signs. Such a conflict exists in the matter of vitamin B_{12}. Further studies are advised to assure the idea that B_{17} may have an effect on depression due to the conflicting results of different studies.

P51

The Role of T Helper 17 in Pathogenesis of Multiple Sclerosis

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Multiple sclerosis (MS) is an autoimmune disease of the central nervous system (CNS) which causes

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demyelination of the nerve fibers. The etiology of this disease is not well understood but it is believed that T helpers play a central role in MS. Numerous findings support the view that Th17 cells play an essential role in pathogenesis of MS and IL-17 secreting T (Th17) cells have a role in inflammation and demyelination of the CNS.In some studies suggest that "There was no significant relationship between the serum levels of these cytokines and Expanded Standard Disability Stated Scale (EDSS) and disease Progression Index (PI)". New drugs targeting specific points of the Th17 pathways are already being tested in clinical trials and provide basis for the development of biomarkers to monitor disease activity. Some examples of the results of other studies are given below: 1. TGF-β negatively regulates the differentiation of encephalitogenic Th17 cells. 2. miR-27a may probably inhibit negative regulators of Th17 cell differentiation, thus promoting its differentiation while miR-214 has an adverse effect. Also both miR-141 and miR-200a show up-regulation in relapsing phase of MS patients compared to remitting and control groups. 3. IFN-β inhibits the expansion of Th17 cells in active multiple sclerosis. 4. JAK2 as a critical factor that stabilizes IFN-γR2 surface expression in Th17 cells from AMS patients, making them sensitive to IFN-γ. 5. Vitamin A modulate the imbalance of Th17 and Treg cells through multiple molecular pathways. 6. IL-11 as a new Th17-promoting cytokine. 7. CXCR3 signaling in glial cells in negatively regulating Th17 cell expansion during EAE. According to these results focusing on the role of Th17 cells and use of its pathways and their biomarkers of diagnosis and disease activity are new windows to effective therapies.

P52

Interferon beta-a1 vs. Teriflunomide for Multiple Sclerosis

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Multiple sclerosis (MS) is an inflammatory disorder, in which neurons in central nervous system (CNS) become demyelinated .MS is named for its formation of plaques due to sclerosis of myelin. Variable degree of demyelination results in mild, mediate or severe symptomatic episodes of this disease. It is commonly characterized by recurrent relapses and often followed by severe neurological disabilities. The hallmark of its symptom is optic neuritis, muscular spasms, depression, anxiety and dysphagia. Etiology of MS is uncertain. However, it is mostly believed to be an autoimmune disease in which T and B lymphocytes disintegrate oligodendrocytes which are in charge of myelin production. Therefore, Interferon beta -1a and Teriflunomide seem to improve such condition as they inhibit proliferation of leukocytes. Nonetheless, there are some inconsistencies concerning their outcome. The aim of this study was to designate one of them to optimal remedy option. 40 percent of the resources regarding Teriflunomide denied its potency for prospective treatment as a result of frequent relapse-remitting of MS, as well as liver problems, hair loss, nausea and diarrhea. 20 percent of this category confirmed 90 percent desired result and the rest ensured demanded outcome. On the other hand, though, 100 percent of the resources concerning Interferon beta-1a confirmed its efficacy with minor side effects. In a general sense, forthcoming outcome of prescribing Interferon beta-a1 for multiple sclerosis patients is foreseen more beneficial.

P53

Stem Cell Therapy for Treatment of Autoimmune Diseases (with Emphasis on Multiple Sclerosis)

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Autoimmune diseases have been described as an interesting and poorly understood group of disorders. There are many challenges in the respective scientific societies concerning the nature, causes and the therapeutic approaches of these diseases. In accordance with the evidences the nature and etiology of these disorders is multifactorial and complex but the clearest definition could be expressed as "mosaic of autoimmunity". Multiple Sclerosis (MS) is an autoimmune disease that affects the Central Nervous System (CNS) resulting in degeneration of the myelin sheaths surrounding the axons of the neurons and death of oligodendrocytes that leads to a wide range of disabilities the in MS patients. The therapy for multiple sclerosis (MS) has changed dramatically over the past decade but overally Treatment of multiple sclerosis (MS) has three aspects: immunomodulatory therapy, therapies to relieve or modify symptoms and cell therapy. Cell Therapy is an emerging form of treatment MS. There are different strategies in stem cell therapy for MS such as using autologous hematopoietic stem cells to restore the individual's dysfunctional immune system and stop inflammation, utilizing the capacity of autologous mesenchymal or other cell populations for tissue repair and/or disease modification and cell replacement approaches to generate oligodendrocytes and induce demyelination by these cells. Furthermore stem cells provide a valuable advantage to study MS and testing newly developed drugs. In the reviewed the authors discussed the Capabilities of stem cells in multiple sclerosis (MS) treatment and research. In this

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review we emphasized on the great opportunity of using stem cells and also a number of challenges ahead. At the end we pointed to novel therapeutic strategies that can be applied for treatments for MS patients.

P54

Migraine: a Brain Plasticity Response to Repetitive Stressful Occurrences

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Migraine is a primary headache which is very common

in the society. Although it is usually starts in a salient point in life span of an individual diagnostically, the repetition of acute stress response such as sleep abnormalities, anxiety, and depression are not new in this population. Therefore, they have had almost a long diary of these abnormalities. In order to understand the role/s of stressful occurrences in this population, we have oriented our objectives toward the repetition of acute stress and its effect/s on the brain plasticity response from a critical point in life to the point it starts; e. g., puberty. Based on our search of PubMed, google scholar, science direct, and nature reviews which have included four review and three basic research articles, we can conclude that migraine is more probable to occur as a result of brain plasticity response due to the repetition of acute stressful occurrences in a developmental process in the life span of an individual.

P55

Sleep Disturbance and Epilepsy: an Inflammatory Pathway

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Sleep plays a vital role in regulating physiological mechanisms in the human body. Nowadays, by the change of lifestyle and as a consequence of longer work hours and increased accessibility to media, sleep disturbance becomes a common problem in modern society. Many studies demonstrated that sleep disturbance triggers a systemic low-grade inflammation by increasing the level of several cytokines, chemokines and acute – phase proteins. Increased pro – inflammatory cytokine gene expression is reported by Irwin et al. when a night of sleep restricted to 4 hours. Sleep disturbance increases the levels of IL-6, high-sensitivity C-reactive protein (hsCRP) and IL-1b in plasma. Also, IL-1 and tumor necrosis factor (TNF) gene expression in brain (hypothalamus, hippocampus, and pre-frontal cortex) increase in response to sleep disturbance in mice. Moreover, studies showed that blood-brain barrier (BBB) disrupts by chronic REM sleep restriction in rats. These data indicates that pro-inflammatory mediators can enter the brain if sleep restriction increases the unselective transportation across the BBB. On the other hand, findings suggest that inflammatory processes can play an important role in epileptogenesis in several ways like pro-inflammatory pathways (such as IL-1β). A study on epileptic patients in 2014 showed that daily generalized motor seizures result in elevated IL-6 levels leading to increased hs-CRP. Also, in 2015, Uludag et al. found increased levels of IL-1β, IL-6, and IL-1Ra among epileptic patients and high levels of IL-1b in patients with temporal lobe epilepsy. Although findings support the idea that sleep disturbance provokes epilepsy in susceptible through inflammatory pathways, further studies is needed to make this relationship more clear. Public education on proper using of media, using herbal hypnotics with lesser side effects and paying attention to sleep hygiene in General Policies, are suggestions that help us to have a better society with healthy brains and lower epilepsy incidence.

P56

Inhibitory Effects of Curcumin, a Regulator of CD4+ T Helper Cell, on HTLV-1-Associated Myelopathy/ Tropical Spastic Paraparesis (HAM/TSP)

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Human T-lymphotropic virus type I (HTLV-1) is an endemic virus in Iran and other regions that is associated with multiple diseases including adult T-cell leukemia/ lymphoma and a chronic debilitating neuroinflammatory disease, HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP). HAM/TSP is seen in approximately 2% of HTLV-1-infected people with symptoms such as back pain, weakness or paralysis of the lower limbs, and urinary tract symptoms. Immunological and Inflammatory responses cause Tissue damage

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in HAM/TSP patients and influence proviral load of HTLV-1. Curcumin (Diferuloylmethane), a natural compound derived from rhizome of turmeric, has been shown to possess anti-oxidant, anti-inflammatory and anti-microbial characteristics. Turmeric is a GRAS (Generally Recognized as Safe) agent, which is used widely in Iranian traditional medicine. Several kinds of immune cells can be infected by HTLV-1, but CD4+ cells as main target for htlv1 drew attention to themself. Patients with HAM/TSP have an increased level of inflammatory cytokines. Recent researches focused on effect of curcumin on four subsets of cd4+ cells: t regulatory (Treg), Th1, Th2, and Th17. curcumin drive the Th17/Treg balance toward the Treg dominance, which in turn suppresses the inflammatory process. Another presumable mechanism of curcumin, as an anti-inflammatory substance, is its regulatory effect that shifts immune system from Th1 to Th2 responses and could inhibit NF-KB inflammatory pathway. Curcumin has also showed Anti-viral properties that may be attributed to direct inhibition of virus replication or due to blockage of viral replication pathways. Considering the important role of proviral load in HAM/TSP development, curcumin may be efficient in lowering this load, too. Few studies have evaluated the efficacy of curcumin in treatment of HAM/TSP, but lack of randomized clinical trials and retraction of papers on this issue due to duplication caused doubts about the efficacy of this substance and urges new researches in this field.

P57

The Effect of Biotin as a Therapeutic Agent for Progressive Multiple Sclerosis

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Multiple sclerosis is an autoimmune disease caused by damage to the myelin of the nerve cells in the spinal cord and brain, MS was classified into 4 types including: Relasping/remitting (RR) primary/progressive (PP), secondary/progressive (SP), progressive/relapsing (PR). PR MS is one of the severe forms of MS that lead to inflammation associated physical, mental and vision dysfunction. Because these intensive debilitating diseases need urgent intervention researchers vigorously searching many materials that are likely benefit effect for these patients. One of the agents under increasing considerations is Biotin. Biotin is a vitamin B and necessary cofactor for five carboxylase, acts on acetyl coenzyme 7 which enable carboxylase partly in order to increase fatty acids and product energy. In turn it can increase the rate of myelin synthesis. In this review we aimed to investigate literatures in effect of biotin on progressive form of MS. According to articles that reviewed here the Biotin intake at a dose of 300 mg in patients with MS lead to promoting remyelination and increase axon myelination, also it can act as hypoxia reduction and can be improved disturbances seen in optic nerve damage. Also in some cases improved fatigue, dysarthria, swallowing difficulties, gait ataxia, sensory signs and urinary dysfunction. Thus, we can suggest, performing more clinical trial in biotin application in the situation of progressive Multiple Sclerosis and also investigating on efficacy of this agent.

P58

Herbal Medicine “Ginseng” as Therapy and Prevention of Parkinson`s Disease

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Parkinson`s disease (PD) is the second most common neurodegenerative disorder after Alzheimer`s disease. It is a chronic and progressive disease, and mostly afflicts elderlies. PD starts with sudden onsets and the exact cause of the disease is still unknown, however there are some early symptoms that can help us to diagnose the disease and ameliorate its effects. PD is a multi-factorial disease, so finding a certain cure is difficult, but recent studies about the role of neuroinflammation and oxidative stress in PD has opened a new window. In fact, in PD inflamed microglias produce the free radical “nitrogen monoxide” which causes neurodegeneration, and neurodegeneration in Substatia Nigara reduces the secretion of dopamine which results in motor symptoms of the disease. These findings clarify that anti-oxidants and anti-inflammatory mechanisms can be very helpful in the PD treatment. Recent research about prevention or reduction of the effects of PD using herbal anti-oxidant medications are promising.one of the herbal medications thought to be effective in PD is Ginseng extract. Studies show significant ant-inflammatory effects of Ginseng which can prevent formation of inflamed microglias at the first stage, also Ginseng changes antioxidant enzymes activity and nitric acid production resulting in restriction of free radicals and controlling oxidative stress. Based on these facts Ginseng is a suggested neuroprotective therapy for PD, but of course using it as an accepted therapy needs enough trials with reassuring results.

P59

Non-Steroidal Anti-Inflammatory Drugs as a Prevention of Alzheimer Disease: Risks and Benefits

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Alzheimer's disease (AD) is a chronic neurodegenerative disease that accounts for 60 to 80 percent of all dementia cases. The exact cause of Alzheimer`s disease is still unknown, but recent studies suggest neuro-inflammation as an important part of the pathogenesis of the disease. This brings in mind using non-steroidal anti-inflammatory drugs (NSAID) as treatment or prevention of the disease. In this review, we weigh up some pros and cons of NSAIDs as Alzheimer`s prevention. Although we know that most of the trials show effectiveness of long-term NSAID use in prevention of Alzheimer's disease, we cannot ignore disadvantages of using them. For example, it is proved that many inflammatory proteins and cytokines have useful and protective functions so we cannot simply suppress them by anti-inflammatory drugs because it may be more harmful than beneficial. Even some studies show increased risk of Alzheimer's disease in heavy NSAID users. Another major disadvantage of NSAIDs is their side effects such as gastrointestinal and nephrologic complications. Using NSAIDs as prevention of Alzheimer`s has been matter of much debate in recent years. We know that still there is no rational explanation for NSAIDs benefit in Alzheimer`s disease, while their harms are quite obvious. Based on these facts, NSAIDs are not yet a recommended medication for the treatment or prevention of Alzheimer`s disease.

P60

The Effect of Melatonin as a Therapeutic Goal on Multiple Sclerosis Through Immune Processes

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Multiple sclerosis is an autoimmune disease of the central nervous system which is accompanied by demyelinating the neurons. The imbalance between the T cell effectors and T cell regulators is thought to have a role in the pathogenesis of the disease. Melatonin is a hormone that is secreted by pineal gland which has an important role in circadian rhythm and immune-modulatory effects. Melatonin has an effect on the regulatory T cells and cytokines that suggests its role as a therapeutic target. The researchers experimented that melatonin is reduced in some MS patients which could increase pro-inflammatory cytokines such as TNFα. We review the effect of melatonin on the immune system and clinical symptoms of MS patients. The purpose of this study was to determine the effect of melatonin in improvement the symptoms and down regulation in the immune system of MS patients.

P61

MicroRNA as a Therapeutic Tool to Prevent Blood Brain Barrier Dysfunction in Neuroinflammation

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Endothelial cells present in brain are unique and differ from other peripheral tissues in a number of ways, which ensures specific brain endothelial barrier properties. Endothelial dysfunction is the earliest event in the initiation of vascular damage caused by inflammation. Various microRNAs (miRNA) have been discovered in different cellular components of the blood bran barrier (BBB). miRNAs are a family of non-protein-coding small RNA molecules that negatively regulate protein expression. Brain endothelial miRNAs regulate barrier function and orchestrate various phases of the neuroinflammatory response, including endothelial activation in response to cytokines as well as restoration of inflamed endothelium into a quiescent state. For instance a recent study showed that miRNA-181c triggered the toll like receptor 4 pathway, resulting in microglial activation and neuroinflammation. This observation suggest the miRNAs are a new set of controllers of BBB permeability under stress and pathological conditions. Pro-inflammatory cytokines affect several families of brain endothelial miRNAs that have important roles in BBB function and in angiogenesis; however, it remains to be elucidated whether these families of miRNAs cooperate during neuroinflammation and whether they form a link between neuroinflammation and angiogenesis in diseases that affect the CNS. Infiltration of leukocytes across the BBB has an important role in neuroinflammatory conditions. Some miRNAs may be able to reduce leukocyte addition to and migration across endothelium in neuroinflammation conditions. Among the highly modified miRNAs, let-7 and miR-98 were predicted to target the inflammatory molecules, CCL2 and CCL5. Further studies can clarify role of these miRNAs in prevention of BBB dysfunction in neuroinflammation.

P62

Markers of Neuroinflammation Related to Alzheimer's Disease Pathology in the Elderly

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Alzheimer Disease (AD) is a neurodegenerative disorder and the most common form of dementia. Increasing evidence suggests that Alzheimer's disease pathogenesis is not restricted to the neuronal compartment, but includes strong interactions with immunological mechanisms in the brain. In vitro and animal studies have linked neuroinflammation to Alzheimer's disease (AD) pathology. Studies on markers of inflammation, In people with mild cognitive impairment or AD dementia Contradictory results. We suggested that distinct blood and cerebrospinal fluid (CSF) inflammatory markers are associated with biomarkers of amyloid and tau pathology in elderly without cognitive impairment or with beginning cognitive decline. For identification blood-based and CSF neuroinflammation marker associated with AD pathology and to research associations of inflammation markers with CSF biomarkers of amyloid, tau pathology, and neuronal injury. Some item identified criteria for having an AD CSF biomarker profile. The best predictor models included 8 serum or 3 CSF neuroinflammatory markers associated with cytokine mediated inflammation, vascular injury, and angiogenesis. Both models improved Resolution to Forecast an AD biomarker profile when Comparison to the reference model. In analyses separately performed in the subgroup of participants with cognitive impairment, adding the serum or the CSF neuroinflammation markers also improved the accuracy of the diagnosis of AD pathology. None of the inflammatory markers correlated with the CSF Aβ1-42 levels. Six CSF markers (IL-15, MCP-1, VEGFR-1, sICAM1, sVCAM-1, andVEGF-D) correlated with the CSF tau and p-tau181 levels, and these associations remained significant after controlling for age, sex, cognitive impairment, and APOEε4 status. Serum and CSF inflammatory markers identify the neural signature improve classification accuracy for Alzheimer's pathology in the elderly. Our results suggest that inflammation, vascular damage and angiogenesis as reflected by CSF markers are closely related to cerebral tau pathology.

P63

Role of Neuroinflammation in Depression

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The prevalence of mental health disorders continues to rise worldwide, such that it is estimated that 1in 4 individuals will be affected by a mental health disorder at some point in their lifetime. Research has found that the most prevalent mental health disorders are depressive disorder. Depression is a common condition, particularly within the aging population. The development of depression, multifactorial disorder with the signing of neuroinflammatory, appears to be associated with the body allostasis disorder. Research has linked neuroinflammation as a major contributing factor to depression diseases. Recent findings link between inflammation and depression and hypothalamic-pituitary-adrenal particular role (HPA) axis in depression have created. This article reviews the clinical and experimental studies investigating the role of axis HPA, HPA hyperactivity (resulting in increased levels of cortisol), as well as pro-inflammatory cytokine tumor necrosis factor, C-reactive protein and interleukins, in depressed patients. The main reason neuroinflammation effects on depression show lie within the dysregulation of the control and release of pro- and anti-inflammatory cytokines. This can come from an internal or external insult to the system, or from changes in the individual due to aging which peaking in immune dysregulation. The need to reduce neuroinflammation has led to extensive research into neuroprotectants. We discuss the efficacy found with nicotine, alcohol, resveratrol, curcumin, and ketamine. Our main focus will be on what research tells us about the connections between neuroinflammation and depression, and the hope that neuroprotectants research gives people suffering from depression stemming from neuroinflammation.

P64

Micro-Rna Disorder and Multiple Sclerosis

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Noncoding ribonucleic acids micro-RNA is involved in the regulation of gene expression have major roles in the post-transcriptional level. A micro-RNA alone several causes down regulation of mRNA transcript of the target. Thus, small changes in the expression of a micro RNA may lead to significant changes in gene expression are different. Micro- RNA as key regulators of immune cell lineage differentiation, maturation, maintain homeostasis and function Known natural immunity. Multiple sclerosis is a chronic inflammatory disease which is characterized by lymphocytic infiltration central nervous system, loss of myelin and axonal damage is determined. Although the causes MS remains unknown, drug targets new to focus on reducing

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central nervous system inflammation and promote healing process is essential. Studies have shown that micro-RNA of Patients with Multiple Sclerosis in the immune system and the system Central nervous system are impaired, their role in the pathogenesis of MS show. The presence of micro-RNA expression patterns in autoimmune diseases such as multiple sclerosis and their role in the pathogenesis of various diseases, new therapeutic strategies for the treatment of autoimmune diseases, inflammatory gene suggests.

P65

Soluble CD18 as an Inflammation Reducing Agent in Parkinson's Disease

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Parkinson's disease (PD) is a very common neurodegenerative disease among the elderly population. Current treatments for Parkinson's disease are based on symptom therapy but not the underlying cause of the disease. This disorder is caused neuronal death which triggers the activation of resident glial cells. This situation leads to neuroinflammation in the central nervous system. Activated glial cells produce molecular modulators such as cytokines and chemokines which induce an inflammatory environment that causes the recruitment of peripheral leukocytes. In physiological situation migration of leukocytes through blood brain barrier (BBB) into central nervous system is constrict limited due to presence of tight junctions and cell adhesion molecules (CAMs) but in an inflammatory environment because of the overexpression of intercellular CAM-1 (ICAM-1) and vascular CAM-1 (VCAM-1) on the BBB' endothelium the circulating immune cells can migrate into the brain. ICAM-1 is a glycoprotein on the surface of the endothelial and immune cells which attach to the integrin type CD18. Some studies have shown that soluble CD18 could block the receptor of CD18 in other places in body so I hypothesized that using of soluble CD18 can block receptors on the BBB's endothelium and avoid entry of peripheral leukocytes into the brain. If it happens it can prevent further cell death in the brain stem so progress of the disease will be so much slower.

P66

A Review of Novel Biomarkers Involved in the Neuroinflammation Caused by Human T Lymphotropic Virus-1

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HTLV-1 is the causative agent for a neurologic disease named HTLV-I- associated myelopathy/tropical spastic paraparesis (HAM/TSP). Paraparesis of the lower limbs which appears gradually is the most common clinical feature of this disease. It has been shown that the indirect involvement of the nervous system by lymphocytes is more probable than the direct attack of the virus to the neurons. HTLV-1 infected CD4+ T cells may primarily contribute to development of HAM/TSP. It has recently been shown that ex vivo $CD80^{+}$ B cells positively correlating to disease severity. Moreover, CD4+ CD25+ CCR4+ T cells, which mainly include suppressive T cell subsets such as regulatory T (Treg) cells under healthy conditions, have been demonstrated as the predominant viral reservoir of HTLV-1 in HAM/TSP. This unique T cell subset is shown to be abnormally increased and functionally altered in this retrovirus-associated inflammatory disorder of the central nervous system. On the other hand, HAM/TSP patients demonstrate reductions in the amount and efficacy of cellular components of innate immunity as the numbers and functions of CD56+ CD16+ natural killer (NK) cells in HAM/TSP patients are significantly lower than those observed in healthy controls. Another study has revealed that HBZ (an important HTLV-1-encoded protein) is exclusively localized in the cytoplasm of peripheral blood mononuclear Cells (PBMC) from patients suffering of HAM/TSP. Long Terminal Repeat (LTR) Circular DNA is also presented as a marker of active viral replication. LTR circles were detected both in chronically infected cells lines and also in the PBMCs of almost all HTLV-1 positive patients. Involvement of miRNAs in the HTLV-1 life cycle and in the progression of HAM/TSP has also recently gained notice. Studies of the epidemiology and pathogenesis of HAM/TSP have led to the identification of several biomarkers. However, these findings have not yet led to an optimal therapeutic strategy for this neurological disease.

P67

The Role of Neuroinflammation in Dysfunction of Adult Hippocampal Neurogenesis

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Neuroinflammation as a protective mechanism for repairing tissue damage in the central nervous system (CNS), has been classified into two types: acute and chronic. It is characterized by the activation of microglia and astrocytes and the increase levels of different chemokines and cytokines. Neuroinflammation can be harmful, and it is a common pathological feature in neurodegenerative and psychiatric conditions. On the other word, neuroinflammation effects on important processes in the brain such as adult neurogenesis. Neurogenesis is the process by which new neurons are generated during the embryonic development of CNS and in the adult brain, and it has an important role in the repairing adult brain. Therefore, we review the effect of neuroinflammation on neurogenesis. Chronic neuroinflammation can impair to hippocampal neurogenesis in the adult brain. It has been proven that chronic neuroinflammation due to increased microglial activation and increased production of pro-inflammatory cytokines (e.g. interleukin-1β, interleukin-6, and tumor necrosis factor-α.) has deleterious effects on neurogenesis. It has been also demonstrated that microglial activation by reducing cell proliferation and newborn cell survival leads to hippocampal neurogenesis dysfunction in the adult brain. It can be concluded that neurogenesis as a physiologic phenomenon in hippocampus affected by neuroinflammation. New studies in this field can be helped to treat related neurodegeneration disease.

P68

The Role of Neuroinflammation in Post-Traumatic Stress Disorder (PTSD)

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with men. Neuroinflammation is associated to anxiety and related disorders such as PTSD. It is an early, specialized immune reaction fallowing tissue damage and/or pathogen invasion in the central nervous system (CNS). Regarding the importance of anxiety disorders especially PTSD and their effect on patients' personal and social life, considering of the neuroinflammation in PTSD can be used for new treatment. Several studies have been reported that there is a relationship between neuroinflammation and PTSD. Current epidemiological evidence shows that increased expression of pro-inflammatory cytokines such as interleukin-1β, interleukin-6, and tumor necrosis factor-α, and decreased anti-inflammatory factors have a key role in creating PTSD. Also, neuroinflammation induced by inhibition of the NADPH oxidase (NOX2) can lead to trigger the PTSD symptoms. Various inflammatory markers such as Cortisol, C-reactive protein (CRP), Th1 cytokines, and Th2 Cytokines are associated with PTSD. Furthermore, micro÷glial activation may also initiate inflammation and can be involved in PTSD. Thus, scrutinized research about PTSD and understanding of creating mechanisms of this disorder is essential and can provide grounds for its treatment.

P69

Effect of Serum Zinc Element in Epilepsy Paitaints

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Epilepsy is a disorder categorized by recurrent seizures and leads to changes in neuronal death and neurogenesis. Recently the search for new targets in the therapy of epilepsy has focused on brain inflammation because brain inflammation and the associate blood brain barrier damage appears to be basic part of epilepsy pathophysiology-erythropoietin (EPO) regulates biological processes counting neuroprotection and neurogenesis in several diseases, such as epilepsy and neurological disorder in epilepsy. Significant low serum level of zinc reported in recent investigations. One of the important trace elements is zinc and high level of zinc observed in Hypothalamus. The high level of zinc observed in hypothalamus. Zinc is one of the active cofactor in several enzyme systems and have important role in regulating inflammatory and biological activity in central nervous systems. Since in the epileptic brain, the assemblage of $GABA_A$ receptors are finely zinc delicate in hippocampus, and the emergence of a zinc-delivery system is unique in the epileptic hippocampus, the formulation of a hypothesis suggested that zinc release during repetitive initiation of the dentate gyrus may lead to a failure of inhibition seizure initiation.at least This could contribute to the limbic hyper excitability and temporal lobe epilepsy. Zinc is a substantial trace element and have mildly beneficial effects in children with stubborn epilepsy, therefor further investigation especially on oral medications for intractable epilepsy

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in children recommended. According to lecture, therapy with Pharmaceutical supplements will be used as a reliable option in the treatment of obstinate epilepsy.

P70

Primary Progressive MS and Affecting Genes

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Multiple sclerosis is a CNS autoimmune disease configured by demyelination, inflammation, and degeneration of axons. This disease inflict great harms to patients. The most common problem is inability to control musculoskeletal system and decrease in mobility. These consequences could vary from patients to patients. About 10-15% of all MS patients develop primary progressive MS (PPMS). Despite the most common appearance of MS, which is progressive-relapsing MS (PRMS), PPMS affects older adults. Its process has no recovery periods, and gender distribution measurements indicate no differences. Etiology of MS is still unclear but it is believed both environmental and hereditary factors are involved. MS susceptibility in population of a specific region and immigrants indicates possibility of environmental role and knowledge, there isn't a definite way to cure PPMS. Thus, identifying risk factors might be very useful. As far as we know, there isn't a single and specific gene with certain role in PPMS susceptibility, but nearly all studies came to an agreement that Human leukocyte antigen (HLA) genes are probably the most impressive genes in MS occurrence and its process. Although some other researches mentioning non HLA genes such as Interleukin 4 (IL4) and NAD (P) H: quinone reductase 1 (NQO1), could have undeniable effects on the disease course. Thus, in this review article we divided affecting genes into HLA and non HLA related genes. Moreover there are external factors that influence genes expression such as retroviruses. With these great expansion in PPMS affecting factors, we suggest further investigations in order to achieve a certainty and remedy production improvement.

P71

A Review on the Role and Efficacy of Vitamins in Depression

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Depression, as a mental disorder, affects more than 300 million people of all ages every year. It is said to be associated with neuroinflammation. Depression has affected women more than men. Over the world, people are disabled due to depression rather than any other condition. Neuroinflammation is the brain's reaction to injury. Apparently there is a link between neuroinflammation and depression. Vitamins have a prominent effect on brain-related diseases such as depression. This review was done to determine the relationship of neuroinflammation and depression and in order to analyze role of vitamins on it. Vitamin B_1 is said to be effective in improving depression. The role of vitamin B_2, however, is not clearly figured out, some studies showed it is effective while others show the opposite. Vitamin B_3 is proved to have a role in depression, although further investigation is needed to realize the relationship. One study showed that B_6 is no better than a placebo in treatment of depression although in another study it was observed that deficiency of pyridoxine is related to symptoms of depression. There was observed a conflict in the role of folic acid, some studies suggested its role in improving depression while others stated that it may not reduce the condition's signs. Such a conflict exists in the matter of vitamin B_{12}. Further studies is advised to assure the idea that B_{17} may have an effect on depression due to the conflicting results of different studies.

P72

Serological Changes of Cytokines, in Diagnosing and Treatment Children with Autism

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Autism is a severe neurodevelopmental disorder that characterized by abnormal bilateral social interaction, deficiency in verbal and nonverbal communication, restricted interests and repetitive behaviors .autism caused by the inappropriate immune response which released several cytokines. One of the most important and main causes of autism is a defect in, the formation of the neuronal synaptic circuit. IL6 acts in this circuit. Autistic children have increased IL6 serum and CNS level. This additional IL6 made by excess activity of neuroglia or CNS resident cells or maternal IL6. In autistic cases, CNS IL6 level in frontal cortex and cerebellum is high which lead to increased brain ventricles volume, anatomical and functional changes. In the animal model of autism, BTBR mice, inhibition of IL6 trans-signaling increased active glutamate release in synaptic space that improves communicational behaviors. TH17 cells are one of the main sources for IL6, that activated by IL23and produce IL17. IL17 is to produce IL6 and TNFα. Elevation of IL6 serum level caused asthma, which is one of the

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disorders of autistic children. Decreased Extracellular density of IL2, IL15 and Glutathione is observed in autism, lead to decrease activity and performance NK cells. Finding that has been observed in 45% of cases. To investigate the association of cytokine changes behaviors in autism, the Autism Diagnostic Interview (ADR) was used. ADI results suggest that elevation serum level of IL-1β, IL12 and IFNγ causes repetitive behaviors, whereas increase IL-6 to IL-10 ratio, lead to social behaviors and interactions impaired. As a result, an increase in inflammatory cytokines causes more severe behavioral disorders. Autism immunotherapy: decrease the production of IL6 by use of Anti IL23 or inhibition brain IL6 trans-signaling.

P73

The Effect of Hypnotherapy and Cognitive Therapy in Management of Multiple Sclerosis Pain

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Multiple sclerosis is a severe disorder of the central nervous system. This chronic and progressive disease involves unpredictable episodes of inflammatory attacks. It can cause functional limitations, disability and reduced quality of life. Pain is a common and significant problem in lots of people with multiple sclerosis (MS). And it is inversely correlated with aspects of life quality in individuals with MS. Articles report a prevalence of 40-75% among the population. The presence and severity of pain in persons with MS has also been shown to be associated with catastrophizing, mood of the patient and depression. Unfortunately, relatively few treatments have been identified as efficacious for the treatment of MS-related pain. Literature reviews have concluded that hypnosis can be effective for a variety of acute and chronic pain conditions. So no pharmacological approaches such as cognitive restructuring (CR) and hypnosis have been evaluated as potential treatments for managing pain in these patients. In reducing pain intensity and pain interference from pre- to post-treatment, hypnosis was prior to progressive muscle relaxation.CR can also work and involves teaching patients to evaluate their thoughts about pain and give help to challenge them. Extra trials are necessary to show more supports for these findings in a larger population with controlling different variables. And also may determine if self-hypnosis training has any specific effects on chronic pain beyond the placebo effects. Pain is a common complaint in many MS people that affect their life quality. There are supports for the beneficial effects of self-hypnosis training for reducing pain intensity in individuals with MS. If these hypotheses are supported, then they would have important clinical implications. For example, patients with chronic pain who report high levels of pain intensity may benefit more from learning how to use self-hypnosis to control the pain.

P74

Neuro-Inflammation and Quality of Life-A Narrative Review

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Neuro-inflammation is a growing concern that is the result of nervous tissue inflammation. Some common causes that induce this problem are toxic metabolites, aging, autoimmunity, air pollution, et cetera. Glial cells and a group of proteins which are called cytokines are associated in neuro-inflammation. Some nervous diseases such as: Parkinson's disease, Alzheimer's disease and Multiple Sclerosis are caused by the inflammation of nervous system. Some studies have showed that exercising is a good preventive way for neuro-inflammation and also some drugs are produced to decrease the complications of these diseases. Based on the uprising number of people who suffer from neuro-inflammation and its following complications, it is vital to realize the relation between this disorder and the quality of life. In this narrative review, we aim to express the effect of neuro-inflammation on the quality of life among patients and find out how their life aspects may be affected by this disorder.

P75

Inflammation in the Pathogenesis of Depression

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Depression is a mental disorder that results from changes in the central nervous system (CNS) that may result from immunological abnormalities. According to the World Health Organization, major depression will become the leading cause of disability worldwide. Accumulating evidence has indicated the existence of reciprocal communication pathways between nervous, endocrine and immune systems. The immune system affects the CNS through cytokines, which regulate brain activities and emotions. Cytokines affect the activity of the two biological systems that are most associated with the pathophysiology of depression: The hypothalamic-pituitary-adrenal axis and the catecholamine/ sympathetic nervous system. Pro-inflammatory cytokines and stress are important in inflammatory

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and neurogenesis and neuroprotection. Stress induces pro-inflammatory cytokines over secretion, which result in activation the HPA axis and neurotransmitter turnover, thus leading to depression. The use of cytokine inhibitors and anti-inflammatory drug is effective in the treatment of the depression. Although there are known effective treatment for depression, fewer than half of these affected in the world. In this review calculated recent literature related to the effect of inflammation on the pathogenesis depression.

P76

Assay of Alterations of Cytokines to Remedy of Traumatic Brain Injury

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Traumatic brain injury (TBI) is a global health concern that typically causes emotional disturbances and cognitive dysfunction. It elicits a complex secondary injury response, with neuroinflammation as a crucial central component. Secondary pathologies following TBI may be associated with chronic neurodegenerative disorders and an enhanced likelihood of developing dementia-like disease in later life. The damage to the brain occurs in two phases, the initial primary phase being the injury itself, which is irreversible and amenable only to preventive measures to minimize the extent of damage, followed by an ongoing secondary phase, which begins at the time of injury and continues in the ensuing days to weeks. This delayed phase leads to a variety of physiological, cellular, and molecular responses aimed at restoring the homeostasis of the damaged tissue, which, if not controlled, will lead to secondary insults. Many of the issues that TBI patients face are thought to be mediated by the immune system. TBI induced a moderate increase in both pro- and anti-inflammatory cytokines/chemokines. Estrogen therapy following diffuse TBI has led to reducing pro-inflammatory cytokines while induced the brain IL-10 level, and the changes of cytokines by estrogen may be regarded as one of the action mechanisms of its antiedema effect. Post-injury administration of MW151 that induced overproduction of proinflammatory cytokines towards homeostasis without immunosuppression in a closed head injury model of mild TBI suppressed acute cytokine up-regulation and downstream cognitive impairment. The development of novel treatments following TBI should aim at minimizing secondary injury by modifying, rather than eliminating the inflammatory response, while creating optimal conditions for regeneration to date there is no effective treatment available to patients, and morbidity and mortality remain high.

P77

The Role of Interleukin-1 in Neurogenesis and Alzheimer Disease

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One of the most serious neurodegenerative disorders is Alzheimer disease (AD). As the population is aging and due to the fact that AD is more common in aged people, more attention must be paid to this disease. One of the main characteristics of AD is dementia which starts with loss of short term memory, then progresses and causes various brain dysfunctions such as loss of long term memory. Interleukin-1 is a pluripotent cytokine that initiates inflammatory responses in different parts of body such as brain. In a study, it has been shown that IL-1 upregulates the expression of β-amyloid precursor protein (β-APP) that plays an important role in AD pathogenesis. A previous study represents that IL-1 induces production of substrates that are essential for making neuropathological changes characteristic of AD. Another research says that IL-1 affects the pathogenesis of AD through increasing the translation of APP mRNA. Interestingly an article claims that IL-1β-driven neuroinflammation has a possible adaptive role in AD. It has been broadly demonstrated that neuroinflammation influences adult neurogenesis. The duration of inflammation leads to inhibition or promotion of neurogenesis. Hence, modulating neuroinflammatory cytokines such as IL-1 may help in the prevention of AD or halting its progression.

P78

The Role of Kynurenine Pathway in Suicidal Behavior and Depression

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According to global statistics, over 80,000 deaths occur by suicide annually. Up to 90% of complete suicides are based on psychiatric disorders specifically major depressive disorder (MDD) and bipolar disorder. Furthermore high levels of inflammation have been indicated in suicidal patients in both central nervous system and the peripheral blood. Two biological

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mechanisms that play a key role in suicidal behavior and ideation are: 1-presenting cytokine receptors on neurons in specific area, 2- Activation of kynurenine pathway of tryptophan catabolism. The kynurenine pathway is started by the conversion of tryptophan (TRP) to N-formylkynurenine. This conversion is occurred by any of these enzymes: indoleamine 2,3- dioxygenase 1 (IDO1), IDO2, or tryptophan 2,3-dioxygenase (TDO). The resulting N-formylkynurenine is converted to kynurenine (KYN), which is a precursor of bioactive compounds, such as quinolinic acid (QUIN), kynurenic acid (KYNA), picolinic acid (PIC), and 3-hydroxyanthranilic acid (3-HAA). The first evidences about the relationship between the dysregulation of the kynurenine pathway and suicidal behavior was reported in 2011. A high level of kynurenine in plasma was detected in suicidal attempters with depression compared to non-suicidal depressed patients. Based on recent study the levels of tryptophan in plasma was 40% decreased and KYN/TRP was 40% increase in suicidal adolescent with MDD, compared to non-suicidal individuals with MDD and healthy controls. Imbalance of neuroactive metabolites is a result of association between inflammation and dysregulation of kynurenine pathway in suicidal patients. Furthermore levels of kynurenine metabolites and inflammatory cytokines are increased in the cerebrospinal fluid of suicidal patients. Therefor any aberration in this pathway causes a specific pathogenic mechanism linking inflammation and suicidal/depressive symptoms.

P79

Neuroinflammation: A Common Phenomenon between Chronic Pain and Opioids

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Chronic pain is a prevalent and debilitating condition, conveying immense human burden. Suffering from chronic pain is not only caused by painful symptomatology, but also through a wide range of psychopathological and physical consequences, including depression and anxiety disorders, impaired sleep and cognition, cardiovascular morbidity and impaired sexual function, all contributing to diminished quality of life. Opioids are highly effective analgesics because they target both of sensory and emotional elements, by inhibiting pain pathways and alleviating negative affect (including depression) by engaging reward or hedonic pathways. Prolonged exposure to an opioid induces hyperalgesia and tolerance, which negatively affect pain management in turn and significantly hamper the application of opioids. Neuroinflammation occurs in a wide range of neurological disorders -from central nervous system (CNS) infection and trauma to neurodegenerative diseases and psychiatric disorders. Recently demonstrated that chronic opioid administration in rats induces a robust neuroinflammatory response via toll-like receptor 4 (TLR4) signaling in the periaqueductal gray (PAG), a key site for opioid-mediated analgesia, that drives tolerance. Morphine is a powerful analgesic for treating severe pain, Morphine tolerance is a complex physiologic process, and various mechanisms have been proposed, such as glutamatergic receptor activation and neuroinflammation. In various preclinical chronic pain models, cytokines and neurotrophic factors have been identified as pivotal mediators involved in neuroimmune activation pathways and cascades, and in neuron–glia interactions. Both chronic pain and chronic opioids promote neuroinflammation in limbic brain structures leading to the genesis of negative affective states. This negative effect may increase the likelihood of opioid misuse and addictive-like behaviors in the chronic pain population. Understanding the mechanisms underlying opioid-induced neuroinflammation is paramount to developing effective pain management strategies that minimize the risk of dependence, abuse, and long-term consequences of chronic neuroinflammation.

P80

Effects of Vitamin D on Migraine

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Migraine is a disabling headache disorder. That is characterized by recurrent unilateral pulsatile headaches. It is one of the most common neurological disorder in the world that nearly one billion of people are affected by migraines. The main Migraine's features are headaches. Accompanying symptoms are nausea, sometimes vomiting, photophobia, neck pain and muscle tension. Its attacks last several hours to 2-3 days. Migraines are believed to be related to a mixture of environmental and genetic factors. Neuroinflammation is caused by a variety of cues. It results in the release of neuropeptides which affect vascular permeability and helps to initiate proinflammatory and immune reactions at the site of injury. In Migraines calcitonin gene-related protein (CGRP) is released from perivascular nerve endings. CGRP and other factors induce arterial relaxation, thus inflammation occurs. Vitamin D is a very important compound in the human body. Nowadays vitamin D deficiency has become a worldwide problem. Vitamin D has several roles in the body, In addition to regulation of

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calcium and phosphorus serum level, which is important for bone health, vitamin D has a significant role in brain's growth, development, and function. In recent years it has been hypothesized that there is a relationship between vitamin D deficiency and migraine. The objective of this study was to review different paradigm on how vitamin D deficiency and migraines are correlated. In conclusion, recent studies have been shown there is an association between vitamin D deficiency and migraine. There is not any evidence to prove the cause of this effect but we hypothesize that the effect of vitamin D may be related to increasing calcium serum level which has a significant role in healthy brain function and suggest further study especially on inflammatory processes involved in these situations.

P81

Omega-3 Fatty Acid and Oxylipins in Management of Alzheimer Disease

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Neurodegenerative disease are characterized by the progressive loss of neurons from specific origins of the CNS .Alzheimer disease (AD) is a neurodegenerative disorder which affect brain regains that control memory and ability to learn. It is estimated that 27 million people are affected word wide and this number is expected to triple by 2050 due to increase of the population life expectancy. AD is becoming one of the most prevalent neurodegenerative conditions worldwide. Although the disease progression is becoming better understood, current medical interventions can only ameliorate some of the symptoms but cannot slow disease progression. Neuroinflammation, a specialized immune response that takes place in the central nervous system, has been linked to neurodegenerative diseases, and specially, it has been considered as a hallmark of Alzheimer disease. It plays an important role in the advancement of this disorder. Omega-3 (n-3) polyunsaturated fatty acids (PUFAs) are involved in both the reduction in and resolution of inflammation. These effects may be mediated by the anti-inflammatory and proresolving effects of bioactive lipid mediators (oxylipins) derived from n–3 PUFAs [eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)] in fish oil. Epidemiological and animal studies have suggested that dietary fish or fish oil rich in omega-3 fatty acids, (DHA) and (EPA), may have effects in psychiatric and behavioral symptoms in AD. Several studies indicate that Both DHA and EPA can reduce neuroinflammation and cognitive decline, but EPA positively influences mood disorders, whereas DHA maintains normal brain structure. The unique anti-inflammatory and pro-resolving properties of oxylipins from individual n–3 PUFAs will enable the discovery of novel disease-management strategies in AD.

P82

The Transplantation of Human Umbilical Cord Mesenchymal Stem Cells in Neonatal Strokes

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Brain injuries that caused by strokes (result of intra partum ischemia) are a frequent cause of prenatal mortality and morbidity with limited therapeutic options. Transplanting human mesenchymal stem cells (hmscs) indicates improvement in hypoxic Ischemic brain injury (HIBD) by secretion growth factor stimulating repair processes (Hmscs) known as multi potent cells which isolated from bone marrow, adipose tissue ,placenta and fetal membrane, sub amniotic umbilical cord lining membrane, etc. Serum and growth factor are two vital compounds that influence MSC properties during in vitro culturing, which are associated with malignant transformation and multi potency of MSC. Long time culturing of hmscs increase the probability of malignant transformation and also decline their multi potency. Human umbilical cord derived mesenchymal stem cells (huc-MSC) in comparison with other types of stem cells have several unique characteristics such as a higher rate of cell proliferation and clonally , inhibiting caspase 3 expression and reducing apoptotic cells in early stage and later life periods . However limitation of hich.MSC life span, hinder their clinical usage. New research on TERT (telomerase reverse transcriptase) and BDNF (brain derived neurotropic factor) modifies that UCB- MSC may have longer life span and also maintain neural differentiation. This study suggests intra cerebral transplantation of HUCB- MSC that has been co- modified by TERT and BDNF can be sufficient therapy for neonatal hypoxic ischemic brain damage in early phases.

P83

Progression of Hearing Loss in Experimental Meningitis

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Hearing loss and meningitis were correlate in some aspects. Hearing loss is the situation that can be shown in cochlear or retrocochlear defects. Cochlear hearing

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loss is caused by inner or outer hair cell damage (however cochlear hearing loss has another causes, such as defect of arterial spiral ganglion, basilar and tectorial membrane) and retrocochlear hearing loss has neural origin and meningitis is a serious disease in which there is inflammation of the meninges, caused by viral, bacterial or fungal infection (marked by intense headache and fever, sensitivity to light, and muscular rigidity). In 21 articles that reviewed patient with bacterial, viral and fungal meningitis underwent repeated audiological assessment such as audiometry and ABR (auditory brain stem response) recording. All cases of hearing loss were apparent at the time of the first assessment. The severity of hearing impairment varied from mild to profound and was frequently bilateral and irreversible. Both bilateral and unilateral hearing loss were noted. In these articles two types of hearing loss mentioned: Sensorineural and retrocochlear hearing loss. Hearing loss developed during the earliest stages of meningitis. The risk and severity of hearing loss increase with the duration of meningitis and suggested that the cochlear aqueduct is an anatomic pathway for the extension of infection from the cerebrospinal fluid to the cochlea.

P84

Daily Nutrition has a Grand but Inapparent Effect on the Procedure of Inflammatory Dementia

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Alzheimer is age-related, progressive brain degeneration disease. It destroys memory slowly. The number of people living with AD is increasing as fast as the count of aged population and extension of bad nutrition habits. The main cause of AD is not clearly known, but prior studies noted the importance of inflammation in Tau hyperphosphorylation and failure of microglia to clear AB peptide accumulation, and also the excessive production of pro-inflammatory mediators disrupt blood-brain barrier integrity and promote AB production to accelerate AD process. Daily nutrition plays a critical role in the maintenance of total health. Some of the studies have postulated a convergence between nutrition and neuroinflammation in various pathways. The purpose of this review is to determine the relationship between nutrition, inflammation, and AD show how different food's materials can suppress or support advancement of AD Further investigation and experimentation into the effect of anti-inflammatory treatments and antioxidant supplement are strongly recommended.

P85

Anticonvulsant Activity of the Leaves of Glycyrrhiza-Glabra Var. Glandulifera and Antioxidant Effect of Achillea Wilhelmsii in Epilepsy Treatment

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Oxidative stress has been suggested as a consequence and as a cause of epileptic seizures. Researches indicate Achillea wilhelmsii (A.wilhelmsii) has anti-oxidant and anti-spasmic effects on central nervous system. This plant grows in most part of Iran. An Iranian research that done in 2013 showed that the hydroalcoholic extract of Achillea wilhelmsii possesses an antioxidant effect in the brain in pentylenetetrazole induced seizure model. Also later researches in Iran on Wistar rats and mousses showed its anti-oxidant effect. Publications of traditional medicine show that Glycyrrhiza glabra L., Fabaceae, has anti-epileptic effects. This plants grows in Europe and Middle-East such as Iran, Germany Greece, France and Turkey. Many articles show its positive role in gastritis, peptic ulcers and hepatitis treatment but there are few researches about its anti-epileptic effects. A research done in Iran in 2011 showed that leaves of G. glabra have anticonvulsant activity. It indicated that anticonvulsant activity of G. glabra could be mainly attributed to the compounds of sterols/ triterpenes class present in the leaves of this plant. It is suggested to use mixture of G. glabra and A. wilhelmsii as a herbal medicine in treatment and prevention of epilepsy because of their anti-oxidant Anticonvulsant activity and their less side effects rather than anti-epileptic drugs.

P86

CD166 as a Therapeutic Target in Autoimmune Diseases

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About 3 decades ago CD6 identified as one of the first antigens expresses on the majority of T cells and a subset of B cells. CD6 regulates cellular adhesion migration across the endothelial and epithelial cells. In recent years researches indicate its role in pathogenesis of autoimmune diseases. Many researches have been done in recent years to block CD6 by CD6 mono clonal antibodies, IOR-T1 and Tu¨33, but most blockers had short time effects and didn't effect for over a month. Nowadays CD166 has been identified as the ligand of CD6. CD166 expresses in many tissues such as spleen, kidney, skin and brain. It is supposed that interaction between CD6 and CD166 has an important role in

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pathogenesis of autoimmune diseases. Few researches have done on CD166 and its blockers it but it seems CD 166 targeted therapy in autoimmune diseases such as MS shows better results rather than CD6 blockage. So targeting CD166 by mono clonal antibodies in future researches is needed and helpful.

P87

The Amyloid Beta as a Therapeutic Target in Alzheimer Disease

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Alzheimer disease (AD) is a neurodegenerative disorder marked by cognitive and behavioral impairment. Amyloid beta (Aβ) peptides are involved in AD as the main component of amyloid plaques found in the brain. Recent in vivo and in vitro studies have shown that there is a lot of substances that alter Aβ pathogenesis of AD. Aβ induces toxicity lead to increasing ROS. In the other hand, 5-HT6 and Aloe arborescence recently reported to protect cells from this effect. Additionally, Aβ oligomers interact with neurons through Nrx2a and NL1 receptors by blocking these receptors; one can reduce the Aβ-induced memory impairment. Moreover, Aβ aggregation correlates with high concentration of Fe (III) and Cu (II). And chelators decreased significantly aggregation of Aβ in synaptic cleft. By knowing the mechanism of Aβ toxicity, new therapeutic approaches can be developed to prevent AD or alleviate disability caused by it. JC-124 treatment leads to decrease levels of Aβ deposition. Bosentan, a dual endothelin receptor antagonist, offers protection against Aβ-induced endothelial damage. Anti Nrx2a andante NL1 reduces Aβ-induced memory impairment in mice. Clicaquinol inhibits disaggregation of Aβ at low pHs. In this article we review the substances that have a role in the toxicity of Aβ and can be considered as a new target for the management of AD.

P88

Matrix Metalloproteinases in Neuroinflammation

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Matrix metalloproteinases (MMPs) are a family of neutral proteinases that are important in normal development, cellular differentiation or migration, angiogenesis, neurogenesis, wound repair, and a wide range of pathological processes such as oxidative stress and neuroinflammation. MMPs have been demonstrated to increase the permeability of the blood–brain barrier (BBB) by degrading the components of the basal lamina and contribute to the neuroinflammatory response in several neurological diseases. In response to cellular stress the brain cells express both constitutive and inducible MMPs. The MMPs belong to a larger class of metalloproteinases (MPs), which includes proteins containing a disintegrin and metalloproteinase domain (ADAM). MPs have complex roles at the cell surface and in the extracellular matrix. At the cell surface they are essential for cell survival and death. MMPs are inhibited by specific endogenous tissue inhibitors of metalloproteinases (TIMPs) that regulate the activity of them. MMP inhibitors have been developed for the treatment of several neurological disorders such as Alzheimer's disease, Multiple sclerosis, spinal cord injury, and traumatic brain injury. Since MMPs have both beneficial and detrimental roles; understanding their expression in numerous CNS insults and the use of MMP inhibitors is a good topic for scientists that will help in the treatment of the neurological disease.

P89

Reduction of Neuroinflammation in Epilepsy by Using Stem Cells Derived Astrocytes

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Epilepsy is neurological disorders that afflict many people around the world with a higher prevalence rate in children and in low income countries. Temporal lobe epilepsy (TLE) is result from hippocampal sclerosis is a neurological disorder with difficult treatment. Stem cells can transform into any type of cells such as glial cells, consequently stem cells can use for medical treatment. Stem cell therapy in epilepsy result in prophylaxis against epilepsy and improve cognitive function after seizures. Astrocytes have many roles in the brain such as protection of neurons and endothelial cells, feeding, inhibiting over activation of microglia, modulate k changes, managing of extracellular ions, regulating density of y-amino butyric acid, glutamate and adenosine. Excessive activation of microglia cause brain

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inflammation that lead to epileptic seizures. Adult cell from patient have the capability to alter to embryonic cell and become stem cell by using transcription factors. Astrocytes by secretion of glial cell derived neurotrophic factor (GDNF), controlling the proliferation, adheration and movement of microglial cells .also astrocytes reduce generation of lipopolysaccharide (LPS), IL1B, TNF .astrocytes are as a source of protection mediators that decreased neuroinflammation. In this hypothesis I suggest using stem cell therapy in epilepsy to reduce neuroinflammation induced by microglia and reduce occurrence of seizures.

P90

The Role of miR-146a in Inflammatory Process of Mesial Temporal Lobe Epilepsy (MTLE): Implication for Therapy

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Epilepsy is the third most common chronic brain disorder which is characterized by an enduring predisposition to generate seizures. Mesial Temporal Lobe Epilepsy (MTLE) is the most common type of refractory epilepsy. Increasing evidence indicates that neuroinflammation plays a critical role in the pathogenesis of MTLE. Hundreds of micro-RNAs have been found to be abnormally expressed in epileptic tissues, whereas only several mi-RNAs, such as miR-146α have been reported to function in the pathological process of epilepsy. The miR-146a has been recently identified as a potentially endogenous regulator of TLR (Toll like Receptor) and cytokine receptor signaling, suggesting a link between mi-RNAs and inflammatory process in diseases like epilepsy. Up-regulation of both IL-1 and miR-146a expression levels associated with seizures in animal models and human MTLE, supporting the hypothesis that IL-1 and miR-146a are mediators of inflammation, which facilitate the epileptic process. Despite the recent advent of additional antiepileptic drugs (AEDs) and respective surgery, the treatment of epilepsy remains a major challenge. Understanding the role of miR-146a MTLE–associated pathologies may be relevant for the development of new therapeutic strategies. Therefore the aim of this paper is to introduce miR-146a as a recommended therapy for epilepsy in future studies. MiR-146a and specific inflammation related pathways, are as a probable therapy for some MTLE patients who are resistant to available AEDs.

P91

The Evaluation of Serum Level of 25-Hydroxy Vitamin D in Patients under Sodium Valproate Medication

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Epilepsy is one of the common neurologic disorders in children. Drug side-effects are one of the important problems in treating patients with epilepsy. Using the antiepileptic medication may cause different side effects such as disorder in bone and vitamin D metabolism. Sodium valproate is one of the antiepileptic medications used widely. Some of its side effects include digestive disorder, increasing of level hepatic enzymes, fatal hepatitis, decreasing level of vitamin D, and etc. The evaluation of serum level of 25-hydroxy vitamin D in patients who are treated with sodium valproate for a long time and presenting suggestions to decrease its side effects. This review study is performed by searching valid internal and external scientific databases (Science Direct, PubMed ,Google scholar, SID and etc.) by related keywords. Recent researches have shown that antiepileptic medications including sodium valproate commonly cause a decrease in vitamin D level. Also, decrease in the measurement of bone marrow density (BMD) in patients treating with sodium valproate for a long time has been proved. So, periodic measurement of the level of 25-hydroxy vitamin D in children with epilepsy who were treated with sodium valproate is suggested. Also, consuming prophylactic vitamin D is necessary for these patients. Considering the mentioned side effects, it is suggested that this drug should not be consumed by children under 2 year old and pregnant women.

P92

A Critical Balance between Repair and Demolish of Proinflammatory Factors to Improve Effects of Neuroinflammation

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One of the most important problems in neuroscience researches is the understanding what is the communication between the immune system and central nervous system. Proinflammatory factors play an important role in this communication. The dysregulation of proinflammatory factors such as cytokines and chemokines is a central feature in the development of neuroinflammation.one

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of the important cytokines is tumor necrosis factor superfamily molecules that role of this cytokine is in the activation, proliferation, differentiation ,and migration of immune cells into the central nervous system. Another important cytokines especially in the onset of inflammatory process is interleukin-1 because of overexpression of this factor which affects with produces many reactions that cause dysfunction and neuronal death. Neuroinflammation is inflammation of the nervous tissue and it is immune response to variety of cues such as infection, toxic metabolites, traumatic brain injury, or autoimmunity. The central nervous system is an immunologically privileged site because the role of blood brain barrier, it has special structure that is composed of astrocytes and endothelial cells. This review will focus on how proinflammatory factors affect neuroinflammation process.

P93

The Role of Marine Compounds in Neuroinflammation

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Extensive research in the last decade has defined that most chronic diseases display dysregulation of multiple cell signaling pathways that have been linked to inflammation. Neuroinflammation is reaction of living nervous tissue to injury. It may be initiated in response to neurological disease. Including infection, traumatic brain injury, toxic metabolites and autoimmunity. The natural compounds possessing anti neuroinflammation actions included: dietary fibers, lipids, antioxidants, phytochemicals, and microorganisms. The marine environment contains a wide range of biological and chemical diversity that can be applied to various aspects of food processing, storage, and fortification. Further, numerous marine invertebrates based compounds have biological activities and also interfere with the pathogenesis of diseases. Isolated ingredients from marine invertebrates have been shown to activity of pharmacological and are effective for the discovery of bioactive compounds. Many of these compounds (polyunsaturated fatty acids (PUFAs), sterols, proteins, polysaccharides, antioxidants, and pigments) have biologically or pharmacological activity. Role of these compounds in neuroscience research and development of new therapies targeting the central nervous system will be addressed, with particular focus on neuroinflammation. Marine natural products are chemical multiple-target molecules obtain in animals and plants, and microorganisms.

P94

The Systemic Inflammation after Spinal Cord Injury

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Spinal cord injury (SCI) actuate to complex cellular and molecular interactions within the central nervous system in a heave to repair the initial tissue damage. The pathophysiology of acute spinal cord injury (SCI) involves primary and secondary mechanisms. Neuroinflammation is an important secondary injury process in SCI. The local inflammatory microenvironment within the injured spinal cord is a collection of degenerating neurons, damaged endothelial cells, degraded myelin sheath, and this microenvironment produces various kinds of pro-inflammatory mediators. There are many other factors such as dysregulation of the neuroendocrine system and changed neuroimmune regulation that important determinant of the onset and progression of post-SCI systemic inflammation. Epidemiological analyses have unfolded a functional link between systemic inflammation and pathogeneses of post-injury complications. On the other hand cognitive impairment is associated with extensive cerebral inflammation after SCI. SCI triggers systemic inflammatory responses marked by increased circulation of immune cells and pro-inflammatory mediators, which result in the permeation of inflammatory cells into secondary organs and durability of an inflammatory microenvironment that chip in organ dysfunction.

P95

Result of Alcohol Excessive Drinking in the Brain and Varying Mental Health Side Effects

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Alcohol directly affects astroglial cell function, including inflammation-related activity. And it also affects microglial cell development and function in specific ways that interfere with microglial interactions with the immune system and with neurons. Neuroinflammatory processes might be involved in alcohol-induced brain damage. Alcohol use, misuse and getting used to it causes different kind of mental disorders. Alcohol can cause dementia and it can speed up the rate of neurodegeneration or may contribute at various mechanistic points in the genesis and sustenance of Alzheimer Disease pathology via neuroinflammation. Children prenatally exposed to alcohol can suffer from serious cognitive deficits and behavioural problems as well as from alcohol-related changes in brain structure Children with fetal alcohol syndrome (FAS) exhibit problem behaviours, such as alcohol and drug use, hyper activity impulsivity and poor socialization and communication skills. Frontal lobes are the most damaged region of the brains of alcohol abusers but other regions of the brain are also affected. When the rate of blood alcohol goes up brain damage causes impairments in judgement and decision- making and social skills. These brain changes explain the poor behavioural control and impulsivity, which tend to worsen the existing addiction problem.

P96

Role of Thrombin in Inflammatory Central Nervous System (CNS) Diseases

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Thrombin is a multifunctional enzyme which has key roles in coagulation cascade and inflammatory events. The pro-inflammatory functions of thrombin occur by different mechanisms including increasing mast cell degranulation, up-regulating the expression of cell adhesion molecules (CAMs) and promoting the secretion of inflammatory chemokines and cytokines. Dysregulated signaling functions of thrombin contributes to the pathogenesis of pro-inflammatory diseases such as coronary thrombosis, pulmonary emboli, atherogenesis, and cancer and of special interest in this poster in central nervous system (CNS) associated inflammatory diseases. In support of the proinflammatory signaling function of thrombin in inflammatory CNS disease several reports demonstrated that PAR-1activation by thrombin elevates concentration of pro-inflammatory mediators like arachidonic acid, increases neutrophil chemoattractant-1, IL-1 and IL-8 in astrocytes. Similarly, PAR-1 has pro-inflammatory role in oligodendrocyte by inducing the expression of TNF-α and MMP-9. Furthermore, thrombin with activation of PAR-4 can induce pro-inflammatory signaling pathways including mitogen-activated protein kinase (MAPK) and nuclear factor-kappa B (NF-kβ) in microglia cell line. PAR-4 also increases the expression and release of TNF-α from microglia leading to up-regulation of inducible nitric oxide synthase (iNOS) and as a consequence, incense in degeneration of dopaminergic neurons occurs. Consistently, thrombin plays a key role in the pathogenesis of neuro-degenerative diseases including stroke, multiple sclerosis Alzheimer and Parkinson. In support of these findings, it has been shown that administration of thrombin inhibitors including hirudin and a-NAPAP could decrease CNS inflammation related disease. Understanding the detail of pro-inflammatory signaling functions of thrombin and designing novel therapeutic agent to targeting this inflammatory serine protease can be a useful strategy for treatment of CNS inflammatory disorders, however the unfavorable pharmaceutical activities, toxicity, and risk of bleeding of these compounds needs to be further investigated.

P97

Neurodegeneration Induced by Tau protein

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Tau is one of several types of microtubule-associated proteins (MAPs), responsible for the assembly and stability of microtubule networks that is present only in neurons and predominantly localized in axons which its functions are tightly regulated by phosphorylation. Via as yet unknown mechanisms, tau becomes hyperphosphorylated and accompanies with neuronal degeneration, loss of synapses, aberrant calcium homeostasis, imbalanced neurotransmitter release, and ultimately with neuronal death which aggregated in several neurodegenerative diseases, collectively known as tauopathies including Alzheimer's disease (AD), several frontotemporal dementias and etc. Neurodegeneration is the progressive loss of structure or function of neurons and neuroinflammation is a critical marker of these diseases, in addition to protein aggregates. This review looks at various factors that are considered for explaining mechanism of tau proteins that can induce neurodegeneration. These different factors include two conformations of phosphorylated

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tau (*cis* p-tau and *trans* p-tau), the level of PH-tau expression, hyper phosphorylation of certain amino acids in tau proteins, mutations in the tau gene (MAPT), tau neurotoxicity and etc. Taking together, these results help to consider tau as a remarkable factor for treatment of tauopathies. The effect of tau disturbances on neurodegenerative disorders has been looked in many different ways. According to these results, tau can be studied as a potential target for drug development especially in the field of AD and other treatment options including immunotherapy.

P98

Neuroinflammation and Nanomedicine,How about Cerebral Palsy

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Cerebral palsy (CP) is the most common motor disability in children, usually occurring during fetal development; before, during, or shortly after birth, or during infancy. CP takes place in about 2.1 per 1,000 live births .There is no exact reason but birth injury or inflammation of the central nervous system, infections and hypoxia are related risk factors of this disease. Often, symptoms include poor coordination, stiff muscles, weak muscles, and tremors and they may have impairment of sensation, vision, and hearing, swallowing, and speaking. .In fact treating CP is complex, but in some ways like nanomedicine we can manage the treatment and attenuating the symptoms. Nanomedicine is a branch of medicine that uses nanoparticles and nanodevices like dendimers in diagnosis and treatment of diseases. One of them is neurodegenarative diseases that are always accompaind by neuroinflammation. There is a blood brain barrier (BBB) in CNS to prevent entering micro organisms or something like drugs so we need something to overcome this barrier (BBB) and start diagnosis or treatment of diseases like CP. The aim of this study is looking into the use of nanomedicine in CP and providing a new way without any side effect for treatment of CP. Studies on animal models show that by nanomedicine we can overcome BBB, make drugs available and treat just the area of the brain that is involved in CP , also we can apply it for diagnosis but in some studies there was a toxic effect of nonomaterial so it is offered we can make new particles or devices by material from our own body that can be absorbed fast but with out any side effect or any change in its structure when it is binded to drugs to get the best effect and generalize it for human.

P99

Self-Assembling Peptide Scaffolds as New Therapeutic Method in TBI: Focused on Bioactive Motifs

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Traumatic brain injury (TBI) is a common reason of brain tissue loss as a result of tumors, accidents, and surgeries. Renewal of the brain parenchyma is restricted by many reasons such as inimical substances produced as the result of trauma and also inflammatory responses. A strong cascade of inflammatory responses begins as a result of TBI which include recalling peripheral leukocytes into the damaged site of brain. Brain tissue engineering is a new and promising treatment for TBI which includes designing an artificial extracellular matrix (scaffold) and stem cell transplantation into the damaged site of the brain. Tissue scaffolds moderate inflammatory cascades of reactions in tissue around the injury and reduces scar formation as a result of suppressing the amount of glial cells and leukocytes. There are many substances considered as scaffolds .One of the promising and desirable scaffolds is RADA16-I because it induces supportive migration of microglia and astrocytes and also can carry stem cells. Furthermore attaching trophic motifs to the RADA16 scaffold is an effective way for inducing endogenous gliogenesis and neurogenesis. Different type of self-assembling peptide with different peptide sequences like RADA16-IKVAV (Laminin) and RADA16-BMHP (bone marrow homing peptide) are designed. Stem cells showed high viability, differentiation, and important improvement on cell spreading and adhesion on these scaffolds. Although recent studies focused on tissue engineering, using peptide based scaffolds, conjugated with bioactive motifs, still specific attention should be paid to role of the all kinds of scaffold and attaching bioactive motifs in controlling inflammatory reactions to determine their efficacy and finding the best treatment of TBI.

P100

Stem Cells as Neuroinflammatory Modulator in TBI: A Narrative Review

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Traumatic brain injury (TBI) is physical damage to the brain structure which has a high global rate of mortality

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and morbidity. TBI can cause intense inflammatory response due to accumulation of leukocytes in cerebral matrix and activation of microglia. Microglia can differentiate into M1 macrophages or M2 macrophages following the changes in biochemical properties of brain tissue. M1 sub type release cytodestructive substances that are toxic to neurons but M2 cells are anti-inflammatory neuroprotective subtype. As the time passes after TBI, the amount of M1 cells begin to increase and fraction of M1:M2 rises. Results show that use of Stem Cells can modulate inflammatory responses of immune system. Transplantation of stem cells into injury site increases M2/M1 ratio as a result of inducing M1 macrophages apoptosis. Different types of stem cells have different mechanisms for anti-inflammatory responses. Even exosomes derived from stem cell can affect the functional recovery and reduce neuroinflammation after TBI. Human Mesenchymal stem cells (hMSCs) are most used in TBI cases due to their immunomodulatory impact and therapeutic effects on recovery of motor and cognitive function. Although many studies conducted to determine effects of hMSCs on TBI prognosis, further investigations are required to support clinical use of hMSCs. Specific attention should be paid to role of growth factors and motifs in suppressing inflammatory responses. Future studies are needed to determine the efficacy of combined therapy.

P101

Ginger and Honey Mixture as an Anti-Inflammatory Drug in Neuroinflammation:Alzheimer Disease

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Nowadays, many people prefer to use herbal remedies instead of chemical treatments, because of a fewer side effects. It seems that herbal drugs can be useful to neuroinflammation diseases like Alzheimer. Alzheimer Disease (AD) is caused by the oxidative stress and inflammatory compounds such as cytokine and beta-amyloid. It causes learning problems due to damage in cells in the hippocampus area .Studies showed 60 to 70% of people over 65 years have AD. Ginger is a plant with unique properties containing vitamins (especially E and C) and Shogaol (which has anti-inflammatory properties).Ginger helps to reduce the symptoms of AD and increases the concentration power. Studies on 60 middle-aged women with AD showed that daily consumption of ginger greatly improved memory and relieved the symptoms of this disease. Vitamin E and C in ginger, maybe reduce the probability of prevalence and incidence of AD. Because vitamin E is an antioxidant and protects cells membrane's from oxidative damage. Vitamin C (soluble in water) found in ginger is oxidized and caused the reduction of vitamin E (fat-soluble) and enables it to better inhibit free radicals. Honey is another effective ingredient in improving nerve inflammation. It again contains vitamin E and C and likes ginger prevent oxidative damage. Honey has free amino acids such as glutamic acid and phenylalanine that increase concentration power and improve the function of the nervous system. This nutrient also helps the absorption of drugs. We suggest that daily consumption of ginger and honey mixture maybe increase the effectiveness and absorption of drugs and potentially prevent cells death.

P102

The Study of Some Factors Which Effect on Beta-Amyloid Signaling in Neuroinflammation

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Neurological inflammatory diseases are developing rapidly. Different factors involved in the pathogenesis of these diseases. In this article, we discuss some of the mechanisms are dealt with. An aberrant procedure of beta-amyloid precursor protein (BAPP) to form neurotoxic beta-amyloid peptides and an accumulated insoluble polymer of beta –amyloid (BA) that forms the senile plaque. The above process shows one of the major pathogenic involving in Alzheimer's disease. Actually, the mutations in the preseniline genes PS1 and PS2 cause irregular beta -amyloid precursor protein processing with consequent overexpression of beta-amyloid42 (BA42) and related neurotoxic peptides. The overexpression of RAGE (receptor for advance glycation end products) causes neuroiflammation (NI). This process caused the aggregation of beta-amyloid which increased inflammation and destruction memory. The RAGE signaling in microglia contributes to inflammatory reaction that impaires neuronal function. For improving this condition; the blockade of microglial RAGE may be effective. Intracerebral streptozotocin (i.c.stz) causes NI by increasing Ptau, ABPP, AB42 and reducing the level of synaptophysin and specially IGF1. Since T3D-959 increased IGF1, AKT and P70S6K, using it may be effective for improving brain signaling and reducing NI. Also, in another study, the usage of Tobacco had the same effects. Tobacco suppressed the expression of proteins required for signaling through AKT, P70S6K and increased ABPP-AB. With above expressions, we can conclude that BA may be the most important factors in neuroinflammatory processes. With these interpretations, we can propose that the control of the production BA and prevent the development of inflammatory diseases is gene therapy. In this way, it

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will be appropriate if we control the genes mutations to control the production of the factors which ultimately lead to overproduction of BA.

P103

Effects of Physical Exercise on Neuroinflammation

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Neuroinflammation is inflammation of the nervous tissue. It be initiated in response to infection, toxic metabolites, auto immunity, traumatic brain injury and variety of causes. Exercise is a promising mechanism of prevention and treatment for disease characterized by neuroinflammation. The benefits of exercise and physical activity (PA) are well known and have effects on function of the central nervous system (CNS), like improved mood and mental health, enhanced memory and cognitive function. PA is known to be an important preventive action in dementia and neurodegenerative disease, able to slow down progression and ameliorate disability. Previous research has established that long-term exercise reduce acute neuroinflammation resulting from traumatic brain injury. It is now well established from a variety of studies, that PA leads to decreased level of pro-inflammatory cytokines such as tumor necrosis factor (TNF)-α and interleukin (IL)-1β and increase in anti-inflammatory cytokines such as IL-10.These cytokines can cross the blood brain barrier (BBB), and can communicate between the CNS immune system and the peripheral. Exercise also induces IL-6 in the muscle tissue. IL-6 can suppress the function of pro-inflammmatory cytokines such as TNF-α and IL-1β. Clinical studies indicate that leading a physical active life-style can reduce the risk of developing Alzheimer's disease (AD) and Parkinson's disease (PD). My main reason for choosing this topic is personal interest to exercise. The most obvious finding to emerge from this study is that exercise can lead to increased levels of neurotrophic factors,changes in the level of different cytokines and altered microglial functions in different parts of the brain.More research is needed to determine parameters influencing the effect of exercise, such as intensity,in order to find the optimal program.

P104

Effects of Human Neural Stem Cells in Cure Neuroinflammation of Traumatic Brain Injury

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Traumatic brain injury (TBI) is defined as an external mechanical injury to the brain. Neuroinflammation plays a vital role in the pathophysiology of TBI. Microglia and astrocytes play a central role in the initiation and regulation of inflammation. Numerous pro-inflammatory mediators including cytokines, chemokines, reactive oxygen species (ROS) and nitric oxide (NO) released by microglia. In response to TBI, astrocytes also endure phenotypic changes, swelling in size, up regulating production of glial fibrillary acidic protein (GFAP) and Vimentin, and releasing inflammatory mediators. To date, there is no effective clinical treatment to repair neural structure and functional recovery. Cell therapy is a new strategy to repair and regenerate injured brain tissue. Adult neural stem cells (NSCs) primarily are confined to the subventricularzone and the dentate gyrus of hippocampus. Human neural stem cells are ideal candidate that can ameliorate inflammation and ongoing neurodegeneration. Transplantation of Human neural stem cells, including fetal- and iPS-derived hNSCs, should also be assessed in order to verify if an optimal cell population exists to support in the recovery of brain function after TBI.

P105

The Role of LRRK2 Inhibitors in Treatment of Parkinson's Disease

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Parkinson's disease is the second most common age associated neuron degenerative disorder in developed societies. With the prevalence ranging from 41 per 100000 in the fourth decade of life to over 1900 per 100000 in people over 80 years of age.it characterized clinically by resting tremor, slowness of movement, rigidity and postural instability in the result of progressive loss of dopaminergic neurons in the substantia nigra. Although a variety of possible pathogenic mechanisms have been proposed over the years its etiology has not yet been fully understood. Chronic inflammation is one of the etiologies of Parkinson's disease and play vital role in the degeneration of dopaminergic neurons. Mutations in the leucine-rich repeat kinase 2 (LRRK2) gene are found in Parkinson's disease, as in immune related disorder including Crohn's disease and leprosy. Increasing evidence suggests that LRRK2 protein play an important role in innate immunity. A process that occurs in neurodegenerative disease including Parkinson's disease.LRRK2 is a large and complex protein with a unique multiple domain architecture and that can function as a protein kinase with many putative substrates identified and can also function as a GTPase

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that may serve in part to regulate kinase activity. The combined genetic and biochemical evidence supports a hypothesis in which the LRRK2 kinase function is involved in the pathogenesis of sporadic and familial form of Parkinson's disease. This finding suggests that LRRK2 kinase inhibitors may potentially offer new treatment for Parkinson's disease.

P106

Effects of Dimethyl Sulfoxide on NLRP3 Inflammasome and Alzheimer's Disease

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Alzheimer's disease (AD), the most ordinary form of dementia and extracellular accumulation of Amyloid-β (Aβ) in senile plaques, is an important and a main event in the pathogenesis of AD. Deposition of Aβ Peptide initiates a spectrum of cellular responses that are interposed by the resident neuroimmune cells of the brain, the microglia. Recently, a novel inflammasome signaling pathway has been uncovered and Aβ can activate the NLRP3 inflammasome in microglia, which is fundamental for the secretion of pro-inflammatory cytokines and subsequent inflammatory events .More importantly , the activation of NLRP3 inflammasome has demonstrated a serious role in AD pathogenesis by interposing a harmful chronic inflammatory response, while inhibition of NLRP3 mainly protected from loss of spatial memory and decreased Aβ deposition in an AD mouse model. Dimethyl Sulfoxide (DMSO) is an amphipathic molecule that is widely used as a solvent for biological compounds .In addition, DMSO has been studied as a medicine for the treatment of inflammation, cystitis, and arthritis. Based on the anti-inflammatory characteristics of DMSO, the effects of DMSO on activation of inflammasomes has elucidated, which are cytoplasmic multi-protein complexes that interpose the maturation of interleukin (IL)-1β by activating caspase-1 (casp1). The aim is discussing about effects of DMSO on NLRP3 inflammasome and AD. It has proved that DMSO attenuates IL-1β maturation, casp1 activity, and ASC pyroptosome formation by NLRP3 inflammasome activators. DMSO is a selective inhibitor of the NLRP3 inflammasomes. The anti-inflammatory effect of DMSO was further proved in animal studies, LPS-endotoxin sepsis, and inflammatory bowel disease models. DMSO shows anti-inflammatory characteristics, attenuates NLRP3 inflammasome activation. According to studies, it is hypothesized that DMSO inhibits activation of inflammasomes, NLRP3, CASP1 in Alzheimer's disease that are pathogenesis by mediating a harmful chronic inflammatory response.

P107

P2x7 Receptors: as a Novel Targets for the Treatment of Neuroinflammation

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P2x7 receptors are Purineric receptors that are extracellular ATP-gated ion channel. These receptors require high dose or prolonged exposure to ATP for initial activation. The Activation of these receptors facilitates the formation of inflammasome which activates caspase 1. The P20 and P10 subunits of caspase 1 form active enzyme that then releases active interleukin (IL)-1 β and IL-18, tumor necrosis Factor-α (TNF-α), IL-6 important proinflammatory cytokines which can induce inflammation Although other cytokines such as, IL-8, IL-1α, IL-2, IL-4, IL-13 Can be released by activation p2x7 receptors. P2X7 receptors are widely expressed in neurons, microglia, astrocytes, oligodendrocytes and Schwann cells where they can induce neuroinflammation also neuroinflammation is an essential step in neurodegenerative inflammatory diseases which include: multiple Sclerosis, Alzheimer's, Parkinson's, Huntington's disease, atrophic lateral sclerosis, frontotemporal dementia, and traumatic brain injury also previous study in recent years reports overexpression of p2x7 receptors in neuroinflammation subsequently neurodegenerative diseases. In this study we aim to overview the role of p2x7 receptors in neuroinflammation as a novel targets for the treatment of neuroinflammation. P2X7R emerges as a promising target to treat neuroinflammation because this receptor is involved in the release of proinflammatory cytokines that play an essential role in the development of neuroinflammation subsequently neurodegenerative diseases so an antagonist for this receptor might halt the inflammatory cascade and thus further progression of neurodegeneration.

P108

Evaluation of Calcineurin Role in Neuroinflmmation: Possible Targets for Early Detection and Treatment

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Calcineurin (CaN) is a Ca2+/calmodulin (Ca2+/CaM)-dependent serine/threonine protein phosphatase expressed in most mammalian tissues but found at higher concentration in brain. In the last decade there have been a steadily increasing number of studies identifying

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neuronal CaN as a primary suspect in neuronal vulnerability, synapse loss, dendritic atrophy, synaptic dysfunction and neuroinflammation Subsequently despite the apparently selective association of CaN with neurons and neuronal signaling cascades, many studies found that CaN, can also appear in primary glial cells and glial cells of healthy brain tissue and astrocytes, prominently following inflammatory insult The clear connection between glial cells / astrocytes and neuroinflammatory signaling, in addition to the well-known the role of CaN in cytokine production in peripheral immune cells, suggested a potent association between glial CaN and the neuroinflammation inherent to most acute and chronic neuroinflammatory autoimmune diseases. In this review we aim to evaluate the Calcineurin role in neuroinflammation as an early event. Recent studies highly confirmed CaN as a major modulator of immune/ inflammatory processes in glial cells and astrocytes that higher expression of CaN associated with early stage of neuroinfllammatory autoimmune disease that makes CaN as novel target for early detection and treatment of neuroinflammatory autoimmune diseases.

P109

The Potential Preventive-Therapeutic Effects of Flavonol Consumption on Alzheimer's Disease

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Alzheimer's disease (AD) is considered as the greatest thread to adults aged 65 and older. It stands as the most common cause of dementia, the prevalence of which is around 24 million worldwide. AD is a progressive and degenerative disorder that combats the neurons, leading to impairment of different cognitive functions. AD is characterized by an accumulation of two factors normally present in the brain: Amyloid-Beta plaques (Aβ) and neurofibrillary tangles, outside and within the cells, respectively. Present drugs for treatment of AD have severe side effects. Thus, more researches are required to find substitutes with less harm. Flavonoid is a large group of antioxidant plant-derived compounds which can be found in high concentrations in cocoa powder and chocolate. It has been indicated that flavonols can display several roles on the brain, improving memory and learning. For optimal function of the neurons, a persistent blood flow is needed to supply adequate glucose and oxygen. Flavonoids act on the endothelium of brain vessels, stimulating NOS, causing nitric oxide (NO) to increase. A growth in the amount of NO can lead not only to the increscent of cerebral blood flow, but also a limitation in the production of Aβ. Flavonols provoke angiogenesis and neurogenesis in the sub ventricular zones and hippocampus (regions involved in memory and learning) as well. The current study was designed to determine the effect of flavonol consumption on Alzheimer's disease. Taken together, various findings have implied that long-life consumption of flavonoid-rich food, like cocoa powder and chocolate, has the potential ability to limit neurodegeneration and prevent age-related cognitive decline.

P110

Evaluating the Role of Histone Hyper Acetylation in Induction of Neuroinflammation

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Microglia is the effector cell of the innate immune system in central nervous system (CNS). These cells mediate inflammatory responses in injuries. Besides external factors, microglial function is also controlled by internal factors, including epigenetic regulations. Mechanisms of epigenetic regulation mainly consist of DNA methylation, histone modifications and use of non-coding RNAs. Recent studies have demonstrated that these epigenetic processes can alter the function of microglia and thus, adjust neuroinflammation. Neuroinflammation is believed to play a significant role in development of numerous neurological disorders, including Multiple Sclerosis (MS), which is the most prevalent chronic inflammatory disease of CNS. Therefore, it has been hypothesized that the aforementioned epigenetic processes could act as a potential therapeutic target for neuroinflammatory diseases and many studies have been performed in this field. Among various histone modifications, histone acetylation is the most studied subjet. Previous studies demonstrate that histone hyper acetylation in various tissues can contribute to inflammation. Although no studies have specifically evaluated the role of histone hyper acetylation in inducing neuroinflammation so far, but multiple studies have acknowledged the beneficial use of histone deacetylase in limiting neuroinflammation. Thus, it can be concluded that histone hyper acetylation is associated with neuroinflammation. We believe that more research is needed to assess the relationship between histone hyper acetylation and neuroinflammation, and to investigate whether or not hyper acetylation in microglia can induce inflammatory response in CNS. Moreover, we suggest evaluating the possibility of epigenetic transgenerational inheritance of neuroinflammation through histone hyper acetylation, since most of the previous studies in this field have focused on epigenetic inheritance in neuronal behaviors through miRNAs and DNA methylation.

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P111

Use of Stem Cell Therapy for Treatment of Temporal Lobe Epilepsy (TLE)

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5Epilepsy is one of the most common neuroinflammatory disorders that affect more than 50 million people worldwide. Excessive electrical discharges in neurons following neural cell damage or loss leads to recurrent seizures, which are described as epilepsy. One of the most common and difficult to treat types of epilepsy is Temporal Lobe Epilepsy (TLE), which results from hippocampal sclerosis. Currently, drug therapy is one of the most used treatments for epilepsy, but anti-epileptic drugs can induce undesirable side effects and are not effective in all TLE patients. Therefore, developing new treatments for TLE is necessary. Recently, some studies have surveyed the use of stem cells for treatment of TLE. Stem cells have numerous significant advantages over current drug therapies for epilepsy. Researchers have used various stem cells in animal models for treatment of TLE, but there is no conclusive evidence in support of using stem cells for treating TLE yet. However it is important to acknowledge that this field is still in infancy, and the initial studies are promising. Thus, we suggest more researches need to be done on the use of stem cells for treatment of TLE.

P112

Combining Granulocyte-Colony Stimulating Factor (G-CSF) and Stem Cells in Treatment of Traumatic Brain Injury (TBI)

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Traumatic brain injury (TBI) is described as a situation in which the brain is damaged by an external force. It is considered to be a chief problem in health care. Pharmacotherapy and stem cell therapy are the main treatments used in TBI. The purpose of these procedures is to control the inflammation in injured regions of brain. Currently, there is no effective care for TBI that could regenerate neurons and rehabilitate the patients. According to recent studies, there are stem cells settled on different parts of CNS that can play an important role in treatment of TBI. In stem cell therapy, we use the capacity of undifferentiated cells for healing the injured tissues. G-CSF can broadly be defined as Granulocyte-colony stimulating factor or colony-stimulating factor 3. It is a cytokine with the potential to suppress encephalitis, and control glutamate levels. Studies that used stem cell therapy and G-CSF administration in combination with each other, reported a significant increase of neurogenesis and a considerable decrease in neuron apoptosis, compared with when each of these procedures were used separately. However, few researches has been done in the field of combined therapies for TBI so far. Hence, we believe further studies need to be performed to validate these results and evaluate the benefit of combined cytokine and stem cell therapies for TBI.

P113

Effect of Curcumin on Microglial Cells in MS

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Multiple sclerosis (MS) is the most common autoimmune disease, especially among young's. Neuroinflammation results from inflammation in CNS and it may cause different disorders and diseases .It is also known as a detriment in multiple sclerosis. In fact, it causes problems and symptoms in MS. In MS the self-immune cells attack the myelin of neurons, it maybe the nerve in brain or spinal cord. Demyelination causes inflammation in the area and activation of the microglial cells. Microglial cells (macrophages in nervous system) protect nervous system and they are also important in neuroinflammation especially in autoimmune condition. Microglial cells can cause inflammation in MS in these ways: Presenting of neural autoantigens to autoreactive T cells, Secreting of proinflammatory cytokines.) TNF-alpha, IL-1 beta, IL-6), increasing permeability of blood vessels, releasing nitric oxide (NO). Anti-inflammatory drugs can decrease symptoms of MS. As we know Curcumin or diferuloylmethane is a yellow pigment and principal curcuminoid of turmeric. Anti-inflammatory properties of curcumin are obtained by the control of secretion nitric acid through decreasing the level of MRNA and protein producing nitric oxide and restraining LPS that result in releasing cytokines. Regulating inflammatory cytokines such as IL-1beta, IL-6, IL-12, TNF-alpha and IFN-gamma and associated JAK-STAT, AP-1, and NF-kappaB signaling pathways in immune cells are decreased by curcumin. The purpose of this study is to suggest that turmeric can be as a natural herbal drug for inflammatory diseases like MS. According to the evidences , we can say turmeric,

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which contains curcumin can use as a prevention in MS or any diseases with inflammation like autoimmune diseases and Curcumin as an herbal matter has anti-inflammatory effects especially on microglial cells so it can be considered as a benefit dietary factor in patients with MS.

P114

The Impact of Chamomile on Parkinson Disease (PD)

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After Alzheimer disease, Parkinson disease (PD) is considered the most common neurodegenerative disease. Its prevalence is the same in all area but it is common in males than females. From every 100 persons who are above 60 years old, one of them has PD. Also 5-10% of patients are less than 40 years old. This progressive neuroinflammation disease isn't fatal. In 60% of cases is with depression. The reason of PD in some cases is use of medications such as phenothiazine, tumor, infection, inflammation, genetic problems, and lack of folicacid and generally, loss of dopamine – secreting cells. At first, PD`s sings are mild and usually occur on one side of the body. Parkinson is accompanied by painful symptoms for example bradykinesia, rigidity and insomnia. This symptoms are decreased by gene therapy, antioxidative drugs and prescription dopamine. Free radicals cause oxidative damage to nucleic acids, proteins, lipids, unsaturated fatty acid peroxidation in cell membrane, increasing vascular permeability, mitochondrial dysfunction and endlessly inflammation and edema. . The most effective agent of reduce the effects of oxidative stress is increasing level of dopamine in the nerves cells to normal range. Chamomile, an annual plant, is one of the most popular and widely used in traditional medicine for treatment of numerous gastrointestinal disorders, abdominal bloating, relaxing muscle, anxiety effects (relaxation and calming) and also, anticancer, antimicrobial, anti-inflammatory and antioxidant effects. Several evidences suggested that Chamomile contain GABA, noradrenalin, dopamine, serotonin neurotransmission. Chamomile because of having striatum dopamine that can crossing the blood – brain barrier, can increase dopamine of the nerve cells. Studies show that chamomile maybe improved Parkinson`s sings. It may be possible that every formulation of chamomile (e.g., oil, vapor, tea) or every section of this plant (e.g. flower, leaf) may caused different anxiety results.

P115

Potential Therapeutic Targets Related to Neuroinflammation in Treatment and Prevention of Autism

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Autism spectrum disorder (ASD) is a mental condition, present from early childhood, characterized by great difficulty in communicating and forming relationships with others and using language. In the last four decades many studies have shown that immune responses in different regions of brain play an important role in ASD pathogenicity. A conservative estimate based on the research suggests that a great percentage of patients with ASD have microglial activation or neuroinflammation. Microglial activation or dysfunction affects neural development and results in neurodevelopmental disorders like ASD. Powerful immune-modulators like poly unsaturated fatty acids (PUFAs) and specially n-3 PUFAs exerting anti-inflammatory properties are important during brain development. Omega-3 fatty acids have an important role in neurogenesis, neurotransmission and protection from oxidative stress. Some of these effects are mediated by inhibiting the formation of prostaglandin E2 from arachidonic acid (an omega-6 PUFA). So PUFA deficiency during pregnancy can cause neurodevelopmental disorders including ASD in children. Considering the fact that most of patients with ASD have neuroinflammation and possibly immune responses in the brain is an important part of ASD emergence, curing the inflammation by inhibiting microglial cells and reducing pro-inflammatory cytokines, can be effective to improve symptoms. A recently published article showed that Fingolimod (FTY720) administration to autism rat models can cause improvement in behavior, learning and memory by inhibiting activation of microglial cells and lowering the level of pro-inflammatory cytokines like interleukin-1β (IL-1β) and IL-6 in the brain and reduces neuronal loss and apoptosis of pyramidal cells in hippocampus. So more studies needed to prove the effect of this drug on the treatment of ASD and finding new ways to inhibit microglial activation to stop neuroinflammation. PUFAs play a vital role in neurodevelopment by immunomodulation so it is suggested that an improved diet including enough amounts of PUFAs during pregnancy can be profoundly effective to prevent ASD in children.

P116

The Effect of Galectin-3 and Lanthionine Ketimine Ester in Neural Recovery after Spinal Cord Injury

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Spinal cord injury (SCI) is a trauma that disturbs motor, sensitive and autonomic function and directly impacts the quality of life. After physical damage, releasing of pro-inflammatory proteins and cytokines occurs and with collaboration of immune system cells, an immune response begins in the brain tissue. The result of neuroinflammation is edema, apoptosis and release of axonal growth inhibitory factors and accordingly nerve function loss. Neural damage spreads further so the paralysis can extend to higher segments. Experimental studies on animals have shown that galectin-3 (gal3), a protein that belongs to carbohydrate ligand lectin family expressed by different cells, contributes to neuroinflammation after SCI by activating lymphocytes, macrophages and microglial cells and shifting the microglial phenotype toward M1. Neuroinflammation activated by gal3 also causes neuropathic pain which poorly responses to common analgesics. It has been shown that inhibition of gal3 by intrathecal administration of modified citruspectin (MCP) reduces inflammatory response and leads to better motor recovery. On the other side lanthionine ketimine (LK), a natural brain sulfur amino acid metabolite and its synthetic brain penetrating ethyl ester (LKE) have anti-inflammatory and neurotrophic activities that promote growth factor-dependent neurite extension and suppress microglial activation by shifting the microglial phenotype toward a more neurotrophic M2 character resulting in promoting locomotor recovery after SCI. Taken together, considering the activators effect of gal3 and inhibitory effect of LKE on neuroinflammation and the key role of neuroinflammation in secondary damage after SCI and neuropathic pain, it seems that the lack of gal3 and using LKE as a drug can lead to reduction of neuropathic pain and better recovery of neurons after SCI and providing a chance of better quality of life for the patients and a step forward to make them walk.

P117

Endocannabinoid System as a Novel Therapeutic Target in Epilepsy

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Endocannabinoid (ECB) system plays a vital role in responses to stress. Moreover, ECB and its receptors cause anti-inflammatory, anti-oxidative and neuroprotective effects by modulating neuronal, glial and endothelial cell functions. A number of studies have demonstrated ECB system notably defects in neurotraumatic and neurodegenerative diseases like epilepsy, TBI, Alzheimer's disease and Parkinson's disease. ECB system comprise of various compartments, including 2 G-protein-coupled receptors (GCPCRs), named CB1 and CB2 receptors, which create two pathways. Most cannabinoid-based drugs used in treating neurodegenerative disorders affect CB1 pathway. Activating CB2 moderate inflammatory response and stimulate the secretion of anti-inflammatory mediators in microglial cells and astrocytes. CB2 function increased in inflammatory condition in neuronal and endothelial cells, which limit neuroinflammation and blood-brain barrier disruption. In conclusion, involvements of ECB system in neurological disorders make it a suitable therapeutic target. In this review, we discussed the role of CB1 and CB2 receptors in neurodegenerative diseases. CB2 pathway is believed involved in inflammation response, but its relationship with epilepsy remains unclear. Therefore, we suggest surveying the expression of CB2 receptor in an animal model of epilepsy.

P118

Osteopontin: An Early Player in Neuroinflammation Disease

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Osteopontin also known as bone sialoprotein (BSP). Osteopontin role as a linking protein. it is an extracellular structural protein and therefore an organic component of bone. Osteoprontin (OPN) binds to several integrin receptors containing $\alpha^{۴}\beta^{۱}$, $\alpha^{۹}\beta^{۱}$, and $\alpha^{۹}\beta$ ۴expressed by leukocytes. These receptors have been well-stable to function in cell adhesion, migration, and survival in these cells. It has been shown that OPN drives IL-17 production.as we know it has important effect on neuroinflammation Biomarkers qualified of predicting the clinical method and the level of disease progression in multiple sclerosis are currently unavailable. Our objective is to examine if the levels of proteins associated with axonal and neuronal degeneration T-cell-mediated autoimmunity (Osteopontin) are altered in the cerebrospinal fluid (CSF) of MS patients, and to assess their potential in reflecting the clinical severity and predicting the progression and clinical evolution of early MS.

P119

Role of Gut Bacteria on Alzheimer's Disease

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Alzheimer's disease (AD) is a neurodegenerative disease that is the most common type of dementia.AD includes 60_80% of dementia and most people with AD have more than 65 years old.AD causes losing neuronal activity by abnormal proteins. Plaques of beta-amyloid and tangles of "tau" protein can lead to AD. Recently evidence has found that AD may come from outside of central nervous system (CNS) and originate in gut by git bacteria. These bacteria can release a large quantity of beta-amyloids that play roles in AD. Also in a study on gut bacteria in mice, researchers found different composition of bacteria between healthy mice and diseased mice. These studies show direct links between gut bacteria and Alzheimer, can help us have better strategies to prevent and cure AD. There are mutual communication between brain and gut. Regulation of the gut flora with diet and nutrition shows microbiota have key role in maintaining brain health. In some studies it has been found that gut bacteria may produce enzymes that these enzymes make toxic metabolites dor neurons like D-lacrtic and ammonia. Also has been found some molecules in both enteric nervous system (ENS) and central nervous system (CNS) that can cause neurodegenerative like beta-amyloid and tau. Study on free-germ mice revealed *significantly less beta-amyloid in them* .furthermore researchers transported gut bacteria from diseased mice to free-germs mice and saw that mice developed more beta-amyloid plaques in the brain compared to if they had received bacteria from healthy mice. These researches show direct link between gut bacteria and Alzheimer.

P120

The Effect of Steroids on the Optic Neuritis in MS

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Optic neuritis an inflammatory disorder of the optic nerve inflammation is usually in young's with symptoms of eye pain and vision loss that occurs is a common symptom of MS. Optic neuritis is one of the first symptoms of MS that observable into disorders in white matter of cerebral cortex. The risk of MS in patients with optic neuritis during 2 years 20% in 15 years, 45-80% is calculated. In 1968, in Michigan a study showed that 65% of ophthalmologist and 90% of neurologists used corticosteroids for the treatment of Optic neuritis. Optic neuritis causes disorder in Brain-Blood Barrie (BBB), which causes inflammation of blood vessels. In treatment of optic neuritis in patient with MS used Methylprednisolone into intravenous and oral that caused accelerated recovery of vision but not caused visual improvement. In the treatment of optic neuritis from Methyl prednisolone ,high doses of intravenous (IVMP) used that mechanism of action is unknown and reduce the potential for visual arousal and up to limit caused the return vision site to normal condition. Intravenous use of methyl prednisolone in a short period of time and in high doses can be reduced inflammation vision. Using Methylprednisolone continuously reduce disorder of brain-blood Barrie (BBB) and brief improvement of neurological side effects. Despite the effect of steroid drugs on nervous attack and neuroinflammation in patient with MS, more studies should be carried out on the long time effect these drugs and their combination with other drugs.

P121

Neuroinflammation and Diabetes

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Obesity is a chronic disease that shows the most serious global health problems .It relates to the body fat .Obesity is caused by an imbalance between energy intake and expenditure, this balance is regulated by genetic and environmental interactions. Obesity, which is a risk factor for chronic diseases, has become epidemic in the developed countries. Resistance to Leptin hormone is one of the main reasons for obesity; this hormone is released by fat cells, substantially more fat causes more releasing of Leptin hormone. In fact discovery of Leptin hormone has opened a new field of research in obesity studies. Leptin hormone controls metabolism, reconciliation and energy consumption. Obesity plays a key role in the development of diabetes. Effect of obesity on the development of diabetes through precipitation of Amylin. Amylin is a peptide hormone that is co-secreted with insulin from the pancreatic β-cell and in patients with diabetes, there is deficiency of that. As we know, in diabetes, insulin resistance and pancreatic beta cell hypertrophy occurs in the early stages, in this condition Amylin is increased .Accumulation of Amylin is in the hypothalamus of the brain and it can lead to neurological disorders through inflammatory reactions. Increased insulin sensitivity in the body is related to the proper functioning of the hypothalamus. Also hypothalamus controls Leptin, when the level of Leptin hormone is increased it is bonded to its receptors in hypothalamus. The purpose of this paper is investigation the relation between Amylin and Leptin in the hypothalamus in the process of neurological inflammation and diabetes. Leptin hormone resistance is a characteristic of obesity; studies have been shown the inflammation of the hypothalamus that is results from fat is an important

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mechanism that will cause to increase Leptin resistance and precipitation Amylin.

P122

Dietary Habits and Multiple Sclerosis

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Multiple sclerosis (MS) is an inflammatory and autoimmune disease.MS is characterized by blood brain barrier breakdown, inflammation, axonal injury, degeneration of the myelin sheath and etc. A significant association between inflammation and degeneration of neurons is observed in the brain. Also active MS lesions always associated with inflammation thus inflammation is a target for treatment of MS. Etiology of MS is unknown. Genetic, infections, immunological and environmental factors have all been blamed. It is commonly accepted that dietary habit is one of the environmental factors that are involved in the pathogenesis of MS. Now MS therapy is not associated with a particular diet probably due to lack of information on the effects of nutrient on the disease. Wrong dietary habits include viral infection, heavy metal poisoning, smoking, low vitamin D and influence of dietary habits are more than infections and smoking. Researches demonstrated that patients with MS ate bread, cereal products, fish, some vegetables and fruits less than healthy people. Also, there is a correlation between deficiency of Omega-3, vitamin D, antioxidant vitamins and folic acid in the diet and exacerbation of symptoms of MS. Therefore it is purposed to study more about the relationship between MS and dietary habits for treatment aims and remission of symptoms. Studies revealed that healthy dietary molecules have an important role in MS. Nutritional intervention with anti-inflammatory foods and dietary supplements can reduce side effects of drugs and the symptoms of chronic fatigue syndrome. So dietary habits can be effective on MS but it is suggested more information is needed to find the exact association between diet and the risk of MS.

P123

A Promising Approach in Inflammation Management, Cytokine Therapy for Alzheimer's Disease

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Alzheimer's disease (AD) is a neurodegenerative disease characterized by cognitive and other CNS impairments which is increasing worldwide and poses a major public health problem, yet no effective treatments are found and available drugs only alleviate the symptoms temporarily. Postmortem analyses on the brains of AD patients and observed lower risk of AD in NSAIDs users among other studies have presented inflammatory evidence for this problem. Further research proved an imbalance between pro (such as IL-6, IL12) and anti-inflammatory cytokines and also a prolonged activation of microglial cells. In such state, an increase in IL-1-ß, TNF-α, IL-12 and IL-6 and a decrease in TGF-ß levels plus the accumulation of amyloid beta (Aß) in the brain can be seen. Altering the cytokine levels can be a promising approach for AD treatment although it should be considered that each cytokine is a member of a body network, so we suggest target therapy by using nano carbon made capsules able to pass the blood-brain barrier coated with surface markers specified for microglial cells containing IL-4 and TGF-ß because IL-4 and TGF-ß both can promote phagocytosis of amyloid deposits , or IFN-y as a substitute for IL-4, which has the same but weaker effect than IL-4.Also using anti-IL-6 receptor(tocilizumab) accompanied with 1,2 dihydroxy vitamin D_3 as IL-12 production inhibitor can be useful. As well it should be noted that prolonged usage of IL-4 and TGF-ß can cause more Aß deposition, so we suggest a periodic usage cycle including a consumption period followed by a withdrawal period to prevent prolonged complications.

P124

Decrease Signs Parkinson`s Disease with DOPAMINE in Apple

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After Alzheimer's disease, Parkinson's disease is the most common nerve-damaging disease. Parkinson's is a progressive and chronic disease where cells secrete dopamine-cut black flesh and in the absence of dopamine in the brain destroyed the irregular body movements. Man eats the food that causes the formation of the neurotransmitters. Tthree neurotransmitters: dopamine, serotonin, norepinephrine were studied in relation to foods. These studies show that certain brain neurotransmitters features in forming foods make us feel happy. If we have a body in adequate amounts of dopamine, brain cells that produce dopamine from L-phenylalanine uses alanine as raw material. L-phenylalanine, an essential amino acid, which is found in blood plasma and brain, can be converted to tyrosine in the body of the material used in the synthesis of dopamine. A compound found in apples called quercetin, an antioxidant that is known to prevent cancer is also more important role in the prevention of neurodegenerative disorders related to

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it. Daily consumption of apples can be avoided to the extent of developing Parkinson's disease. Patients with Parkinson's disease with the consumption of these foods can be improved to some extent.

P125

Treatment of Multiple Sclerosis with Mesenchymal Cells Starfish

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Arm grooves of star-fish have mesenchymal stem cells derived from adipose tissue cell. Widespread studies have shown that stem cells derived from adipose-derived mesenchymal stem cell from bone marrow have many similarities in terms of the ability to differentiate into a variety of tissues. Most of the animals live in warm and shallow waters and the waters of this feature is that it is low in dissolved oxygen. By cutting arm of this animal's mesenchymal cells and neural network drived into again. But the specific condition of neuronal myelin sheaths around the axon man is broken at best so that the nature of this beast is higher than man. Thus, differentiation of mesenchymal cells starfish nerve cells in ways that lead to nerve cells and then transplanted human features.

P126

The Relationship between Bell`s Palsy and Diabetes

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Bell`s palsy is explained as a sudden paralysis/paresis of all muscle groups on one side of the face due to inflammation of facial nerve, The frequency of Bell palsy in diabetic patients is a matter of controversy. There are some reports that refer to Bell`s palsy as occurring more commonly in patients with diabetes, or even prediabetes. AmosD, Korczyn in his studies had shown that a high frequency of diabetes mellitus is reported in patients presenting with Bell's palsy; The frequency was 45% at age ten to nineteen, and increased with age, the frequency for the whole patient being 66%.it also was reported The rates of diabetes and hypertension in association with Bell's palsy were significantly high compared with those of the general population. But according to most authors, the percentage of diabetics among patients with Bell palsy is not higher than in the general population, Kudoh et al reported that noninsulin-dependent diabetes mellitus (NIDDM) or hypertension doesn't seem to have any specific influence on palsy scores and electroneuronography results, although patients with both NIDDM and hypertension are slow to recover. According to the information that is available about the relationship between Bell`s palsy and Diabetes, we can't report the exact results about this relationship and the frequency of Bell's palsy in diabetic patients is a matter of controversy.

P127

The Role of CGRP in Dental Pulp Nerve repair

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Tooth pulpal nerves include sympathetic and sensory nerves that originate from trigeminal ganglion .From the terminal of these sensory nerves neuropeptides are secreted that are neuromodulators and neurotransmitters. One of them is CGRP (Calcitonin gene-related protein) which has a vasodilatory effect on pulpal vessels. Near abscess and moderate injuries, this neuropeptide can lead to neural fibers sprouting using its receptors (The Calcitonin receptor _ like receptor and receptor activity-modifying protection), thus, its modulating effect can lead to pulpal healing. For treating this inflammation, several common anti-inflammatory drugs such as steroid drugs (also named corticosteroids or Cortone) can be used. One of the side effects of these drugs is osteoporosis. But non-steroidal anti-inflammatory drugs do not have this adverse effect and can inhibit the effect of both kinds of cyclooxygenase enzyme leading to the inhibition of prostaglandins synthesis and thus inhibiting the inflammation .But these drugs can cause digestive problems including peptic ulcerative .This is because of the inhibition of type 1 cyclooxygenase. There is a group of these drugs named "Celecoxibs" which only inhibits the effect of the second type of cyclooxygenase and does not have adverse effects on stomach and coagulation characteristic of blood. Considering all these, if we synthesis a substance with anti-inflammatory characteristics of substances such as non-steroidal anti-inflammatory drugs and antipyretic and anti-pain characteristics similar to GCRP, we can expect that it induces these effects on pulpal nerves and leads to tissue healing and Axonal sprouting in an inflamed and exposed pulp by attaching to this neuropeptides receptors and inducing it and in this way to some great extents, we can overcome to tooth nerves inflammation and the process of pain generation followed by caries or different pulpal or dentin related injuries like endodontics treatment.

P128

Sleep Disturbance and Epilepsy: An Inflammatory Pathway

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Sleep plays a vital role in regulating physiological mechanisms in the human body. Nowadays, by the change of lifestyle and as a consequence of longer work hours and increased accessibility to media, sleep disturbance becomes a common problem in modern society. Many studies demonstrated that sleep disturbance triggers a systemic low-grade inflammation by increasing the level of several cytokines, chemokines and acute – phase proteins. Increased pro – inflammatory cytokine gene expression is reported, when a night of sleep restricted to 4 hours. Sleep disturbance increases the levels of IL-6, high-sensitivity C-reactive protein (hsCRP) and IL-1b in plasma. Also, IL-1 and tumor necrosis factor (TNF) gene expression in brain (hypothalamus, hippocampus, and pre-frontal cortex) increase in response to sleep disturbance in mice. Moreover, studies showed that blood-brain barrier (BBB) disrupts by chronic REM sleep restriction in rats. These data indicates that pro-inflammatory mediators can enter the brain if sleep restriction increases the unselective transportation across the BBB. On the other hand, findings suggest that inflammatory processes can play an important role in epileptogenesis in several ways like pro-inflammatory pathways (such as IL-1β). A study on epileptic patients in 2014 showed that daily generalized motor seizures result in elevated IL-6 levels leading to increased hs-CRP. Also, in 2015, Uludag et al, found increased levels of IL-1β, IL-6, and IL-1Ra among epileptic patients and high levels of IL-1b in patients with temporal lobe epilepsy. Although findings support the idea that sleep disturbance provokes epilepsy in susceptible through inflammatory pathways, further studies is needed to make this relationship. Public education on proper using of media, using herbal hypnotics with lesser side effects and paying attention to sleep hygiene in General Policies, are suggestions that help us to have a better society with healthy brains and lower epilepsy incidence.

P129

The Role of Overexpression Transcription Factor BRN 4 in Multiple Sclerosis

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Adult neurogenesis is a process of producing nerve cells from their progenitor that occurs in some areas in the brain such as the hypothalamus. Low activity in this area plays a role in neural degeneration and diseases such as multiple sclerosis, epilepsy and depression. MS is a neurodegenerative disease with a permanent disability that the main reason for it is axonal degeneration and neuronal death. Therefore, increased neurogenesis in the hypothalamus is an appropriate treatment for this disease. Transcription factor BRN4 is a transcription factor of the POU3F4 gene (which encodes a member of the POU-III class of neural transcription factors) that regulating the differentiation of striatal multipotent and precursors stem cells in the hippocampus of the adult brain. This factor also increased during the developing neural tube and the peak of neurogenesis at the time of embryo. IGF-1 is a factor that upregulate BRN4 by activating P13/Akt signaling pathway also BRN4 can regulate Ctbp2 and Notch2 genes that are related to neuronal differentiation. In one study in 2017, observed that after Lentivirus-mediated brn4 injected to dentate gyrus of rat hippocampus, differentiation and maturation of neural stem cells significantly increased. These results suggest that overexpression of brn4 enhance neurogenesis and neural differentiation in the hippocampus. We hypothesized that Overexpression of transcription factor BRN4 by injection of lentivirus-mediated brn4 or increased level of IGF-1 can be used as a great treatment modality in patients suffering from Multiple sclerosis, without injection neural stem cell. We suspect that with increase expression of BRN4 in the hippocampus of patients with the score 4- 5 because of disability affects daily routine (According to scoring EDSS) we can see significant improvement in their daily activities and treatment process.

P130

The Role of Host T- Cell Lymphocyte in Immunopathogenesis of HTLV-I-Associated Myelopathy/Tropical Spastic Paraparesis

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Human T-cell lymphotropic virus type 1 (HTLV-1) is associated with adult T-cell leukemia/lymphoma (ATL) and HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP). Only a limited percentage of infected individuals develop disease in response to the virus while the majority remain asymptomatic and HAM/TSP is the most common clinical manifestation of the virus. HAM/TSP is an inflammatory disease of the central nervous system (CNS). The mechanism by which HTLV-1 induces HAM/TSP is not clear yet. Several factors have been hypothesized to contribute to an HTLV-I-infected individual's progress to HAM/TSP. One of the most important factors is the host immune response against HTLV-I and T cell lymphocytes plays a key role in the immune response against HTLV1 virus. HTLV1 attacks different types of cells in the body but

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CD4(+) T lymphocytes are the main target of HTLV-1 that have an important role in the immunological response to this retrovirus. HTLV-I-infected CD4+ T lymphocytes migrate to the CNS tissues and CD8+ HTLV-I specific cytotoxic T lymphocyte (CTL) attack HTLV-I-infected lymphocytes. Recent data indicate that HTLV-I and its expression are localized in infiltrated lymphocytes within the spinal cord lesions of HAM/TSP patients rather than in resident central nervous system (CNS) parenchymal cells. Hyperactive CD8(+) cytotoxic T lymphocytes (CTL) that generate in response to HTLV-I-infected lymphocytes likely play a key role in the genesis of pathologic abnormalities associated with HAM/TSP and also a high HTLV-I proviral load in peripheral blood lymphocytes (PBL) increase this pathological response and cause spinal cord lesions in HAM/TSP patients. Although the exact mechanism underlying the high HTLV-I proviral load in PBL in HAM/TSP patients is still unknown, we must consider therapeutic approaches in HAM/TSP that eliminate HTLV-I-infected CD4+ T lymphocytes and also the regulation of efficiency and activity of hyperactive CD8 (+) cytotoxic T lymphocyte (CTL).

P131

Connection Process Inflammation and Improvement Alzheimer's Disease

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Platelet aggregation beta amyloid main causes inflammation of neurons in Alzheimer's disease. In fact, creating this inflammation due to inappropriate actions in blood brain barrier (BBB) and astrocyte and microglia during the last century that studies conducted in this case nothing has been found. The only thing that can be done to prevent and reduce pro-inflammatory factors such as cytokines and beta-amyloid. Non-steroidal drug have little effect on reducing inflammation and may even aggravate the inflammation. The combination of polyphenols can inhibit the accumulation of beta-amyloid and oxidative stress. This combines decrease the production and accumulation caused by cytokines. One of the significant factors in the recovery process of Alzheimer's disease is TNFSF-10 that have anti-tumoral feature and reduce the accumulation of beta-amyloid clumps. According to research conducted by intraperitoneal injection of TNFSF-10factor has been effective in improving Alzheimer's disease. Reducing the activity of microglia and astrocytes cause of reduced accumulation of beta-amyloid also reduces inflammatory biomarkers from astrocytes and microglia.

P132

The Role of Depression in Prevention or Therapy of Neuroinflammatory Diseases

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Depression alone is a severe and acute disease, but its close relationship with neuroinflammation and neurodegenerative diseases has emphasized its importance. In fact, the linking bridges between depression and these diseases are cytokines, especially pro-inflammatory cytokines which have fundamental roles in organizing different parts of central nervous system and emotions. Studying the relationships of depression and neuroinflammatory diseases shows two different aspects: either depression and cytokine disturbances lead to neuroinflammation and so brain diseases or depression appears as a result or contributor of neuroinflammatory diseases due to factors like stresses during the treatment, feeling of weakness and dependence on others or linked physiologic mechanisms of depression and neuroinflammation. These facts represent depression as an important factor in diagnosis, prevention or therapy of neuroinflammatory diseases. We review the role of Depression in neuroinflammatory diseases as a therapeutic strategy. According to mechanisms of inflammation-associated depression, which cytokines play a major role in progression of these disorders, new possibilities are opened for developing anti-inflammatory drugs or new anti-depressant compounds targeting neuroinflammation or its pathways. Also an effective strategy for detecting depression in the early stage can help us prevent neuroinflammation and subsequently neuroinflammatory disease.

P133

Neuroinflammation in Alzheimer 's Disease

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Alzheimer's disease (AD) is a neurodegenerative disorder and the most common form of dementia. Almost 47 million people suffer from dementia worldwide. AD accounts for approximately 60%–80% of all dementia cases. Three major pathologies characterize the disease: senile plaques, neurofibrillary tangles and inflammation. We review the literature on events contributing to the inflammation. Those inflammatory processes play a significant role in the pathophysiology of AD. Histopathologically is characterized by the presence of two major hallmarks, the intracellular neurofibrillary tangles (NFTs) and extracellular neuritic plaques (NPs) surrounded by activated astrocytes and microglia. The main component in the NP is the amyloid-β peptide (Aβ). Neuroinflammation is characterized by the activation of astrocytes and

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microglia and the release of proinflammatory cytokines and chemokines. Neuroinflammation is one of the main factors neurodegenerations. Study of the factors and pathways able to the first step of the inflammatory response induced to identify potential therapeutic targets through which to stop the progress AD. Evidence confirms that neuroinflammation, by neuronal, glial, and immune components, is a contributing cause of Aβ aggregation, tau hyper phosphorylation, and neuronal damage and death, so production of cytokines and pro-inflammatory molecules has initially a neuroprotective role, but subsequently becomes the cause of further neurodegeneration. Therefore, future studies must intensively investigate the intricate ways of the neuroinflammatory process and define the best time to control it, so it will be possible to achieve more focused therapeutic strategies in the hope of not only alleviating but also modifying AD progression.

P134

Use of Zinc in Drugs to Improve Neuroinflammation Disease

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Zinc is a substance that regulates neural excitability by binding whit sodium channel and potassium channel. The efficiency of free zinc ion, make down the neural survival rate, reduced the peak amplitude of Na+ and make depolarization Na channel, increased the peak amplitude of transition outward k+ currents and delayed rectifier. Also it is an effective blocker of one subtype of tetrodoxine (TT-X) insensitive sodium channel than other sodium channel. Absolutely zinc effects on other proteins membrane and the suitable level of free zinc ion help to membranes function. In neuroinflammation zone, the membrane get into new environment and the protein channel hardly make impulse. In some neuroinflammation disease that neuron's problem is inability to make impulse, the efficiency of free zinc ion makes that and increase level of zinc ion, help neuron makes impulse. For example in Alzheimer, if the levels of zinc get increased, neuron make impulse and the process of disease get slowly. But neuroinflammation disease that neuron make excessive impulse and this is main problem, like Multiple Sclerosis, efficiency of free zinc ion help to decrease membrane activities. In neuroinflammation disease, when the protein membrane get into damage, use of zinc in drug in other to improve the efficiency of free zinc ion, can help to protein channel and membrane function. In the other site, increase the level of free zinc ion make excessive impulse. So free zinc ion in matrix is really important for action protein membrane and in neuroinflammation environment, changing the level make different result. If in a disease neuron action get down, zinc in drug can help to make impulse by binding whit protein channel.

P135

The Effects of Exercise on Treatment of Neuroinflammatory Disorders

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There are many reasons for decrease of the functional activity of the brain like aging of the neurons, bacterial diseases and neuro inflammations. Nowadays, the effects of exercise on physical and mental health have been proven but its effects on motor functions as well as neuro inflammation is an issue that has recently been studding. Neuro inflammation is a mechanism that can be caused by Bacterial diseases or aging the neurons of the central nervous system. The cause of the inflammation can be due to increasing of inflammatory cytokines such as interleukin (IL) 1β or decreasing of anti-inflammatory cytokines. Neuroinflammation leads to gradual destruction of neurons in CNS that can originates Alzheimer disease (AD), Parkinsonism & Multiple sclerosis (MS).In some studies such as O'calaghan et al, Radak et al & Schweiter et al, it is shown that regular aerobic exercises can facilitates learning in AD mice.Similar to these studies, Larson et al performed another study that showed regular aerobic exercises can delay the onset of AD in human by reducing the inflammatory cytokines. In addition to the beneficial effects of exercises on AD, it has demonstrated its good effects on Parkinsonism and MS. The exact mechanism of exercise in reducing the inflammatory cytokines is unknown but it's assumed that exercise can reduce inflammation by decreasing fat. Although most of the studies showed aerobic exercises are more beneficial but endurance exercises can increases anti-inflammatory cytokines too. In addition to the kind of the exercises, the period of exercise is important too. Short term aerobic an exercise (just for 3 weeks) increases oxidative stresses and the chance of neuro-inflammation. Long term aerobic exercise can prevent and treat neuro inflammations diseases like AD, MS and Parkinsonism by reducing inflammatory cytokines and increasing the anti-inflammatories. However, the exact mechanism is unknown Future studies should consider the intensity and the best kind of exercise to have the most beneficial effects.

P136

The Role of Blood Brain Barrier in Multiple Sclerosis

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Multiple sclerosis (MS) is an inflammatory disorder, in which neurons become demyelinated. To date, its etiology has remained unknown. Nevertheless, certain features are inspected to provoke MS. For instance, improper function of immune cells is widely believed to be the basis of such disorder. In this concept, MS is stated as an autoimmune disease, which was asserted by major of studies, as CD8+ T-lymphocyte reacts to any agent containing major histocompatibility complex class 1 (MHC 1). They pass epithelium of brain capillaries to access locations where oligodendrocytes abnormally contain production of such gene. In this hypothesis, most articles assign peripheral immune system for disrupting the structure of blood brain barrier (BBB), which involves series of sequential mechanisms regulated by numerous genes of endothelium of BBB, as well as immune system. During last few decades other etiologies have been proposed, which are in intimate relationship with BBB. In 2015, a new pathway was discovered in which leukocytes might recruit and travel more easily in and out of the brain tissue. These newly discovered lymphatic vessels also might be in association with MS. The aim of this paper is to present the role of BBB in pathogenesis of MS. In conclusion, autoimmunity of MS is well asserted by most studies, which aimed to propose further etiological facts of MS. However, importance of each molecules and genes leading to its autoimmunity or disruption of BBB is yet to be more particularly determined.

P137

Central Nervous System Involvement in Systemic Lupus Erythematosus

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Systemic Lupus Erythematosus (SLE) is a complex clinical syndrome which its components are less clearly recognized and includes heterogeneous demonstrations engaging both central and peripheral nervous system along with disabling effects. This disease is called "thousand faces" due to these heterogeneous demonstrations. This gap exists while 75% of adults and children with SLE may deal with its nervous demonstrations and experience disability during disease period. Different factors contribute to the body's immunity performance disorder including genetic, hormonal and environmental factors. However, disposing factors leading to nervous demonstrations in some patients are not clearly understood. Today, Lupus nervous involvement is considered as "the most clinical challenging 'visceral' involvement," "causes high morbidity and mortality" and put a "heavy financial and economic and social burden on the society". Lupus nervous involvement covers a wide range of clinical demonstration intensity. NPSLE was first described by Morris Kaposi in 1872. In 1999, ACR attempted to name and define neuro-psychotic syndromes recognized in SLE. Different CNS demonstrations of Lupus are investigated in this paper. Neurologic demonstrations of focal SLE, mostly the secondary ones, as vascular events is due to anti-phospholipid antibody. These demonstrations are usually acute and resist against treatment at first and can be accompanied by structural abnormalities in autopsy while pathogenesis mechanism of CNS demonstrations is less recognized and these demonstrations are harmful and develop slowly; they are reversible after treatment and usually not accompanied by structural pathology. Although headache and mood disorders are common neurologic complains of patients with SLE, seizure, brain vessel disease, acute confusional state, and neuropathy are the most common syndromes related to SLE. CNS demonstrations of SLE patients include: Cerebrovascular disease, Seizure, Myelopathy, Lupus psychosis, acute confusional state, Cognitive dysfunction, Movement disorder, Aseptic meningitis and demyelinating syndrome.

P138

Improving Neuroplasticity Through Neuroinflammation Pathways as a Therapeutic Goal in the Treatment of Autism

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Neuroplasticity is the brain's ability to reorganize itself by forming new neural connections throughout life. Neuroplasticity allows the neurons in the brain to compensate injury and disease and to adjust their activities in response to new situations or to changes in their environment. At the other side, it is now well established that neuronal function is strongly influenced by both central and peripheral inflammation and it is able to modulate the efficacy of synaptic transmission and the induction of the main forms of synaptic plasticity. Astrocytes, Glial and microglial cells are recognized as active elements of synapses, playing a central role in neuro-inflammatory processes. This feature can be used as therapeutic goal in many brain diseases such as Multiple sclerosis, Alzheimer, Epilepsy, Parkinson's disease, Autism spectrum disorder, etc. Autism, or autism spectrum disorder (ASD), refers to a range of conditions characterized by challenges with social skills, repetitive behaviors, speech and nonverbal communication, as

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well as by unique strengths and differences. Newly, some studies suggested that an abnormal function of glia and astrocytes may be involved in the development of autism. Recent studies reported some markers that are effect ASD mechanism including glial fibrillary acidic protein (GFAP), aquaporin4, connexin43, methyl-CpG-binding protein 2 (MeCP2) and etc. The present review summarizes the latest understanding of novel ASD treatment that effect neuron plasticity, especially proteins and cytokines that are involved in neuro-inflammation pathways and the process that damages neuroplasticity. However, there is yet no direct evidence showing how neuro-inflammation pathways can improve neuroplasticity in the brain of autistic patient and improve the life of these patients. This review indicates more research is essential to study on these markers as a therapeutic goal in the treatment of autism.

P139

Curcumin anti-Inflammatory Effect in Neuroinflammatory Disorders: Prospective and Challenges

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Curcumin is a hydrophobic polyphenol and major bioactive component of turmeric with known anti-inflammatory, neurogenesis, antioxidant, and anti-carcinogenic effect. Curcumin antagonizes many steps in the inflammatory cascade, including Inhibition of NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells), activator protein-1 transcription and iNOS (induced Nitric oxide synthases). Inhibition of NFkB is believed to be the central pathway of curcumin's mechanism. Curcumin induced the antioxidative protein HO-1 which reduced the microglial pro-inflammatory cytokines such as TNF α, IL 1β and IL 6. All these mechanisms indicate the anti-inflammatory effect and neuroprotective action of this chemical against neuroinflammation also Neuroinflammation is implicated in the pathogenesis of many neurodegenerative diseases. Curcumin administration has been reported to attenuate neuroinflammation.in contrast to nonsteroidal anti-inflammatory drugs whose adverse side effects includes gastrointestinal ulceration and liver or kidney toxicity, curcumin seems to be relatively safe but Despite its multi anti-inflammatory properties, curcumin's clinical use is limited because its poor oral absorption, rapid systemic elimination, rapid metabolism and limited blood brain barrier permeability but the most challenging factor is curcumin's low aqueous solubility. Clinical studies on humans and rodents have reported low bioavailability of curcumin shown by the lower level of serum and tissue curcumin.in this study we aim to overview the prospective and challenges on curcumin use as an anti-inflammatory drug. Curcumin's anti-inflammatory effect is well-established but clinical administration of this chemical is limited due to its poor bioavailability, so approaches to enhance the bioavailability of curcumin is needed and maybe using the structural analogues of curcumin, adjuvants like piperine, phospholipids and biodegradable nanoparticle mediated delivery of curcumin are the best ways to increase curcumin's bioavailability.

P140

Stem Cells in Multiple Sclerosis

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Multiple sclerosis (MS) is an inflammatory disease of the central nervous system (CNS). Inflammation caused by immune cells destroy the myelin and then axon. CNS failure to complete repair results in permanent disabilities. Some types of stem cells have special potentials to repair these injuries and even cure MS. Neural crest stem cells with a mutual origin with CNS and the ability of differentiation to different types of neural cells can replace lost cells. They also increase survival and development of neurons by secreting neurotrophins. Mesenchymal stem cells have a high potential to identify affected areas and migrate to there. They reduce inflammation and autoimmune reactions by affecting on all types of immune cells. Mesenchymal stem cells change phenotypes of T cells from inflammatory form to anti-inflammatory form by decreasing of INF and increasing of IL4 production. It also increases regulatory T cells (Treg) and reduce killer T lymphocytes proliferation. And on the other hand reduce pro-inflammatory factors interaction with nerve cells by improving the blood-brain barrier performance. Also their impact on demyelination and restoration of nerve cells has been demonstrated. Olfactory ensheathing cells leads to regeneration of axons and myelin by production and secretion of growth factors and principal components of nerve cells membranes. It accelerates the healing by reorganization of glial scar, tissue support and stimulate vascularization. Hematopoietic stem cells can rebuild the immune system and completely suppress autoimmune reactions. Oligodendrocyte precursor cells regenerate myelination by differentiation to oligodendrocytes. Endothelial precursor cells suppress inflammation. Conclusion: Stem cells have significant potential to treat MS with

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various mechanisms. Knowing the features of these cells and their effect mechanisms are very important to find an effective treatment for MS.

P141

Mesenchymal Stem Cells as Treatment in Neuroinflammatory Disease

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Mesenchymal stem cells can be obtained from deferent tissues like adipose tissue, umbilical cord, placenta, skin, bone marrow, etc. These cells have regulatory effects on all types of immune cells such as dendritic cell, natural killers and lymphocytes. Mesenchymal stem cells induce inhibitory phenotypes of Antigen Presenting Cells (APCs) following their activity. They also change T cells phenotype from pre-inflammatory form to anti-inflammatory form by decreasing interferon gamma (INFY) production and increasing IL4 production. They increase T reg cells proliferation and decrease Natural Killer Cells proliferation and differentiation of alloantigen Induced Lymphocytes. MSC decrease destruction of axon and myelin and also improve regeneration of them. The role of mesenchymal stem cell in suppression of neuroinflammation is obvious. They have great immunomodulatory impact. Mesenchymal stem cells increase neurons survival by secreting neuroprotective factors and significantly decrease their apoptosis. These type of stem cell can play an important role in treatment of neuroinflamatory disease. Studies have shown that the use of mesenchymal cells are safe but more studies are needed to show they long term influences.

P142

Air Pollution's Triggering Role in Tau Protein Hyper Phosphorylation; A Sign of Alzheimer Disease

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Nowadays, air pollution is one of the major problems in developed and developing countries. In recent years, effects of air pollution on neuroinflammatory diseases such as Alzheimer disease and Parkinson disease have been studied. Researches on polluted cities citizens indicate increasing in central nervous system (CNS) inflammatory factors in comparison with clean cities; also air pollution exposing, increases risk of developing Alzheimer disease by the percentage of 138%. This review is going to discuss about triggering role of air pollution components, special Diesel exhaust particles, in increasing inflammatory factors and Tau protein hyper phosphorylation. Studies indicate that increasing of these factors, has a direct relation with Tau protein hyper phosphorylation. Tau protein is one of the main components for microtubules shaping and association. Hyper phosphorylation of Tau protein is a reason of microtubules dissociation; this causes shape of neurofibrillary tangles (NFTs).These tangles develop neural and synaptic dysfunction, which are early Alzheimer's hallmarks. Air pollution components break tight junctions, pass the blood-brain barrier and penetrate into the central nervous system, influx of these substances create amoebic form of microglia and overactive it, also increase secretion of inflammatory cytokines like interleukin-1β (IL-1β), tumor necrosis factor-α(TNF- α) etc. Increasing of these inflammatory factors is one of the agents that cause Tau protein hyper phosphorylation by pathways such as CDK/P35. Although the air pollution abolition needs a comprehensive contribution, special from governments, a diet which contains anti-pro inflammatory cytokines as a primary prevention could be prescribed to suppress widespread complications that may occur for residents in coming years; diets that contain flavanols, gallic acid etc. are suitable; These are found abundantly in dark chocolates, teas etc.

P143

The Effect of Platelet Activating Factor on Inflammatory Response in Multiple Sclerosis

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Multiple sclerosis is an autoimmune disease of the central nervous system which its main characteristic is an inflammation and demyelination and subsequent, neural degeneration. Many studies have shown that inflammation causing neuronal demyelination. MS is the most common cause of chronic neurological disability in during youth which the prognosis is that can be death. Platelet activating factor is a phospholipid mediator in central nerve system which acts as a messenger and plays role in platelet aggregation and inflammatory responses. Furthermore, these inflammatory mediators involve in many pathophysiological processes such as brain edema reperfusion injury through interactions with its receptor. PAFR (platelet activating factor receptor) is a seven transmembrane proteins that belongs to G protein receptor and express in many brain cells such

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as neurons and microglia. Expression of this receptor causes the release of many inflammatory cytokines like IL-6, IL-1β and TNF-α and also apoptosis marker including caspase-3 and bax/bcl-2. PAF and its receptor provides a strong inflammatory response and increased inflammation and recurrence of the disease. Thus, blocking the path connecting and interacting PAF and PAFR can significantly reduce inflammation and protect nerve cells. We hypothesized that Ablation of PAFAR gene for example through knock-out can be prevent the binding PAF to PAFAR and realizing inflammatory cytokines .so this can be a convenient way to reduce inflammation and recurrence of the disease. We suggest that it can be great target treatment in patients with relapsing-remitting MS.

P144

Sunflower Mannose binding Lectin-Associated Serine Protease Inhibitor-1 (SFMI-1) and -2: Significant Inhibitors of Mannose binding Lectin Pathway which Helps in Multiple Sclerosis Treatment

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One of the important parts of innate immunity is complement system that occurs in three different ways; the classic, the alternative and the lectin pathway. The four pattern recognition molecules that have been identified till now are Mannose binding lectin (MBL), a component of lectin pathway, and three ficolins (ficolin1,-2 and -3) which compound to the carbohydrates of the cell surface. MBL associated serine protease1 (MASP-1), MASP-2 and -3 are three proteases which associate with recognition molecules. Also MBL-associated protein 19 and MBL-associated protein 44 are two non-catalytic molecules that their role is association with recognition molecules. MASP-1 and MASP-2 activate the lectin pathway but function of MASP-3 is unclear. Although some researches show that MASP-3 down regulates activation of two other MASPs and has a similar role like MBL-association 19 and MBL-association 44 that they inhibit MBL pathway too. Researches show that MBL pathway has a critical role in pathogenesis of autoimmune diseases such as multiple sclerosis (MS). Researches indicate that levels of MBL pathway activator components (MASP-1 and MASP-2) are higher in serum plasma of MS patients. Inhibiting activators of MBL pathway seems to be useful for MS treatment and reducing its disabilities. Sunflower MASP inhibitor-1 (SFMI-1) and sunflower MASP inhibitor-2 (SFMI-2) are two peptides with 14 amino acids that inhibit MASP-1 and MASP-2 and block the lectin pathway activation. This article suggests using SFMI-1 and SFMI-2 in drugs to targeted therapy of MS and decreasing its symptoms.

P145

A Review of Animal Models of Absence Epilepsy

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The most common type of childhood-onset epilepsy syndrome is childhood absence epilepsy (CAE) with well-defined electro clinical features but unknown pathological basis. The incidence of absence epilepsy is about 2 and 8 out of every 100 000 children up to the age of 16, and the prevalence is 2 and 10% of children with any form of epilepsy. Children with CAE suffer from high rate of pretreatment attention deficits that persist despite seizure freedom. Many researchers have still focused on the phenomenon of the absence seizure because of the unclear mechanisms involved in its pathophysiology. Although several models used for screening, quantification and evaluation of absence epilepsy but the key issue is reproducibility of the full clinical syndrome and pathogenesis as well as its different etiology. Considering that each substance has limited duration of action and specific time to observe the seizure. This review includes pharmacological animal models (Systemic Penicillin, Low-dose pentylentetrazole, tetrahydroisoxazolo pyridine and gamma-Hydroxy-butyrate, AY-9944 and methylazzoxymethanol acetate (MAM)-AY-9944 models and genetic animal models (tottering, lethargic, stargazer, mocha, slow-wave epilepsy and ducky mouse, and WAG/Rij, GEARS and Legacies rats). As regards, childhood absence epilepsy has variable genetic etiology; it seems that genetic animal models are more suitable than chemical models, as close correlation of EEG features and behaviors of genetic animal models to the human condition. Among genetic models in mousses and rats the GAERS and the WAG/Rij strains of Wistar, have asserted to be valid and predictive of human absence epilepsy. Most publications were designed based on the WAG/Rij rats. Altogether in both models the thalamocortical circuits obviously involved as the critical generator of absence seizures. Multidisciplinary studies of these two strains, lead to find wealth information about the role of the cortex and the thalamus, and other subcortical circuits.

P146

The Best New Antiepileptic Drugs to Prevent Orofacial Malformations as Side Effects of Antiepileptic Drugs

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Mothers exposed to Antiepileptic drugs (AEDs) are at high risk to born babies with orofacial malformations such as cleft lips. About 9 percent of congenital abnormalities in babies of mothers with epilepsy relates to orofacial abnormalities. Maintaining safe antiepileptic drugs for millions of mothers with epilepsy is very important. Researches show that new AEDs have less side effects than the old AEDs. But it should be consider that these new drugs are initially licensed for adult patients and there are few researches about their effects on pregnant women. So the new AEDs with the least side effect should be definite to make better choices in treatment of pregnant women with epilepsy. Vigabatrin, lamotrigine, rufinamid, levetiracetam, oxcarbazepine, zonisamide, topiramate, lacosamide, eslicarbazepine, Valproate and perampanel were included in our study. Articles indicate that Valproate has the most risk of orofacial malformations overall. Also lamotrigine and rufinamide are the most tolerated drugs and the least prevalence of orofacial malformations have been seen in babies that their mothers used the drugs during pregnancy. Standard dose of lamotrigine and rufinamide in pregnant women should be consider and need more studies.

P147

Role of Sparstolonin B in Intracerebral Hemorrhage-Induced Inflammatory Brain Injury: Blocking the Formation of TLR2/TLR4 Heterodimer

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Intra-cerebral hemorrhage (ICH) is a particularly severe type of stroke accounting for 10–15 % of all strokes and is associated with a mortality rate of 30–50%. Neuroinflammation contributes to ICH-induced secondary brain injury and understanding the mechanisms causing neuroinflammation can be helpful to find new treatments of ICH. Recent studies demonstrated that toll like receptor 2 (TLR2) forms a heterodimer with TLR4 mediated ICH-induced inflammatory injury and the Hemoglobin released following an intracerebral bleed, triggers the formation of TLR2/TLR4 heterodimer through the myeloid differentiation primary response gene 88 (MyD88). Sparstolonin B (SsnB), a novel bioactive compound obtained from a Chinese herb Sparganium stoloniferum is believed as an anti-inflammatory compound that can suppress the inflammatory responses of macrophages to ligands for TLR2 and TLR4. SsnB has also been shown to block signaling pathways following TLR2 and TLR4 activation. It has been suggested that SsnB may be an antagonist to TLR2 and TLR4. But its effect on formation of the TLR2/TLR4 heterodimer remains still unclear. We hypothesize that SsnB would block TLR2/TLR4 heterodimer formation. So the administration of SsnB can interfere with the assembly of the TLR2/TLR4 heterodimer and may be a potential therapeutic approach in the treatment of ICH.

P148

The Role of Cannabinoids in Ischemia Stroke

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Inflammation serves a protective function in controlling infections and promoting tissue repair, and can also cause damage to tissue and disease. Many types of cells involved in this process, expressing the components of the cannabinoid signaling system that can be controlled endogenously or pharmacologically. Cannabinoids inhibit neuroinflammation and the immune cells express the whole machine which constitutes a functional cannabinoid signaling system. Two cannabinoids G receptors coupled to proteins were cloned; CB1 receptors were expressed primarily by neurons and CB2 receptors, which are mainly expressed by immune cells. Cannabinoids have anti-inflammatory effects Animal models of neuroinflammation. Inflammation of the central nervous system can be occurred as a secondary injury in Ischemic stroke is a major cause of death and disability in major industrialized countries. It has been reported that activation of CB1 and CB2 receptors prevented neuronal death in response to ischemia. We suggested the evaluation of agonist or antagonist of cannabinoid receptors may be effective on apoptosis and inflammation in ischemic stroke.

P149

Effect of Glycoprotein IIb/IIIa Inhibition on Acute Ischemic Stroke Injuries

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Ischemic stroke accounts for about 87 percent of all cases. It occurs as a result of an obstruction within a vessel of the brain and sudden loss of blood circulation to the corresponding area resulting in the loss of brain function. It is caused by thrombotic or embolic occlusion of an artery and is more common than hemorrhagic stroke. We know that most of the injuries after an acute ischemic stroke are due to thrombosis formation and the following neuroinflammation (thromboinflammation). So by blocking this pathway we can ameliorate the injuries and the infarct size and improve the brain function after an acute stroke. Platelet von willebrand factor (VWF) is a glycoprotein involved in hemostasis. It is released from the platelet alpha granules and binds to glycoprotein IIb/IIIa complex which forms a bridge between the sub endothelial surface and the platelet and promotes thrombosis formation. On the basis of this information, we hypothesize that we can alleviate the injuries of acute ischemic stroke by blocking the glycoprotein IIb/IIIa complex via a specific antagonist antibody and so preventing thrombosis formation. It can be a potential therapeutic approach in the treatment of acute ischemic stroke.

P150

The Role of Blood Brain Barrier Restoration in the Multiple Sclerosis

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Blood Brain Barrier (BBB) is a specialized non fenestrate barrier that formation by the endothelial cells and controls the transportation of the cells and molecules in to the brain. Reducing in function of BBB is one of disruptions in neurological diseases like multiple sclerosis. Endothelial progenitor cell (EPC) help to the BBB to control the diapedesis of inflammatory cells & molecules in to the Brain with decrease ICAM-1 in blood. However they increase angiogenesis around the BBB. Multipotent Adult Progenitor Cells (MAPCs) increase the proliferation of the M2 macrophage-like cells and apoptosis of the M1 macrophage-like cells at 3-7 days after transplantation. However this mechanism of MAPCs decrease the inflammation of the Central nervous system (CNS) and BBB but because of the short acting time, it's not very useful. Mesenchymal stem cells (MSC) are of the mainly hope to repair BBB. They can balancing surface of the B-catenin and cadherin. This balance can create some tight junctions in to the BBB to help controlling the transportation. In addition they can increase secretion of the metalloproteinase -3 which can improve the function of the BBB. Hematopoietic stem cells are the one the best candid for MS treatment. They can restart immune system to control proinflammatory mediators. They also can decrees the expression of the CD8 & CD4 to prevent of developing of inflammation. However there are many studies to investigated cell therapy and its effect on CNS, there is no powerful study to peruse cell therapy for restoration of the BBB in multiple sclerosis. We suggest to study on it as an Individual treatment.

P151

Clinical Trial in Cell Therapy of Multiple Sclerosis

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Neuroinflammation is a disorder that causes neurological disease. Neuroinflammation has a significant role in induce of Multiple sclerosis (MS) and one of the situations that must be treated stops the ongoing process of inflammation against the CNS by self-reactive lymphocytes. According to the successful results that were obtained from the pre-clinical phase of cell therapy, many studies were performed in clinical phase which resulted in the improvement of clinical symptoms, and in most of them, the quality of life and reduced relapsed were observed. In a comprehensive study, 500 MS patients worldwide were treated with Hematopoietic cells. 100% of the patients suppressed or reduced inflammation as well as the brain atrophy, which was also an inflammatory complication that was reduced within the patients. All studies in the field of cell therapy show high performance and effectiveness of this approach for the treatment of patients with multiple sclerosis. Even in the most severe stage of the disease (aggressive and resistant forms), the treatments resulted in a positive outcome within the patients; proving that this treatment is optimal for current patients suffering this disease. According to this successful therapeutic method, in recent years the complications were dropped and its severity was minimized crucially in patients who

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were under the age 40 and the duration of the disease was less than 5 years. Considering that these pharmacological interventions were available for MS patients to have numerous side effects such as Leukoencephalopathies, Thrombocytopenia, Autoimmune and Kidney disease, therefore Phase III clinical trials for comparing and selecting the best possible method is needed to enable the most effective diagnosis of cell therapy treatment for these patients. Also, cohort study in this field should be done to discover the advantages and disadvantages of this method.

P152

Mesenchymal Stem Cells as a Therapeutic Target in Multiple Sclerosis

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Neuroinflammation has a significant role in induce of Multiple sclerosis (MS) many approaches have been used to treat MS, but none of these methods have not been able to fully improve. One of the methods can suppress inflammation and regenerate the nervous system is the use of cell therapy. Using cell therapy in pre-clinic phase can be realized, it's mechanism and potency to suppress neuroinflammation. The best way that plenty of researchers use it to simulate the MS condition is experimental autoimmune encephalomyelitis (EAE) which is method can be induced neuroinflammation in laboratory animals. In this context, a lot of researches have been done on EAE model. Many of these studies have been done on mesenchymal stem cells (MSC). MSC is a heterogeneous subset of, mesoderm stromal progenitor cells that are almost derived from connective tissue. MSCs can be obtained from adipose tissue, bone marrow, and umbilical cord that regulatory and inhibitory effect on the immune system. Transplantation of adipose-derived stem cell (ASCs) has demonstrated striking therapeutic effects and unique immunomodulatory capacities when delivered at the peak or later in the course of the disease in EAE rats. Recent studies have shown that umbilical cord-derived mesenchymal stem cells (UC-MSCs) exert a regulatory effect on the functions of immune cells. UC-MSCs could improve the impaired function of T-regulator cells (Treg) from MS patients and also enhanced the capacity of Tregs to release IL-10. There is still controversy about the use of UC-MSCs and ASCs, and more research is needed to determine the advantages and the disadvantages of them also in vitro method such as EAE cannot simulate all condition of disease therefor more research in clinical phase should be done.

P153

Neuroinflammation in Multiple Sclerosis

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Multiple sclerosis (MS) is a complex disease which is correlated with increasing inflammatory factors, demyelination and axonal loss. In this auto-immune disease, Neuroinflammation is mediated by different types of T cells with macrophage/microglial activation and B cells involvement that interact in a collaborative manner. Focal inflammation is the main cause for the onset of relapses and could be presented in different regions of the central nervous system (CNS), but as the disease advances to progressive form, immune-inflammatory and oxidative stress pathways, which lead to axonal damage, play major role in the development of the disease. Neuroinflammation in MS can be studied for many approaches. For instance, a recent study have reported new classification of MS lesions including active, mixed active/inactive and inactive lesions for better comparison of tissue pathogenesis based on the inflammatory activity, demyelinating areas and duration of the disease. Moreover, it has been recently shown that the presence of leptomeningeal inflammation could be an important hallmark mainly for diagnosis of the progressive form of MS. Neuroinflammation could also be found in deep gray matter with pathological and clinical relevance. Finally, CNS injury in MS is associated with inflammatory reactions with further axonal degeneration. Therefore, control of inflammation with anti-inflammatory therapies must be taken into account as one of the main purposes of MS treatment parallel with other immunomodulatory and immunosuppressive treatments.

P154

The Role of Inflammation in the Seizure Occurrence

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Most common hypotheses of seizure initiation are increased neural excitation, decreased inhibition or both. But, the conditions that lead to these activation states

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not to be clear yet. Recent studies challenge traditional concepts and indicate new evidence that a key epileptogenic process may actually begin in the blood vessel. Seizures could be initiate by a variety of insults to the brain, such as trauma, infection, hypoxia, fever. Recent research has highlighted putative role of blood-brain barrier (BBB) permeability in the evolvement of epilepsy. Local or global changes of the brain's homeostatic environment can pathologically alter neural activity via admission of intravascular proteins such as albumin and altered electrolyte levels such as potassium. Investigations showed Immune-mediated damage to the nervous system is extruding as an important contributor to epileptogenesis, both directly through inflammation and indirectly by causing BBB leakage. pilocarpine administration as a valid temporal lobe epilepsy in the animal models increased in cell adhesion molecules in brain blood vessels, which are important moderators for leukocyte extravasation during inflammatory processes. Chiefly, expression of the some intercellular adhesion molecule such as: vascular cell adhesion molecule-1 (VCAM-1), P-selectin and E-selectin was increased at one day and seven days after status epilepticus, compared to control groups. In the similar studies researchers expressed enhanced leukocyte adhesion in the central nervous system vessels. These findings indicate that seizure activity is accompanied with leukocytic inflammatory alteration in the central nervous system vasculature. More important point is that leukocyte-endothelial interactions determined whether seizures occurred, and subsequent extent of structural, cognitive and physiological damage, including the development of epilepsy.

P155

The Roles of Microglia in Neurodegenerative Diseases

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Microglia is a type of glial cell located throughout the central nervous system (CNS), which is sensitive to CNS injury and disease. Responsibility of microglia as the resident macrophage cells for injuries suggests that these cells have the potential to act as diagnostic markers of disease beginning or progression. Function of Microglia is strongly synchronized by the microenvironment of brain and spinal cord, many evidences suggest that neurodegeneration and ageing, can affect microglial phenotype and function. Distinctive potassium channels responsible for sensitivity of the cells to even small modifications in extracellular potassium after even small pathological changes in CNS .Microglia cells are very plastic, and undertake a variety of structural transformations based on location and needs. There are different forms and types of microglias in CNS such as Ramified, Non-phagocytic, Phagocytic, Amoeboid, Gitter cells, Perivascular and Juxtavascular. Microglia cells have a significant role in neurodegenerative disorders, for example there are numerous over expressing InterlukinL-1 microglia in the brains of person with Alzheimer's disease. This over expression of InterlukinL-1 leads to extreme tau phosphorylation that is associated with tangle development in Alzheimer's disease. So according to characteristics and behavior of these cells in different neurodegenerative diseases, activated microglia cells can be one of the main cellular target for therapy.

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