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The 2nd International Neuroinflammation Congress & Student Festival of Neuroscience

Abstract Book

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Oral Presentations

O1

Modeling of Mesenchymal Stem Cell-Derived Magnetite Nanoparticles for The Rehabilitation of Immune System Function and Reducing Inflammation and Promoting Myelination in the Treatment of MS Disease

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By Using the modeling of the mesenchymal (bone marrow) stem cell nanoparticles, the reinstatement of the immune system leads to the treatment of MS, result in the formation of a new immune system for the body by stem cell. The presence of stem cells promotes and s trengthens myelination, and that, using simulation and 3D modeling, stem cells can be transmitted correctly to the target and place of injury and the location of inflammation. Using mathematical modeling and magnetic resonance images and 3D simulation of magnetite nanoparticles that carry stem cells and direct its guidance and modeling the new immune system to improve the function of immune cells and protect myelin in nerve cells and reduce inflammation from the modeling model provides a predetermined data that makes the pathway that the stem cell passes through in a patient that results in higher accuracy and ease of work. Modeling shows that stem cell infiltration can be better controlled by nanoparticles, and the presence of mesenchymal stem cells (BMSCs) plays a major role in rebuilding the immune system and reducing inflammation, and the presence of stem cells to generate signals with surrounding cells Being nervous and res toring the immune system reduces inflammation and thus restores myelin in the central nervous system and the spinal cord. Modeling the pathway in which the nano-particle carrying a stem cell that needs to travel to the site of a damaged lesion in the brain and spinal cord has a pre-designed and planned map that provides better controlled transmission in inflammation. It reduces inflammation and rebuilds the myelin, and its pathway can be seen in MRI images and can be captured at a molecular cellsurface.

O2

Flaxseed Reduces Proinflammatory Factors IL-1β, IL-18 and TNF-α in Injured Spinal Cord Rat Model

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): O2

The pathophysiology of acute spinal cord injury (SCI) involves primary and secondary mechanisms of injury. Secondary injury mechanisms include inflammation, oxidative stress. The secondary inflammation of spinal cord tissue after SCI was critical for the survival of motor neuron and functional recovery. Flaxseed is a rich source of lignan phytoestrogen, α-linolenic acid. Flaxseed has remarkable anti-inflammatory effect. Adult male wis tar rats (n=24) were assigned to four groups: control, laminectomy, SCI and SCI+Flaxseed groups. The SCI model was exerted by placing a 50 g weight for 5 min by a platform applied at the T10 vertebral level. After 4 weeks the blood serum of all rats were collected and the effect of flaxseed on proinflammatory factors level, locomotion score and histologic alterations were assessed. The use of flaxseed significantly decreased the level of proinflammatory factors IL-1β, IL-18 and TNF-α compared to SCI group and improved the motion of the animals in the SCI+Flaxseed group and decreased the demolition of spinal cord tissue after injury. Our study for the first time showed the anti-inflammatory effect of flaxseed on spinal cord injury model in rat, further studies can be done to assess the intake of flaxseed as an effective therapeutic agent for the relative recovery of patients with SCI.

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O3

Pharmacological Modulation of Thalamic KCNQ-Potassium Channels: Insight from Knock-out Mice

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The channels belonging to the KCNQ gene family consis t of 5 different subtypes, which assemble as pentameric channels. The KCNQ2-5 subunits are highly expressed in the ventrobasal thalamus (VB) where they function primarily as KCNQ2/3 heteromers. They underlie an outward potassium (K+)-current, called M-current (IM), which provides a hyperpolarizing drive, thus regulating neuronal excitability. In order to understand the contribution of the KCNQ3 channel subunits to the regulation of the firing patterns and the generation of IM in VB neurons, we performed electrophysiological recordings using a mouse line lacking this subunit (KCNQ3 KO). Application of the specific channel activator Retigabine (Ret) induced hyperpolarization of the resting membrane potential, and significantly reduced the number of action potentials elicited in response to a given current step in control animals. In a similar manner, voltage-clamp experiments showed an increased IM following Ret application, while adminis tration of the specific channel inhibitor XE991 reversed this effect. Preliminary recordings performed in KCNQ3 KO mice indicated a smaller IM amplitude in the same experimental conditions. However, increasing the group size and using other specific modulators will help us understanding better the role of KCNQ3 in VB and indentify potential compensatory mechanisms exerted by other subunits.

O4

Central Nervous System Involvement in Rheumatoid Arthritis: Possible Role of Chronic Inflammation and TNF Blocker Therapy

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Rheumatoid arthritis (RA) is a chronic disease, the etiology of which has yet to be clarified, which causes activation of proinflammatory pathways that bring about joint and systemic inflammation. In recent years, the pathophysiology of CNS involvement that can occur in RA has attracted a great deal of attention. Emphasis has focused on the possibility that CNS involvement occurs due to blood-brain barrier (BBB) damage associated with chronic inflammation. The present study was performed to investigate the possible effects of BBB dysfunction and tumor necrosis factor (TNF) blocker therapy on BBB function, which may cause CNS damage in patients with RA. 90 RA patients [65 females, 25 males] and 40 healthy controls [25 females, 15 males] were included in the study. All RA patients were on synthetic DMARD therapy at the beginning. 55 patients continued DMARD therapy, and 35 patients with high disease activity were started on TNF blocker therapy. All demographic characteristics of the patients were recorded. Disease activity was evaluated using the Disease Activity Score 35-joint count C reactive protein. The Mini-Mental State Examination was used to evaluate cognitive function, and the Fazekas scale was used to assess cranial lesions visualized by magnetic resonance imaging (MRI). Patients' peripheral blood S100β, glial fibrillary acidic protein (GFAP), claudin, interleukin (IL)-17, and IL-1β levels were measured at the beginning of the study and after 6 months. Demographic characteristics (including sex, age, and body mass index) were similar in the RA and control groups. S100β and GFAP levels were significantly higher in the patient group than in the control group. In the group that was started on TNF blocker therapy, S100β and GFAP levels were significantly decreased 6 months after commencement of treatment. No difference was observed between the RA and control groups in terms of hyperintense lesions seen on cranial MRI. The S100β levels increased with lesions in the deep white matter seen on cranial MRI in patients with RA. next to decreasing disease activity and joint erosions by suppressing inflammation, anti-TNF therapy in RA can also suppress potential CNS involvement linked to BBB (blood-brain barrier) dysfunction.

O5

A Self-Assembled Nanopeptide Scaffold Combined with Mesenchymal Stem Cells Improved Functional Recovery after Traumatic Brain Injury in Rats

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): O5

Traumatic Brain Injury (TBI) is a major cause of death and disability worldwide. TBI can cause cognitive and memory function impairments which Current medical setting is not able to cure. In this study, we hypothesized that mesenchymal stem cells derived from Adipose tissue transplanted with RADA1- GGS IKVAV (GSIKVAV) can rescue cognitive function. An acute model of TBI was carried out in male Wistar rats (n=36). Subjects were divided into 7 groups: Sham (receiving no treatment), PBS, GSIKVAV, Mesenchymal Stem Cells (MSCs), GSIKVAV+ MSCs. MSCs were stained with BrdU. To evaluate the MSCs characterization flow cytometery was performed. IHC was done to s tudy the differentiation and viability. Behavioral tests including Open Field (OF), Elevated Plus Maze (EPM) and modified Neurological Severity Score (mNSS) were done to evaluate the sensory- motor function and anxiety. At day 30, animals were sacrificed. In order to detect inflammation toll like receptor3, 4, tumor necrosis factor α and glial fibrillary acidic protein were assessed using western blot. CD markers (CD 105, 34) were reported to be positive for most of MSCs. The mNSS score significantly decreased among MSCs and MSCs + GSIKVAV groups. We observed a significant increase in the number of enteries to the open arm in the EPM test. The total distance was also increased in the OF tes t. These data suggest that using MSCs with GSIKVAV can be a potential therapy for TBI patients according its neuroprotective and immunomodulatory effects.

O6

Protective Effect of Alpha-Lipoic Acid on Neuronal Degeneration Due to Sciatic Nerve Transection in Rat

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): O6

Peripheral nerve injury induces inflammation and oxidative stress, which are the most significant causes of the neuronal death. Alpha Lipoic acid (α-LA) as a potent antioxidant and anti-inflammatory agent may counteract the oxidative stress and inflammatory response. This s tudy was designed to investigate the protective effect of α-LA on neuronal cell death in L4 dorsal root ganglion (L4-DRG) induced by unilateral sciatic nerve transection (SNT) in rat. Thirty male Wistar rat were divided into 5 groups (n=6); control (intact), SNT+ Salin, SNT + α-LA (100 mg/kg; i.p), SNT+ vitamin C (150mg/kg: i.p) and sham. Treatment was started 1 hour after injury and continued up to 7th-day post-injury. At 21st day pos t-injury, the L4-DRG were dissected out, fixed (formalin 10%), processed for paraffin embedding. Serial sections of L4-DRGs were prepared, stained (H&E and Toluidine blue) and then examined microscopically. The mean volume of L4-DRGs was estimated using Cavalieri principle and neurons count was done using a stereological technique (Disector method). Data were analysed with SPSS statistics 16.0 software ANOVA and intergroup comparisons performed using a Tukey-Post how analysis. In comparison with control, the number of neurons in SNT + α-LA and sham groups had no significant differences. The number of neurons in the SNT+Salin and SNT+ vitamin C were significantly reduced ($P<0.05$). α-LA (100 mg/kg.bw) provides comparable protection of sensory neurons after axotomy unlike vitamin C in rats. It seems that α-LA is a profound neuroprotective and promising anti-inflammatory agent in healing peripheral nerve injury.

O7

Functional Characterization of Human GABAA Autoantibodies in the Context of Limbic Encephalitis

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Limbic encephalitis is an adaptive autoimmune disease, induced by different autoantibodies, which target extracellular neuronal epitopes, such as NMDA or GABAB receptors1GABA(B,2consequences of autoimmune inflammation of the amygdala are largely unknown. The amygdala is central for the generation of adequate homoeostatic behavioral responses to emotionally significant external stimuli following processing in a variety of parallel neuronal circuits. Here, we hypothesize that adaptive cellular and humoral autoimmunity may target and modulate distinct inhibitory or excitatory neuronal networks within the amygdala, and thereby strongly impact processing of emotional stimuli and corresponding behavioral responses. This may explain some of the rather poorly understood neuropsychiatric symptoms in limbic encephalitis.", "author" : [{ "dropping-particle" : "", "family" : "Melzer", "given" : "Nico", "non-dropping-particle" : "", "parse-names" : false, "suffix" : "" }, { "dropping-particle" : "", "family" : "Budde", "given" : "Thomas", "non-dropping-particle" : "", "parse-names" : false, "suffix" : "" }, {

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O8

Evaluating the Impact of Lactobacillus Acidophilus Probiotic Supplement on Sensory-Motor Recovery in a Model of Traumatic Brain Injury

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Traumatic brain injury (TBI) is a leading cause of death and disability worldwide and many survivors experience a wide range of neurological impairments after TBI. Following the initial mechanical injury at the moment of a TBI event, various cellular and molecular processes are activated as the secondary injury. Neuroinflammation is an important mechanism involved in the secondary injury of TBI. Therefore, Neuroinflammation offers a promising opportunity for therapeutic intervention in order to prevent progressive tissue damage and improve the neurological recovery after TBI. The use of probiotics as a novel therapeutic option for modulating inflammatory response has received great attention, but there are still insufficient data on whether probiotics have the ability to regulate neuroinflammation, and further research needs to be done to elucidate the impact of probiotics on neuroinflammation and neurological recovery. If proven effective, probiotics can be used as a cheap, non-invasive, easy-to-use and safe treatment for modulating post-TBI neuroinflammation. Several studies have reported that probiotic compounds reduce serum inflammatory cytokines and increase the levels of anti-inflammatory cytokines. Hence, we hypothesize that using probiotics after TBI might have the potential to regulate neuroinflammatory response and thus, improve the neurological recovery.

O9

Mechanisms and Therapeutic Options in Multiple Sclerosis

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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.

O10

Thrombo-Inflammation in Acute Ischemic Stroke

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Ischemic stroke has been classified as a merely thrombotic disease, so the main goal of its treatment is the recanalization of the occluded vasculature. However, despite fast restoration of blood circulation, progressive stroke still develops in many patients, which has led to the concept of reperfusion injury. The underlying mechanism is only partly known. Though, it is accepted now, that thrombotic and inflammatory pathways are key contributors to ischemic stroke, leading to the concept of thrombo-inflammation. In the acute phase after stroke, thrombo-inflammation occurs at the site of ischemic vascular injury, where platelets bind to von Willebrand factor and become activated. Downstream cascades lead to the activation of the contact-kinin system, resulting in endothelial cell damage, expression of pro-inflammatory cytokines, recruiting immune cells to the side of damage, and supporting immune cell migration into the brain. Activated platelets, the damaged vasculature and attracted immune cells conspire together inducing secondary thrombotic events. In our stroke studies, we significantly improved the s troke outcome in rodents after blocking members of the thrombo-inflammation system (e. g. platelet receptors (GP1b), blood coagulation proteins (Kinin, F12), or immune cells (T cells)) in the acute phase. Therefore, anti-thrombo-inflammatory strategies could become novel treatment approaches in the future.

O11

Novel Approaches to Prevent Neuroinflammation by Targeting the Coagulation System

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There is growing appreciation that other factors not traditionally considered components of the immune system foster inflammation in multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE). The blood coagulation initiating factor XII was introduced as key mediator of central nervous system (CNS) autoimmunity by modulation of adaptive immune response. Moreover, these findings were indicated to be relevant in MS patients, as further demonstrated: levels of factor X (FX) and prothrombin are increased in treatment-naïve MS patients compared to healthy donors. This concept of a pivotal role of the coagulation system in neuroinflammation was further reinforced by our recent identification of the preventive effect of FX inhibition in EAE on neurological deficits and local inflammation compared to control animals. Platelets have been also recognized to contribute to EAE. Thus, the identification of platelet specific candidates relevant for disease activity is crucial for the understanding of platelet mediated contributions to CNS inflammation currently being addressed by ongoing studies. As the pathophysiology of MS remains poorly understood to date, the addition of novel mechanisms of neuroinflammation can broaden our knowledge about disease development and may even help in establishing novel therapeutic approaches targeting the coagulation system.

O12

The Heart and the Brain: Stroke Induced Heart Damage

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Cardiac diseases are common post-stroke and are associated with increased morbidity and mortality. One possible mechanism of acute cardiac injury is the neurogenic myocardial damage, where the cerebral injury is disturbing the normal sympathetic and parasympathetic neuronal outflow to the heart leading to cardiac damage including myocardial infarctions. The exact mechanism is not completely understood and the major objective of this project is to characterize the molecular phenotype of the neurogenic myocardial damage post-stroke. Our data demonstrate acute myocardial damage in wild-type mice after right or left-sided transient middle cerebral artery occlusion or photothrombosis. We analyzed the stroke outcome with a neurologic score and the stroke volume. For the myocardial damage, we measured catecholamines levels, the heart damage marker Troponin, and performed electrocardiograms in mice. Using different inbred wild-type mice strains (C57BL/6 J/N; Balb/c) for purposes of comparison, the stroke induced heart damage evolved highly divers. While some strains were protected agains t stroke and myocardial damage and had a much better survival and stroke outcome, others showed an increased morbidity and mortality. For our mechanistic analysis of the neurogenic myocardial damage, we compared different strains and stroke methods to get the right model with more heart damage, but without an increased mortality.

O13

Human Neural Stem/Progenitor Cells Derived from Epileptic Human Brain in A Self-Assembling Peptide Nanoscaffold Attenuates Neuroinlammation in Traumatic Brain Injury in Rats

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Traumatic brain injury (TBI) is a disruption in the brain functions following a head trauma. Cell therapy may provide a promising treatment for TBI. Human neural stem cells cultured in self-assembling peptide scaffolds have been proposed as a potential novel method for cell replacement treatment after TBI. In the present study, we accessed the effects of human neural stem/progenitor cells (hNS/PCs) derived from epileptic human brain and human adipose-derived stromal/stem cells (hADSCs) cultured in the RADA16 on brain function after TBI. hNS/PCs were isolated from patients with medically intractable epilepsy undergone epilepsy surgery. hNS/PCs and hADSCs have the potential for proliferation and differentiation into both neuronal and glial lineages. Transplantation of hNS/PCs and hADSCs encapsulated in the PM inhibited neuroinflammation, and reduced the reactive gliosis at the injury site of TBI. The data sugges t the transplantation of human stem cells encapsulated in the PM as a hopeful treatment option for cell therapy in TBI.

O14

Application of Neural Stem Cells Derived from Human Meningioma in Traumatic Brain Injury

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Traumatic brain injury is considered as one of the main causes of morbidity and mortality worldwide. Apart from primary mechanical injury, Secondary injuries due to inflammation and apoptosis result in great neuronal damage. Current treatments are not able to regenerate the damaged part and prevent future sequels. Using human stem cells with self-assembling scaffolds may be promising in treatment of traumatic brain injury. Human meningioma stem cells were isolated, cultured and expanded into in vitro condition. The rat models of TBI were divided into 5 groups: sham, PBS, stem cells, scaffold and stem cell+scaffold. mNSS and EEG were performed to evaluate movement and physical activity. IHC were done to assess cell differentiation. Inflammation and apoptosis markers like toll like receptors, caspase 3,8 and TNF-a were analyzed using western blotting and PCR methods. Results showed that inflammation was significantly reduced in cell group. Tissue engineering as a new therapeutic method can be promising in treating brain damage.

O15

Using Stromal Cell-Derived Factor-I as Bio Active Motif in A Novel Self-Assembly Peptide Nanofiber Scaffold: an Approach to Improve Cell Therapy in Brain Injury

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Traumatic brain injury (TBI) is one of the main causes of mortality and morbidity worldwide. Despite extensive investigations over the past few decades, no effective therapies exist to improve the brain function in patients with TBI. Neural tissue engineering is an attractive therapeutic approach to restore the brain structure and function of damaged tissue. Bioactive motif of Stromal cell-derived factor-1 (SDF-1) induces neurogenesis by increasing the migration and proliferation of endogenous neural progenitor cells (NPCs) in the lesion sites. We designed the novel scaffold with SDF1 and RADA16. The Aim of this study is to determine in vitro effects of SDF scaffold on neural stem cells behavior including migration, attachment, and differentiation. Neural stem cells were isolated from the hippocampus and subventricular zone of the lateral ventricle of 17-days rat fetus. In this study, Apoptosis, cytotoxicity, proliferation, neurite outgrowth, and differentiation were assessed. Migration, attachment and differentiation of stem cells significantly increased in the SDF scaffold. Our results showed no significant difference between apoptosis, survival and proliferation of cells in SDF and RADA16-IKVAV scaffolds. Although SDF scaffold increased Migration, and attachment of stem cells in

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vitro, in vivo studies should be conducted to determine the features of SDF scaffold in the brain tissue.

O16

Long Term Video EEG (AC/DC) Monitoring

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Long term video electro-encephalographic (EEG) monitoring (LTM) is defined as the continuous and synchronized recording of EEG and multimedia to analyze brain abnormalities. A conventional LTM system continuously records EEG in the frequency range of 0.5-70Hz. The data synchronization, the high-volume data management, the system reliability as well as noise reduction remain significant challenges in an LTM machine. Moreover, online multi-user access to the data during the recording as well as online EEG control remains another essential point. Beyond conventional LTM systems, recent studies indicate the existence of a potentially clinically-relelvant near-DC field potential (f<0.1Hz) among the EEG data. This DC field potential, although sometimes present in healthy subjects, also appears to be associated with a variety of brain abnormalities such as migraines with auras and some types of epileptic activity. Therefore, the EEG frequency range must be extended to include lower frequencies down to 0.01 Hz. The potential asymmetric changes of the bio-electrodes in ultralow frequencies, effects of the scalp - skull structures and its capacitance features, as well as EEG instrumentation, are significant challenges that need to be addressed to provide DC EEG to a conventional LTM machine. We propose a new technique and system to capture and review the AC/DC LTM data. The proposed system aids in analyzing both high and low-frequencies oscillations of brain electrical activities.

O17

Inflammation in Brain and Spinal Cord

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): O17

our goal in this paper is to describe and compare basic immunopathologic pattern of common demyelinating disorder, that is very important to choose the best treatment. The most common disorders are multiple sclerosis, neuromyelitis optica,Anti MOG associated disease,ADEM and autoimmune encephalitis. ADEM consists of "sleeves" of demyelination centered on small, engorged venules.Significant inflammatory infiltrates consist of myelin-laden macrophages.Variable T and B lymphocytes,and occasional plasma cells and granulocytes May coexit ,Perivenous demyelinating lesions may coalescence to form larger areas of demyelination,but the MS-characteristic joint areas of demyelination, macrophage infiltration, and reactive astrocytes are not typically seen in ADEM.Multifocal cortical-microglial aggregates scattered throughout the cortex, not associated with cortical demyelination. NMOSD is an autoimmune astrocytopathy that causes secondary demyelination..The presence of vasculocentric pattern of immunoglobulin. and complement activation in active NMO lesions suggested a humoral-mediated injury. The water channel aquaporin-4 (AQP4), expressed on pericapillary foot process of astrocytes, has been identified as the target antigen in NMO. T-helper 17 (Th17) cells is very important and elevated serum and CSF levels of interleukin 6 (IL-6), with elevated IL-17 in CSF were seen. In biopsy, active demyelination with macrophages containing myelin oligodendrocyte glycoprotein (MOG), immunoreactive myelin debris, were seen adjacent to periplaque white matter. AQP4 is lost in the active lesion but retained in the periplaque white matter, unlike MS, that show increased AQP4 expression on astrocytes.We can see loss of immunoreactivities to astrocytic proteins, glial fibrillary acidic protein (GFAP) matter. ANTI MOG-IgG demyelination with relative axonal preservation and the presence of complement activation markers, which is similar to some histopathological descriptions of MS pattern II demyelination. Distinct from AQP4-IgG-associated NMOSD, no astrocytopathy is seen in these patients. Multiple sclerosis is an inflammatory, demyelinating, and is not only an autoimmune disease in which autoreactive immune cells against myelin, damage axons and nerves in the CNS , but also a neurodegenerative disease of the CNS. CD4+ helper T (TH) cells types 1 and 17 release cytokines and Inflammatory mediators that cause tissue damage, while CD4+ TH2 cells might be involved in modulation of these effects.Macrophages that containing proteolipid protein-immunoreactive myelin debris, were seen adjacent to periplaque white matter .Confluent demyelNination admixed with reactive astrocytes (Creutzfeldt-Peters cells)are considerable.Evidence suggests significant humoral immunity involvement in the disease process(Bcell) .Lymphocytic inflammatory infiltrates are composed mainly of CD8-positive cytotoxic T lymphocytes, and fewer CD4-positive helper T cells.No MS spesicific autoantigen has been identified. Autoimmune Encephalitis envolvement of cortical and white matter disease cause by antibodies against intra and extracellular antigen.In biopsy clusters of mononuclear inflammatory cells mainly composed of CD3+ , CD4+ , and CD8+ T cells.B cells are scarce

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and mainly restricted to the perivascular space and meninges .Syndromes are associated with antibodies against intracellular (onconeuronal) antigens, appear to be mediated by cytotoxic T-cell responses,against extra cellular antigen related disease that is prominently Bcell type. we can treat MS and ADEM patients with both Bcell and Tcell type associated treatment agains t Autoimmune encephalitis and NMOSD and Anti MOG associated disease that must be treated with Bcell associated ones.

O18

Autoimmune Encephalitis (Pathophysiology, Clinical Signs and Diagnos Tic Tests)

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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. Autoimmune encephalitis involves several types: The first group includes the classic paraneoplastic disorders associated with antibodies to intracellular antigens. The second group involves autoantibodies to extracellular epitopes of ion channels, receptors and other associated proteins, such as the NMDA receptor. 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 consis ts of subacute (days to a few weeks) progressive decrease in the level of consciousness, often with fluctuations, and altered cognition.

DIAGNOSTIC APPROACHES

Antibody testing: Autoantibody testing is extremely important for the proper diagnosis of autoimmune encephalitis.
Imaging
EEG
Biopsy
Cancer screening

Treatment Approaches: Treatment for suspected autoimmune encephalitis is often given empirically prior to specific antibody test results. This may include s teroids and/or IVIG. If a cell-surface/synaptic antibody disorder is diagnosed, initial treatments may include IVIG, plasmapheresis, and/or steroids. 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 pathogen.

O19

Advances in the Treatment and Limitations of Cell Therapy in Neurodegenerative Diseases

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Neurodegenerative diseases are the hereditary and sporadic diseases which are characterized by progressive neuronal loss of the nervous system and are emerging as the leading cause of death, disabilities, and a socioeconomic burden due to an increase in life expectancy. There are many neurodegenerative diseases including Alzheimer's disease, Parkinson's, disease, amyotrophic lateral sclerosis, Huntington's disease, and multiple sclerosis but There are no effective treatments or cures to halt the progression of any of these diseases. Stem cell-based therapy has become the alternative option to treat neurodegenerative diseases. There are several types of stem cells utilized;
1- Embryonic stem cells, induced pluripotent stem cells, and
2- Adult stem cell (mesenchymal stem cells and neural progenitor cells).
In this review, we summarize recent advances in the treatments and the limitations of various stem cell technologies. So, we focus on clinical trials of stem cell therapies for major neurodegenerative diseases especially multiple sclerosis.

O20

NK Cells as Surrogate Marker for Predicting Treatment Efficacy in Chronic Inflammatory Demyelinating Polyneuropathy

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Natural Killer (NK) cells are part of our innate immune system with regulatory and effector functions. Different studies suggest that treatment with intravenous immunoglobulins (IVIg) has an immunomodulatory effect on NK cells. IVIg is a first-line treatment for various autoimmune diseases in particular in chronic inflammatory demyelinating polyneuropathy (CIDP).

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The lack of predictive markers for IVIg responsiveness in CIDP avoids the early preservation of non-responding patients. Using semi-quantitative PCR and flow cytometry in the peripheral blood of patients with CIDP, we analysed the effects of IVIg on the NK cells and correlated changes with the IVIg reponsiveness. IVIg administrations induced a reduction in the expression of several typical NK cell genes. Flow cytometry data revealed that IVIg reduced the cytotoxic CD56dim NK cell population, while regulatory CD56bright NK cells remained almost unaffected or were even increased. Interestingly, the observed effects on NK cells almost exclusively occurred in IVIg responding CIDP patients. Correlation between changes in the NK cell population and treatment efficiency suggests a crucial role for NK cells in the immunomodulatory mechanism of IVIg. Further studies will investigate whether differences in the NK cell status of CIDP patients represent a reliable surrogate marker predicting the outcome of IVIg therapy.

O21

Aquaporinopathy and Cerebral Inflammation

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Many mammalian AQPs, including AQP1, AQP2, AQP4, AQP5 and AQP8, function primarily as bidirectional water-selective transporters. Cells expressing AQPs on their plasma membrane have an ~5- to 50-fold higher osmotic water permeability than membranes that do not. Water transport through single-file pores poses a biophysical limitation on the efficiency with which AQPs can transport water, so that AQPs must be present in the membrane at a high density to increase membrane water permeability substantially. Aquaporin-4 (AQP4), the target antigen of NMO-IgG, is a water channel protein highly concentrated in spinal cord gray matter, periaqueductal and periventricular regions, and astrocytic foot processes at the blood-brain barrier. It is now clear that NMO-IgG (anti-AQP4) plays a direct role in the pathogenesis of NMO. In MS lesions, the distribution of AQP4 protein expression depends upon the stage of demyelination, while in NMO lesions, there is a loss of AQP4 expression that is unrelated to the stage of demyelination. The inflammatory processes in NMO primarily targets astrocytes]; the area pos trema appears to be a preferential target of NMO-IgG antibodies that bind to astrocyte AQP4 water channels, leading to astrocyte dysfunction and the clinical manifestations of nausea and vomiting. Central nervous system involvement outside of the optic nerves and spinal cord is recognized in patients with NMO and NMOSD. Other suggestive symptoms include episodes of intractable nausea, vomiting, hiccups, excessive daytime somnolence or narcolepsy, reversible pos terior leukoencephalopathy syndrome, neuroendocrine disorders, and (in children) seizures. While no clinical features are disease-specific, some are highly characteris tic. Manifestations that can develop with NMO and NMOSD include encephalopathy, fulminant cerebral demyelination, hypothalamic dysfunction, and posterior reversible leukoencephalopathy. Symptoms related to bilateral hypothalamic lesions may include symptomatic narcolepsy or excessive daytime sleepiness, obesity, and various autonomic manifestations such as hypotension, bradycardia, and hypothermia. In rare cases, fulminant diffuse vasogenic edema can lead to brain herniation and death.

O22

Principles of Treatment in Neuro-Inflammation Disorders

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Treating Central Nervous System (CNS) neuro-inflammational disorders is of great importance. A key aspect of this treatment includes regulation of Cell-Mediated and humoral immune systems. Previous treatment mostly consisted of systemic immunosuppressive drugs. In addition to having adverse side effects, these drugs were also inefficient, such as methotrexate- Cyclophosphamide-Azathioprine. Recent advances in medicine introduced more effective methods with less side effects, including:

Intravenous Immunoglobulin (IVIG): Using IVIG neutralizes Pro-inflammatory antibodies, thus regulating the inflammation. It is widely used in treating Guillain-Barre syndrome.

Plasma Exchange: In this method antibodies are removed; Therefore significantly reducing the inflammation. This method is mainly used in treating central and peripheral nervous system inflammations such as Guillain-Barre syndrome and Neuromyelitis optica.

Interferons: The main effect of interferons is the regulation of cell-mediated immunity as in treating Multiple Sclerosis.

Monoclonal antibodies: Introduction of new monoclonal antibodies has made treatments far more specific, helping us to target immune cells that have key roles in immunosuppressive disorders such as CD19 and CD20. These drugs act either by preventing sensitized cells from entering CNS like Natalizumab or directly inactivating these cells. In conclusion, these novel methods may pave the way for better and more effective treatments in the future.

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O23

Modulation of Pacemaker Channels and Rhythmic Thalamic Activity by Demyelination and Inflammatory Cytokines

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The thalamus is a central element for the generation of rhythmic oscillatory activity under physiological and pathophysiological conditions. Especially slow oscillations in the delta and theta frequency band which normally occur during slow-wave sleep are associated with a number of neuropsychiatric conditions if they occur during wakefulness and may be the basis for the generation of characteristic symptoms. This type of slow rhythmic activity requires sustained membrane hyperpolarization and the cyclic interaction between the pacemaker current, Ih, and the T-type Ca2+ current, IT, on the cellular level. Only recently a critical role of the thalamus in neuroinflammatory diseases like Multiple sclerosis (MS) has been appreciated. However it is unclear how Ih and oscillatory network activity in the thalamocortical system are influenced by MS-related pathologies like demyelination and increases in cytokines. Here we found that general demyelination and pro-inflammatory cytokines differentially modulated the voltage-dependency of Ih in thalamocortical relay neurons and that the availability of this current was an essential parameter for determining the parameters (frequency, number of bursts) of rhythmic thalamocortical activity which may explain some aspects of MS pathology.

O24

Functional Role of the K2P Potassium Channel TASK-3 in Glioma

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TASK-3, a two-pore-domain (K2P) potassium channel, has been implicated as important regulator for the effector function and proliferation of T-cells. Interestingly, TASK-3 has also a functional impact on tumor cells. Therefore, we sought to investigate whether TASK3 modulation might have a therapeutic potential for malignant gliomas by a variety of phenotypical and functional in vitro assays mimicking tumor microenvironment such as hypoxia and an in vivo mouse model for malignant glioma. GL261 glioma cells demonstrated higher proliferation rates under hypoxia, while proliferation and viability of WT (wildtype) T-cells was significantly reduced. Of note, TASK3-/- T-cells were more resistant to hypoxia-induced anergy and cell death indicating a potential advantage in immune-mediated tumor defense. In accordance, TASK-3-/- mice demonstrated a longer symptom-free survival in the GL261 malignant glioma model compared to WT mice (31 versus 23 days). To dissect an immune-cell mediated effect, we performed an adoptive transfer of splenocytes in RAG-/- mice, which do not contain mature B- or T-cells. Transferring TASK-3-/- splenocytes, we observed prolonged symptom-free survival (21 days) compared to WT splenocytes (17 days). Therefore, our results indicate a role of TASK3 in immune-cell mediated tumor defense mechanisms providing first evidence for a new therapeutic target in glioma therapy.

O25

Treatment of Autoimmune Epilepsy

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): O25

Seizure and epilepsy can be related to autoimmune diseases and processes. Accurate diagnosis is essential for better treatment of these conditions. Different clinical studies have been conducted to diagnose and treat autoimmune epilepsy. Rapid treatment of these diseases helps to Improve prognosis and less relapses. Conventional epilepsy treatments including antiepileptic drugs are less successful in these cases. Early firs t line immunotherapy (corticosteroids, intravenous immunoglobulin, plasma exchange) provides fair response to autoimmune encephalitides but half or more patients require a second-line immunotherapy. (Rituximab, Cyclophosphamide). A significant percentage of patients are resistant to first and second line immunotherapy and require further treatment.

O26

Targeted Delivery of siRNA in a Nano-Particle Suppress Glioblastoma Stem Cells

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Cancer stem cells (CSCs) are suggested as the most dominant causes of recurrence due to their permanent self-renewal and resistance to common cancer treatment in glioblastoma multiform (GBM) which is recognized as the most malignant of brain tumor. It has been indicated that Retinoblastoma-binding protein 5 (RBBP5), a main part of Mixed lineage leukemia protein-1 (MLL1), plays a significant role in cancer stem cell survival. In this s tudy the viability of CSCs derived from human GBM will be evaluated by knocking down the RBBP5 via their SiRNA. To enhance passing the SiRNA thorough the blood-brain barrier, PLGA nanocarrier will be used. CSCs isolated from Human GBM and cultured. To target specific stem cells involved in the growth and spread of cancer cells, CD133+ as a CSCs antigen will be conjugated on the surface of PLGA and then conjugated to SiRNA. The viability, proliferation, apoptosis, and differentiation of CSCs will be performed as a primary outcome. The expression of self-renewal markers such as NANOG, SOX2, CD133+, and Ki67 will be assessed as a secondary outcome. The probable prediction is descending the population of CSCs in the tumors that supposed to recurrence and short life expectancy. In conclusion, the nano drug can decrease the number of CSCs and increase the survival time in the GBM patients.

O27

Interaction of Cancer Stem Cells and Microglia in Glioblastoma Multiforme

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Malignant gliomas are highly invasive brain tumors with the occurrence of multiple microglia/macrophages in the tumor microenvironment. Macrophages/microglia that found in glioma microenvironment, as tumor-infiltrating immune cells, can play a harmful role in tumor progression. In addition, glioblastoma multiforme (GBM) contains multiple aberrant differentiation and tumorigenic cancer stem cells (CSCs) that contribute to tumor heterogeneity and resistance to anti-cancer therapies. The present study was aimed to understand the interaction between microglial cells and CSCs in a co-culture system. Specific markers used for the characterization of CSCs and microglia in GBM tissues obtained from patients. Then, we applied a co-culture system consisting of permeable membrane allowing secreted soluble factors to diffuse. Measuring the effects of cytokines secreted by activated and non-activated microglia on CSCs, MTS cell proliferation assay were performed. Cell viability in CSCs treated with non-activated microglia was significantly reduced compared to the group that treated with activated microglia. The activated microglia/macrophages may interfere in the process of tumor angiogenesis, metastasis niches, recurrence and support tumour invasiveness.

Workshop Presentations

W1

Somatoform Pain Disorders Fibromyalgia is a Mental or Somatic Disease?

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): W1

Mental disorders with pain as a leading symptom are remnat diagnostic categories for physicians and psychotherapist, even if they are educated in pain treatment. Patients with somatoform pain disorder (ICD-10: F45.4) are often diagnosed only after several years and multiple diagnostic procedures, in some cases after iatrogenic impairment. The clinical practical features, diagnostic procedure and differential diagnosis in somatoform pain patients as well as current psychotherapeutic approaches are outlined.

W2

"Discogenic Pain and the Way to Treatment"

Siamak Radmehr

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): W2

The exact differentiation of the back pain and its backgrounds made the base of much research. Various triggers also have different pathomechanism. This background is used after accurate detection in the treatment of back pain. Discogenic pain is a particular challenge in pathobiochemistry and therapy.

W3

Intra-Operative Imaging and Brain Pathology

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The Intraoperative MRI is of great importance in the surgical removal of gliomas or cranial base tumors. However, the sole use of a intraoperative MRI is problematic, so that a dual system has been developed to optimize the utilization of the equipment, which can be used both for neurosurgical patients and for outpatient or stationary Patients. Within the framework of the new head Center at the Asklepios Clinic Nord/Heidberg, in Hamburg/ Germany a 1.5 T-MRT was connected directly to the neurosurgical operation-tract in 2011, which is provided with two separate entrances. The treatment of the MRI room, such as air purification, is carried out according to the same standards that apply to neurosurgical operation units. The dual system concept is suitable for intraoperative, outpatient or stationary MRI investigations and does not lead to an increased risk of infection or treatment. Moreover, this concept also makes sense from the economic side.

W4

Cancer Stem Cells as a New Target Point for Treatment of Glioblastoma

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): W4

Glioblastoma is a destructive form of brain tumor that kills most patients within two years of diagnosis. Treatments for glioblastoma include the usual options of surgery, radiation therapy and chemotherapy, but there is no effective treatment. The tumors are capable of spreading tendrils out into the brain and it can grow back in a matter of months after being removed. The cancer stem cell (CSC) provides an alternative model to explain the tumor cell heterogeneity. CSCs are thought to be the less differentiated populations in malignant tissues and are considered to be the cells that are responsible for the maintenance of tumor tissues, as well as for the relapse of tumors after conventional treatment. This review provided the confirmation for the presence of CSCs in primary tumors. The CSC hypothesis raises the expectation that targeting of CSCs in tumors will lead to an improved clinical outcome because they are thought to be the "root" of growing tumors.

Poster Presentations

P1

Dextran Curcumin Promotes Novel Object Recognition Memory in Rats after Ischemic Stroke

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Ischemic stroke causes the depletion of energy and induces excitotoxicity and neuroinflammation in the brain that results from thrombotic blockage. Cerebral ischemia leads to many types of memory loss, including impairment of working, spatial and object recognition memoreis. Curcumin shows strong anti-oxidoinflammatory activities but it terapathics limited by its low solubility in water and corresponding poor intestinal absorption. So, in this study curcumin used in conjugate with dextran as polymeric carriers in novel drug delivery system. The purpose of this study was to determine the effect of dextran-curcumin on memory impairment induced by global ischemia. In this study 35 rats individed 5 groups. Pre-treatment and positive control groups, were treated with curcumin and dextran- curcumin (15mg/kg - orally) for 30 days and the vehicle and disease groups received distillated water. For induction of ischemic stroke model, rat were anaesthetized and both right and left carotid arteries were selected and clamped for 5 min by vascular clamps (time of ischemia), There after the vascular clamps were removed for the next 10 min (time of reperfusion), and both carotid arteries were clamped again for 5 min. Finally, the vascular clamps were removed and blood circulation was return in both carotid arteries, 48 hours after induction of model, Novel Object Recognition test was used to determine memory impairment in all rats. Our study indicated that memory impairment increace in ischemic groupe and dextran curcumin has memory-improving effects after global ischemic stroke ($p< 0.01$): Dextran-curcumin has memory-improving capacity better than curcumin in lower does.

P2

The Role of Neuronal Nitric Oxide Synthase on the Anti-Seizure Effects of 5-HT1A Receptors in Perforant Pathway Kindling Model in Rat

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Neuronal nitric oxide synthase (nNOS) plays a role in synaptic potentiation and kindling process.The relationship between nNOS and 5-HT1A receptors also nearly has been specified. In this research,we inves tigate the role of nNOS on the anticonvulsant effect of 5-HT1A receptors. 24 male (280 ± 30 g) were randomly assigned to four groups (vehicle,NI,Way 100635 and NI + Way100635) (n = 6). Animals received one of the above compounds 30 min before application of the kindling stimulus. In the NI + Way100635 group, 10 min later Intra ventricle injection Way100635, intraperitoneal nitroindazole (NI) was injected. After application daily stimulation (12 times a day, with a 5 minute interval), up to five days, the seizure and electrophysiological quantities (after discharge duration and local field potentials) were recorded and measured. Data analysis showed that the Way100635 and NI+Way groups were kindled significantly faster than the vehicle group (P <0.001).The changes in afterdischarge duration increased in the NI + Way group over the five days when compared to the control group, but was not significant (P> 0.05). Also, the slope of field potentials in the NI + Way group was significantly higher than that of the vehicle group (P <0.05). Likely, nNOS is one of the mediators of the inhibitory effect of serotonin 5-HT1A receptors, and activating this pathway augments the anticonvulsant effects of serotonin.

P3

Investigation of the Relationship between Uncertainty in the Disease with the Frequency of Hospitalization in Patients with Multiple Sclerosis who Referred to the Zanjan MS Society Sclerosis

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Multiple sclerosis (MS) affects all aspects of a person's life. Considering the unpredictable progress of MS, Patients suffer from uncertainty about their future health and wellbeing which causes psychological dis tress and social degradation and consequently it affects the development of the disease. This study aims to investigate the relationship between uncertainty in the disease with the frequency of hospitalization in patients with multiple sclerosis who referred to the Zanjan MS Society. This descriptive-correlation research was conducted in Zanjan MS Society in 1395. A total of 100 patients with MS were selected randomly without placement. The data gathering tool that is used is a demographic questionnaire and Mishel Uncertainty in Illness Scale (MUIS-C) questionnaire. Data were analyzed by SPSS version 16 at a significant level less than 0.05. The results have shown that in this research there was a significant positive correlation between uncertainty and frequency of recurrence (r = 0.387 p = 0.002) and hospitalization (r = 0.261 p = 0.013) during 1 year and people with more uncertainty experienced more recurrence and hospitalization. This study shows that uncertainty in the disease affects the frequency of relapse and admission of MS patients. It seems that finding a way to control the amount of uncertainty in the disease can control the tensions caused by uncertainty in the disease and, consequently, the progression of the disease.

P4

Assesse the Health Literacy in Multiple Sclerosis Patients

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): P4

Health literacy is one of the major determinants of health and is one of the important factors in chronic diseases such as Multiple sclerosis (MS) is an inflammatory disease in which the myelin sheaths of the neural cells are damaged in the brain and spinal cord. The aim of this s tudy was to evaluate the Health Literacy Association with Health Behaviors and Health Care Utilization. This study was a cross-sectional study in which 100 patients with multiple sclerosis disease. Selected by the convenience sampling in Qazvin hospital. Respondents completed the Rapid Estimate of Adult Literacy in Medicine, revised (REALM-R) and the Newest Vital Sign (NVS) instrument. For statistical analysis We used logistic and ordinal regression and kruskal-wallis and mann-witney and 2 test. Mean age of the subjects was 33/43±8/53 years. On the NVS, 63% of respondents had a high likelihood of inadequate literacy and 26% had borderline health literacy and 11% had adequate health literacy. On the Realm-r, 7% had inadequate literacy and 93% had adequate health literacy, Using logistic regression the frequency of alcohol consumption and using cigarret (p<0/05) and level of education (p-value=0/045) were associated with higher odds of having inadequately health literacy as assessed by the Realm-r. There was a significant correlation between Realm-r health literacy (p value=0/007) and NVS (p-value=0/005) with visits to the emergency room (ER), and hospitalizations in the las t 6 months. Based on the results of the current study it is necessary to pay more attention to the health literacy level of the affected people and promote it in health programs and health promotion.

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P5

Comparing the Effectiveness of Selected Tai Chi Exercises and Core Stabilization Exercises on Balance and Quality of Life in Parkinson Patients

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Parkinson's disease is one of the most common central nervous system damaging diseases which mainly affects the elderly. The variety of disorders and complications associated with the disease puts the patients in so much trouble. Some of these problems include disorder in balance, walking and Decrease in quality of life. The purpose of this study was Comparing the effectiveness of selected tai chi exercises and core stabilization exercises on balance and quality of life in Parkinson patients. In this study, 36 Parkinson patients were purposefully selected and were divided into three groups; Tai Chi, core stabilization exercises and control group. Biodex balance system meter and Quality of Life Questionnaire were used to measure the patients' balance and Quality of Life. The two experimental groups underwent Tai Chi and core stabilization exercises for 8 weeks. MANCOVA was used to analyze the results. Statistical results showed that there was a significant difference between balance in the control group with tai chi and core stability groups (P<0/05). There was no significant difference between the two experimental groups (P>0/05). This means that the effect of these exercises in improving balance is equal. Because of Improving Balance and Quality of Life, we recommend tai chi and core stabilization exercises as a complementary treatment alongside medicinal treatments for Parkinson patients.

P6

Relationship Among Dimensions of Roy Adaptation Model, General Health and Satisfaction with Life in Parkinson Disease Patients

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Parkinson disease (PD) is a chronic, progressive and debilitating disease. This study aimed to determine the relationship between the dimensions of Roy Adaptation Model (RAM) and General Health (GH) and Satisfaction with Life (SWL) in PD patients. This was a cross-sectional study. Samples were selected from the PD patients in 2016. The data was gathered by demographic, RAM, GH and SWL Scale questionnaires. In PD patients, total adaptation score was 21.26±3.24. The dimensions of adaptation, the scores of physiologic mode, self-concept, role function and interdependence were 74.7±9.33, 32.37±6.04, 24.30±5.00 and 20.93±2.46, respectively. There was significant relationship between education and physiologic mode (P=0.048) and there were significant relationships between PD experience, employment and gender and total adaptation score (P=0.002, P=0.007, P=0.006, respectively). Mean and standard deviation of total general health score was 30.97±5.03 and the means and standard deviations of its dimensions, including somatic symptoms, anxiety and insomnia, social dysfunction and depression were 10.26±2.71, 9.73±2.77, 5.13±2.23 and 5.83±1.91, respectively. There were significant relationships between social dysfunction and physiologic mode and self-concept (P=0.024 and P=0.012, respectively). The mean and standard deviation of satisfaction with life score was 21.47±3.57. Satisfaction with life had no relationship with dimensions of RAM and general health. RAM, GH and SWL make the development of high quality care planning possible by assessing the health status of PD patients. This leads to engagement of the patients in self-care, better adaptation, improved quality of life and also helps the nurses to make medical decisions.

P7

Dimensions of Adaptation, General Health, and Life Satisfaction in Multiple Sclerosis

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Multiple Sclerosis (MS) is a debilitating disease which can affect general health and life' satisfaction. This s tudy aimed to determine dimensions of adaptation, general health, and life satisfaction in MS patients. This study was a cross-sectional that samples were selected from MS patients in 2015. Data was collected by using a demographic questionnaire, Roy Adaptation Model (RAM), General Health Questionnaire (GHQ), and Satisfaction with Life Scale (SWLS) and analyzed by using of SPSS software. Results indicated the physiological and self-concept was associated with the history of MS, while the role function was associated with marital status. In addition, physiological dimension was associated with education level and occupation. Social functioning was associated with marital status. Somatic symptoms were associated with physiological dimension and self-concept. Anxiety and depression were associated with physiological dimension, self-concept, and role function. Social dysfunction was associated with self-concept and role function. Satisfaction with life did not have any significant relationship with demographic variables, RAM, and GHQ. Finding shown that RAM, GHQ, and SWLS can be used to evaluate the health status of MS patients and to design high quality care programs. Such programs can motivate patients to engage in self-care, show better adaptation, and improve their quality of life.

P8

Endocannabinoid System Mediate the Effects of Crocin on Development of Neuropathic Pain in a Rat Model of Chronic Constriction Injury

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Neuropathic pain involves injury or alteration of the normal sensory and modulatory nervous systems to produce a set of symptoms that are often difficult to treat. Previous study indicates that saffron has anti-inflammatory properties that may be mediate by neurotransmitter system. In this study we determine the role of cannabinoids receptors in peripheral and central effects of Crocin on behavior neuropathic pain responses in Chronic Constriction Injury (CCI) model in Rat. In this experimental study we used of adult male Wistar rats (220 to 250 g). CCI was induced by setting four loose ligatures around the siatic nerve. In part 1, after nerve lesion, injections of vehicle, Crocin (60 mg/kg) or Win21212 (0.1 mg/kg) as an agonist and AM251(0.1 mg/kg) as an antagonist of endocannabinoid receptors, were injected intraperitoneally in separate groups and continued every day for 2 weeks. In part 2, two weeks after of nerve lesion, injections of vehicle (0.5µl), Crocin (6 µg/0.5µl), Win21212 (0.1 µg/0.5µl), AM251(0.1 µg/0.5µl) were done in intracerebroventricular (ICV) in separate groups. Pain behavioral responses including mechanical allodynia (von Frey filament testing) and thermal hyperalgesia were measured at day 14. Data analyzed by Two-way ANOVA and tukey test. Results indicated that central or peripheral injection of Crocin decreased thermal hyperalgesia and mechanical allodynia. Also central or peripheral Co-administration of Win21212 or AM251 modulate of analgesic effect of Crocin significantly ($P<0.05$). Findings shown that Crocin have analgesic effects that probably mediated by endocannabinoid mechanism.

P9

Cervical Spinal Cord Extraction in Patients with Multiple Sclerosis Using Magnetic Resonance Imaging for Measuring Cross-Sectional Area

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Multiple sclerosis (MS) refers to the lesions that accumulate in the brain and spinal cord. Magnetic resonance imaging (MRI) is the most sensitive and versatile modality used to show changes in the tissues over time. There has been significant interest in evaluating the relationship between the brain atrophy and disease progression rather than the spinal cord atrophy. The cervical spinal cord has an important effect on the disease progression. Regarding the literature, spinal cord abnormalities in MS patients are more common in the cervical segments in comparison to the thoracic and lumbar regions. This study was conducted extract the spinal cord in MS patients from MRI images in the Department of Neurology, Ghaem Hospital, Mashhad University of Medical Sciences, Mashhad, Iran. In this study, 51 MRI images were obtained from 49 individuals, out of whom 28 were healthy (21 females and 7 males) and 23 had MS (17 females and 6 males). The participants aged between 21 and 45. The MRI images were normalized using an automatic image analysis method. Thereafter, the desired candidate areas were determined using data preprocessing, edging, and edge detection techniques. Appropriate areas were recognized by applying appropriate conditions and using prior knowledge about spinal cord and vertebrae.

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By measuring specific parameters that was conducted under the supervision of a consultant, as an input to a well-suited and well-used artificial neural network, it was decided if the person had MS or not. According to the Dyce index, 72% of the regions were accurately selected by the automated method presented in the image segmentation. In addition, we diagnosed MS by measuring the FP, FN, TP, and TN values in artificial neural network outputs with a precision of over 70%. With the help of the proposed method, we tried to find a simple and effective method for auto-segmentation of cervical MRI images with a specific purpose for parsing, measuring the parameters, and then diagnosing MS in patients. The method presented will be of great help to the physician regarding future decision making, and it is hoped that this method and its results, with improvement and implementation, will be used as a technique and accessible to all individuals related to this issue, especially physicians.

P10

Isolation and Culture of Primary Microglial Cells from Glioblastoma Patients

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Glioblastoma multiform (GBM) is the most common and malignant form of glial tumors. GBM microenvironment contains various cell types showing characteristics of activated or dimorphic macrophages/ microglia. Some of these cells provide significant help for tumor growth, while others are able to inhibit tumor progression. Microglia play a major role in brain function by monitoring tissue for pathogen via phagocytic activities. Following surgical resection, human tissue samples were transfered to the research facility. Then, mechanical and chemical dissociation and enzymatic digestion were performed. Cell pellets were resuspended in media. When reaching complete confluency, mixed glial cultures were shaked to remove astrocytes. Finally immunocytochemistry was performed on remaining cells for characterization. The cells generated from GBM surgeries were likely a mix of microglia and macrophages. A small amount of astrocytes were also presentin the culture. After confirming cell phenotype, a more detailed immunocytochemical analysis was performed. Isolated microglia express Iba1 marker. Microglia obtained from GBM can be utilise for in vitro and in vivo investigation.

P11

Assess the Electrophysiological Activity of Olfactory Bulb in the Animal Model of PTSD and its Relationship with Neuroinflammation in the Olfactory Bulb

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Post-traumatic stress disorder (PTSD) is a mental health condition that's triggered by a terrifying event - either experiencing it or witnessing it. In addition to the relationship between PTSD and neuroinflammation, research indicates that olfactory bulb are effective in anxiety disorders. The aim of this study was to assess the electrophysiological activity of olfactory bulb in the animal model of PTSD and its relationship with neuroinflammation in its area. 36 rats were divided into two groups: control and experiment. The modified Zoladz method was used to establish a PTSD animal model. Then, behavioral tests (open field (OF) and elevated plus maze (EPM)), electrophysiological evaluation (field potential) of olfactory bulb, and assessment of serum cortisol (as an inflammatory marker) were done. It was followed by dissection of the rat's brains, which could be apply for analysis of neurogenesis and inflammatory factors. In OF test, the time spent in the central area and in EPM test, the time spent in the open arm by the animals of experiment group were lower than that of control group ($P<0.05$- $P<0.01$). Moreover, experiment group significantly increased slope, slope 10-90%, amplitude of fEPSP, and also cortisol level compared to control group ($P<0.001$). Based on our findings, it is concluded that can be a relationship between PTSD and

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olfactory bulb function. Of course, more molecular inves tigations have been conducted in order to determine the effect of PTSD on the activity of Mitral and Tufted cells, and its association with neurogenesis and inflammation in olfactory bulb, which helps to identify the role of olfactory bulb in PTSD disorder.

P12

Protective Effects of Nigella Sativa on Synaptic Plasticity Impairment Induced by Lipopolysaccharide

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In the present study the protective effect of Nigella sativa (N. sativa) on synaptic plasticity impairment induced by lipopolysaccharide (LPS) in rats was investigated. Fifty-eight rats were grouped and treated as follows: 1) control (saline), 2) LPS, 3) LPS-N. sativa, and 4) N. sativa. In a Morris water maze test, the escape latency and travelled path to find the platform as well as time spent and the travelled distance in target quadrant (Q1) were measured. Long term potentiation (LTP) from CA1 area of hippocampus followed by high frequency stimulation to Schafer collateral was studied and slope, slope 10-90% and amplitude of field excitatory field potential (fEPSP) were calculated. The escape latency and traveled path in LPS group were significantly higher than those in the control group while, in LPS-N. sativa group these parameters were significantly lower than those in LPS group. The rats in LPS group spent less time and travelled shorter distance in Q1 than the rats in the control group while, in LPS-N. sativa group the rats spent more time and travelled longer distance than the rats in LPS group. LPS significantly decreased slope, slope 10-90% and amplitude of fEPSP while, in LPS-N. sativa group these parameters increased compared to LPS group. The results indicated that the hydro-alcohol extract of N. sativa protected against synaptic plasticity and spatial learning and memory impairment induced by LPS in rats.

P13

Anti-Inflammatory Effect of Glycyrrhizin on TNF-α Produced by Inflamed Microglia Cell (BV-2)

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Glycyrrhiza glabra is a plant used in traditional medicine across the world for its pharmacological value. Its value has been proved such as anti-cough, anti-diabetes, anti-microbial and antioxidant effects. The roots of plant comrised glycyrrhizin which is 50 times sweeter than sugar. Macrophages can stimulated during inflammatory disorders, and with production of multiple inflammatory mediators, they can produce immunogenic effects such as tumor necrosis factor α, but in some cases cause clinical problems and symptoms of infectious and inflammatory diseases. the aim of the present study was to investigate the glycyrrhizin component of the licorice on the level of TNF-α produced as an inflammatory mediator in the cellular model (in vitro). This study was designed through 5 groups (each group has Three times repeatedly). In group 1, BV-2 cells stimulated by lipopolysaccharide, in groups 2, 3, 4 and 5, inflamed cells received 0.2, 0.4, 1.2 and 4 µg/ml glycyrrhizin, respectively. In present study, glycyrrhizin with 0.2 µg/ml concentration had no effect on TNF-α level of inflamed cell. But others groups (0.4, 1.2 and 4 µg/ml) inhibited production of TNF-α in BV-2 cells, rather than first group. We can conclude that licorice is a potential source of natural anti-inflammatory agent. Some studies show that glycyrrhizin have anti-inflammatory effect on microglia cell through reducing other inflammatory factors; also in present study, the inhibitory effect of glycyrrhizin on inflamed cells was confirmed. Although at this time, it still needs further researches for evaluating its pharmaceutical potentialities of its anti-inflammatory mechanisms.

P14

Segmentation Brain Tumors of FMRI Images by Gabor Wavelet Transform and Fuzzy Clustering

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Today, high mortality rates due to brain tumors require

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early diagnosis in the early stages to treat and reduce mortality. Therefore, the use of automatic methods will be very useful for accurate examination of tumors. In recent years, the use of FMRI images has been considered for clarity and high quality for the diagnosis of tumor and the exact location of the tumor. In this study, a completely automated method for the partitioning of T1, T2, and MR images of tumors is presented using T1 and T2 gamma-ray wavelets, which has acceptable results in the presence of data noise. After applying the SWT wavelet, the Gabor filters are applied to the wavelet approximation at all levels; the texture features such as entropy, second-to-fourth-moment moments, and coefficient of variation are obtained. The outputs of this filter are compared with each other and have a maximum localization in the size of these vectors. And finally these images are referred to as pixel-specific attributes, and to obtain an image with a minimum pixel value, it is given to the Fuzzy clustering algorithm, which generates the finalized output. This algorithm appears. It takes the attributes of the corresponding level and generates an optimal segmentation. Goal This design reduces the error in diagnosing a benign tumor from malignant FMRI in medical science.

P15

Mephedrone Exposure in Pegnancy Iinduces Antiprolifrative and Proapoptotic Effects in Hippocampus of Mice Delivered Pups

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In recent years, abuse of synthetic cathinones, in particular, mephedrone, has increased among young adults worldwide. The study aim is to investigate the effects of mephedrone exposure during the gestational period on mice offspring outcomes, focusing on hippocampal neurotoxicity. The pregnant mice received mephedrone (50mg/kg, sc) on a regular schedule (once daily on all days, from day 5 to 18 of gestation) or repeated schedule (thrice daily on day 5, 6, 11, 12, 17, and 18 of gestation) to simulate regular or recreational use of mephedrone, respectively. Immunohistochemistry and TUNEL assay showed an inhibition of cell proliferation ($p<0.05$) and an increase of apoptosis ($p<0.05$) in the hippocampus of delivered pups of the repeated schedule mephedrone group. In conclusion, the present study has shown that repeated use of mephedrone impairs learning and memory processes through hippocampal damage.

P16

Effect of Hesperetin Nanoparticles on Cerebral Gene Expression and Activity of Catalase and Superoxide Dismutase in Alzheimer's Rat

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Hesperetin (Hst) is a well-known bioflavonoid, has low bioavailability. Hesperetin nanoparticles (Nano-Hst) enhance its bioavailability. Nano-Hst were not explored for their potential therapeutic activities in Alzheimer's disease (AD). Hence, the present study was performed to evaluate the protective effect of Nano-Hst in comparison to free Hesperetin on against intracerebroventricular injection of streptozotocin (icv-STZ) induced cerebral gene expression and activity antioxidant enzymes impairments in rat model. Nano-Hst prepared by evaporative precipitation of nanosuspension (EPN) method. The rats were divided into six groups including: Control (received water orally and icv-saline), disease group (received 3 mg/kg/rat icv-STZ) and treated groups received Hst and Nano-Hst (10, 20 mg/kg/d) for 3 weeks after icv-STZ. Activity and gene expression of catalase (CAT) and superoxide dismutase (SOD) were measured in the cerebral cortex. Our result showed that the rat model of AD decreased activity and gene expression of CAT and SOD ($p < 0.001$). Hst and nano-Hst treatment elevated the activities and gene expression of these enzymes ($p < 0.01$). Gene expression studies of antioxidant enzymes using Real Time PCR confirmed the enzymes activity. These results indicate that Nano-Hst was more effective than Hst to attenuation oxidative stress induced by STZ in a rat model of Alzheimer's disease.

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P17

Maternal Administration of Nano-Hesperetin Prevents Iincrease of Kidney's Enzymes and Reduction of Stress-Oxidative in Rat Model of Autism

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Autism spectrum disorders are severe neurodevelopmental disorders, marked by impairments in reciprocal social interaction, delays in early language and communication and the presence of restrictive, repetitive and stereotyped behaviors. Depend on the s tatistic, 1child from each 68 children face with ASD. This statistic shows that probably, the environmental factors play the important role in ASD. Based on this, the aim of this research is to investigate the relation of maternal-offspring in ASD during pregnancy and lactation. In the experimental research, pregnant rats were divided into six groups including: Sham (received water and saline orally), disease group (injected 500 mg/kg valproic acid at gestational day 13) and treatment groups (received 10 and 20mg/kg/day/nano-Hesperetin). Treatment groups received nanocrystal orally for 7 weeks during pregnancy and lactation. The relation of maternal and offspring in ASD was estimated by checking brain anti-oxidant (CAT, SOD, GPx) and kidney's enzymes (AST, ALT, ALP). The results showed that probably injection of valproic acid increases kidney's enzymes ($p \leq 0.001$) and decrease anti-oxidant enzymes ($p \leq 0.001$) in maternal and offsprings compared with control group and treatment of nano-Hst effectively decrease kidney's enzymes (10 and 20mg/kg; $p<0.001$) and increase anti-oxidant enzymes ($p<0.001$) compared with disease group in maternal and offspring. The results of this research showed that probably there relation of maternal and offspring related to ASD and oral administration of nano-Hst prevents reduction of anti-oxidant and increase of kidney's enzymes in valproic acid model of autism-like.

P18

Neuroprotective Effect of Safranal, an Active Ingredient of Crocus Sativus, in a Rat Model of Transient Cerebral Ischemia

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Safranal is a monoterpene aldehyde found in saffron (Crocus sativus L.) petals. It has been previously reported that safranal has a wide range of activities such as antioxidant and anti-inflammatory effects. In this study, we examined the effect of safranal on brain injuries in a transient model of focal cerebral ischemia. Transient focal cerebral ischemia was induced by middle cerebral artery occlusion for 30 min, followed by 24 h of reperfusion. Safranal in the doses of 72.5 and 145 mg/kg was administered intraperitoneally at 0, 3, and 6 h after reperfusion. Neurobehavioral deficit, infarct volume, hippocampal cell loss and markers of oxidative stress including thiobarbituric acid reactive substances (TBARS), total sulfhydryl (SH) content, and antioxidant capacity (using FRAP assay) were also assessed. The focal cerebral ischemia induced a significant increase in the neurological score, infarct volume and neuronal cell loss in the ipsilateral hippocampal CA1 and CA3 subfields ($p < 0.001$) and also oxidative stress markers ($p < 0.01$). Following safranal administration, the total SH content and antioxidant capacity significantly increased, while marked decreases were observed in the neurological score, infarct volume and hippocampal cell loss, as well as TBARS level. This study concluded that safranal had protective effects on ischemic reperfusion injury in the rat model of stroke. Such effects of safranal may have been exerted mainly by suppressing the production of free radicals and increasing antioxidant activity.

P19

The Synergistic Effects of Mixture Extract Portulaca Olerace, Urtica Dioica, Boswellia Serrate on Multiple Sclerosis in Rats

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Multiple Sclerosis (MS) is an inflammatory disease in CNS. One of prevalent symptoms in MS is memory disorders. Main hippocampus function in brain are memory. Nowadays tendency of herbal therapy is increase because of drug's side effects. This study's purpose is review of synergistic effects of mixture extract Portulaca olerace, Urtica dioica, Boswellia

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serrate on multiple sclerosis in rats. This study did on 30 head of male rats with 3 month age and 250-300 weigth that randomly divided into five groups (n=6) including control group, sham group (salin injection), (MS+salin) group, (MS+mixture extract (dose 200 mg/kg)), (MS+mixture extract (dose 400 mg/kg)). MS model was induced by intra hippocampal injection of ethidium bromide (stereotaxic surgery) into single dose (0.01% ethidium bromide sulotion in 0.9% salin) and in 3 microlitre volume with 1 microlitre in minute rate intraperitoneally and mixture extract injected as the treatment for 21 days. The shuttle box test did for memory study and from histopathology and dissector methods used for study on neural amelioration. mixture extract causes neurogenesis and memory's amilioration in two treatment groups in comparison with (MS+salin) group, also neural density in treatment group neared to control group after mixture extract injection. mixture extract because of neurogenesis and amilioration effect can effective in memory recovery and neural necrosis in MS disease.

P20

The Effect of Hydro- Alcoholic Extract of Caralluma Tuberculata on Acute and Chronic Pain in Male Rat

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Neuropathic pain, caused by a lesion or disease affecting the somatosensory nervous system, has a substantial effect on quality of patient life. Given that the synthetic drugs can produce serious side effects they are not suitable for long-term use, therefore it is assumed that herbal medicine can be effective as an alternative to Analgesic agent. The aim of this study was evaluation the analgesic effects of hydroalcholic extract of Caralluma tuberculata (Ct) in male rat. 42 Male Wis tar rats, weighing 200±20 g, were divided into 7 groups (n=6); Groups included: control, sham-treatd Ct (75, 125, 250mg/kg), and positive control groups, respectively diclofenac (5mg/kg) and morphine (2.5mg/kg) and the group receiving (125mg/kg) extract + naloxone (1mg/kg). One hour later Pain was induced by applying 50 µL of 2.5% formalin in distilled water in the subplantar of the right hind paw. Our findings revealed that the Ct treatment can significantly decrease formalin-induced pain in rat. Ct (125, 250 mg/kg) significantly inhibit the aute phase ($P < 0.001$), whereas, the all concentration of Ct were affected on the chronic phase of formalin-induced pain ($P < 0.001$). Our data suggest that the sys temic and peripheral antinociception activities of Ct probably were mediated through the opioid receptors in the periphery and also in the central nervous system.

P21

Local Administration of Methylprednisolone Laden Hydrogel Enhances Functional Recovery of Transected Sciatic Nerve in Rat

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The repair of peripheral nerve injuries is still one of the most challenging tasks and concerns in neurosurgery. Effect of methylprednisolone-laden hydrogel loaded into a chitosan conduit on the functional recovery of peripheral nerve using a rat sciatic nerve regeneration model was assessed. A 10-mm sciatic nerve defect was bridged using a chitosan conduit (CHIT/CGP-Hydrogel) filled with CGP-hydrogel. In authograft group (AUTO) a segment of sciatic nerve was transected and reimplanted reversely. In methylprednisolone treated group (CHIT/MP) the conduit was filled with methylprednisolone-laden CGP-hydrogel. The regenerated fibers were s tudied within 16 weeks after surgery. The behavioral, functional and electrophysiological studies confirmed faster recovery of the regenerated axons in methylprednisolone treated group compared to CHIT/Hydrogel group ($P <0.05$). The mean ratios of gas trocnemius muscles weight were measured. There was statistically significant difference between the muscle weight ratios of CHIT/MP and CHIT/Hydrogel groups ($P<0.05$). Morphometric indices of regenerated fibers showed number and diameter of the myelinated fibers were significantly higher in CHIT/MP than in CHIT/Hydrogel group. Methylprednisolone-laden hydrogel when loaded in a chitosan conduit resulted in improvement of functional recovery and quantitative morphometric indices of sciatic nerve. It may have clinical implications for the surgical management of patients after facial nerve transection injuries.

P22

Studying Antialgesic Effect of Chamomile, Fennel and Saffron, Herbal Compound in Mail Rats

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Some plants have beneficial effects on diseases. In ancient times they were be used as a drug. According to our herbal medicine history we combained chamomile which decrease the pain and healing scares,with fennel, a plant as a antiinflammator and saffron,a plant which is using for treating insomnia and as a antidepressor . Firs t we choosed 15 matured,healthy and male rats that they weigh 200 grams approximately. Then we devide them into three groups.The control group which didn't get any injection, the control positive group which we injected them as regular as the experiment group but its content was just water and the experiment group that were injected regularly and with specific dose. We continued these injections for one week.After one week we tes ted their rate of anxiety by elevated plus maze(EPM). After that we processed our datas by SPSS software. After comparing the datas with each other,we observed that there was significant reducton in the anxiety of our experiment group. It seems that,these reduction is because of regular injecting and specific dose in each time.

P23

The Effects of Captopril, as Angiotensin Converting Enzyme Inhibitor, on LPS-Induced Systemic Inflammation

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It has been shown that the renin-angiotensin system (RAS) plays key roles in the inflammation process. Imbalance in the oxidant-antioxidant system is one of the major causes of inflammation. In the present study, the effects of captopril on total and differential WBC, oxidative stress in systemic inflammation produced by lipopolysaccharide (LPS) were investigated. The rats were divided to: control (saline), LPS (1 mg/kg), 12.5, 25 or 50 mg/kg captopril treated before LPS adminis tration (LPS-Cap12.5 , LPS-Cap 25 and LPS-Cap 50) and captopril 50 mg/kg before saline administration (positive control group) groups. The levels of total and percentage of differential WBC in blood, the levels of malondialdehyde (MDA), total thiol groups, the activities of superoxide dismutase (SOD) and catalase (CAT) in the serum were evaluated. In the LPS group, total WBCs count, percent of neutrophils, basophils, eosinophils, monocytes in blood and MDA levels in serum were significantly higher than the control group ($p<0.05$ to $p<0.001$). Total WBCs count and percentage of eosinophils in the blood of LPS-Cap25 and LPS-Cap50 groups, percentage of neutrophils, monocytes, basophils in the blood and MDA levels in serum of LPS-Cap50 group were significantly decreased compared to the LPS group. Total thiol groups, activity of SOD and CAT enzymes, percentage of lymphocytes in the LPS-Cap50 group were significantly increased compared to LPS group. ($p<0.05$ to $p<0.001$). The results of this s tudy showed that captopril dose dependently reduced total and percentage of differential white blood cells in systemic inflammation induced by LPS in rats and also improved inflammatory responses and oxidative stress.

P24

Clarifying the Relationship between EEG Power and Neuroticism

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The aim of this study was to clarify the relationship between EEG power and neuroticism trait. We used correlational method in order to examine the hypotheses. The participants included twenty-five undergraduate students (age mean= 21.36, SD= 23.39) at Ferdowsi university of Mashhad that were selected as volunteers. All participants were right-handed and had normal or corrected-to-normal visual acuity. We used the self-report version of the Revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992) to measure neuroticism factor. In order to record brain activity, Mitsar EEG-201 (Mitsar Co. Ltd. Saint Petersburg, Russia) was used. As regard to Stepwise regression's results, parietal in alpha band could predict 40 percent of neuroticism ($P<0.05$, $F=15.77$). Also, parietal in alpha band and frontal in delta band could predict 22 percent of neuroticism ($P<0.05$, $F=12.97$). Additionally, parietal and frontal in alpha band and frontal in delta band could predict 7 percent of neuroticism ($P<0.05$, $F=4.95$). Finally, parietal, frontal in alpha and delta bands could significantly predict 10 percent of neuroticism ($P<0.05$, $F=9.98$). To sum up, by considering EEG measuring, neuroticism is associated with reward motivation and punishment, anxiety, depression, behavioral inhibition, brains tem activity and thalamacortical system. In fact, our findings could not completely support previous studies, because there were inconsistent and consistent findings as regard to neuroticism.

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P25

The Effect of Toad Skin Extract on Alzheimer's Disease – Lnduced Depression Based on Behavioral Test in rRats

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Alzheimer's disease is the most common cause of dementia with associated symptoms such as depression, anxiety and psychosis. Increased expression of inflammatory mediators in postmortem brains of people with AD has been reported, and epidemiological studies link the use of anti-inflammatory drugs with reduced risk for the disorder. Present studies have shown that toad medicines decrease inflammation through a variety of mechanisms, including inhibition of NFB and its signaling molecules and pathways. The aim of this s tudy was to evaluate the efficacy of toad skin secretion on recovery from stress and depression caused by AD based on the behavioral tests. 50 rats were divided into 5 groups; 1) control, 2) Alzheimer's recipient of beta-amyloid (1-42) into cerebral ventricular injection of 2 µl, 3-5) Alzheimer recipient toad skin secretion respectively by 20, 40 and 80 ml/kg in 6 times during 20 days; respectively. After this period, the behavioral tes ts (forced swimming test, open field test, elevated plus maze) was used to assess stress and depression. I.C.V infusion of Alzheimer's beta amyloid was increased immobility time in samples. Results showed a significant reduction duration of immobilization in the dose of 20 about ($p<0.01$) and at doses of 40 and 80 ml/kg approximately ($p< 0.001$), respectively. The open field test's result indicate an increase in the number of homes passed were dose-dependent increase in dose level 80 ($p<0.05$) respectively. The elevated plus maze's results indicate an increase the duration of the deployment in open arms dose of 80 to limit ($p<0.001$) respectively. Es tablishment of the close arm time is a measure of stress in a dose of 80 significantly extend ($p<0.01$) declined. the result indicated that the use of toad skin secretion improved depression caused by AD, so compounds in this secretion can be considered as a candidate relive depression and AD. However, further studies are needed to determine its exact mechanism of action.

P26

Vitamin D Administration, Cognitive Function, Blood Brain Barrier Permeability and Neuro-Inflammatory Factors in High-Fat Diet Induced Obese Rats

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Recently neuro-inflammation and cognitive impairment has attracted attention. It has been suggested that obesity lead to cognitive impairments induced by neuro-inflammatory markers like nuclear factor kappa B (NF-kB) and reduced neurotrophin factors like brain-derived neurotrophic factor (BDNF) in the hippocampus. Also, increased blood brain barrier (BBB) permeability. Because of the neuro-protective effects of vitamin D, we aimed to investigate the effects of vitamin D on cognitive function, NF-kB and BDNF concentrations in the hippocampus and BBB permeability high-fat diet induced obese rats. Forty male Wistar rats were fed either a control diet (CD) or high fat diet (HFD) for 16 weeks, then each group randomized in to two subgroups supplemented with vitamin D for 5 weeks. Morris Water Maze test was done at the 21st week to examine cognitive function, BBB permeability was characterized by measuring Evans blue dye in the hippocampus. Moreover, BDNF and NF- kB protein levels in the hippocampus. HFD significantly led to cognitive impairments, due to elevated NF-kB concentrations as neuroinflammatory factor ($P=0.01$) and reduction of BDNF ($P=0.04$) concentrations in the hippocampus. we showed that vitamin D supplementation in HFD group reduced body weight, NF- kB concentrations, BBB permeability ($P=0.001$ and $P=0.03$ respectively) and increased BDNF concentrations ($P=0.002$). Vitamin D reversed HFD induced cognitive impairments via reduction of the NF-kB, elevation in BDNF and modulation of BBB permeability in hippocampus, thus it can be considered as a beneficial therapeutic approach for prevention and treatment of neuroinflammation and cognitive deficits.

P27

KCNK2 and Adhesion Molecules in an in-Vitro Blood Brain Barrier Model

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Two-pore domain potassium channels, like KCNK2, are known to play an important role in inflammatory

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diseases such as multiple sclerosis (MS). Upregulation of cellular adhesion molecules in mouse brain microvascular endothelial cells (MBMECs) of Kcnk2-/- mice resulted in elevated leukocyte trafficking into the central nervous system under inflammatory conditions. The current project aims to gain deeper insights into the role of KCNK2 in the regulation of adhesion molecules and cell trafficking at the blood-brain-barrier (BBB). Therefore, we used a dynamic in vitro model of the BBB to investigate brain endothelial cell – T cell interactions under physiological and pathophysiological conditions. MBMECs from either wild type mice or Kcnk2-/- mice were seeded into flow chambers and T cell migration behavior was investigated under mild shear stress (0.25 dyn/cm2). Experiments showed so far increased T cell migration under inflammatory conditions and decreased migration while blocking cellular adhesion molecule ICAM1 on wild type MBMECs. In future experiments, we will use static transwell assays to assess how different subgroups of T cells are influenced by pharmacological KCNK2 modulation. Overall, our project might identify new therapeutic strategies to influence immune cell trafficking at the BBB.

P28

A Case of Dissociative Amnesia after Hospitalization

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It shows that a possible relationship between anxiety and dissociation disorder. Dissociative disorder includes dissociative amnesia, depersonalization and the realization that they can occur when a person exposes to severe psychiatric stressful events that they have strong negative emotion for him and then he cannot cope with them, so use unsuitable strategies for coping such as memory loss (dissociative amnesia) in order to separate some aspect of traumatic stressful events. In this case report, we presented an 86-year-old man who was presented to the emergency medicine by GI Bleeding because of Ileocecal mass, finally right hemicolectomy and chemotherapy were done. Hospitalization like a traumatic experience increased his anxiety and amnesia of the stressful events that he experienced in this period was occurred. He couldn't remember the specific aspects of his surgery process and hospitalization. Unfortunately, clinicians focused on physical symptoms as the main clinical plan and mental illnesses usually was ignored. Dissociative experiences are life-threatening and can worsen the condition and severity of somatic illness. Such conditions are not uncommon thus routine evaluation of presenting these symptoms is necessary in clinical setting in order to help people to retrieve their memory,

P29

The Role of Platelet Granules in Neuroinflammation

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Platelets are known to contribute to vascular pathologies, however, their role in inflammatory disorders of the central nervous system (CNS), such as multiple sclerosis (MS) and its mouse model, experimental autoimmune encephalomyelitis (EAE), is thus far poorly defined. Although there is emerging evidence that platelets might accumulate in the CNS parenchyma along with an increased activation status and secrete proinflammatory factors thereby triggering immune response cascades during neuroinflammation, the role of platelet granules remains elusive so far. We investigate here the contribution of platelet granules to immune response during neuroinflammation. Therefore, we performed experiments using Munc13-4-deficient mice since mutation in Munc13-4 leads to abolished platelet dense granule secretion and compromised α-granule release. We found that genetic deficiency of platelet granules renders mice less susceptible to EAE. This reduction in disease severity was accompanied by reduced numbers of interleukin (IL)-17A- and interferon (IFN)-γ-producing proinflammatory effector T-helper cells as indicated by decreased cytokine levels of IL-17A and IFN-γ compared to control mice. Taken together, our findings show that genetic inhibition of platelet granule release significantly reduces CNS inflammation in mice, potentially indicating a novel therapeutic strategy for the treatment of MS. To understand the underlying mechanisms, further investigations are required.

P30

Effects of Hemin on Ca2+Influx in Neurons of C57BL/6 Mouse Brain

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Excitotoxicity results in a significant increase in Ca2+ influx; essentially from open N-Methyl-D-aspartate

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receptors (NMDARs) channels that cause a secondary rise in the intracellular Ca2+ concentration. It is correlated with neuronal death induced by Ca2+ overload. Dysfunction of NMDARs is associated with excitotoxic neuronal death in neurodegenerative disorders. In this study, the effects of hemin on Ca2+ permeability in neurons of C57BL/6 mouse brain examined. Isolated from 1-dayold C57BL/6 mice, were cultured in serum-free media. Cells were maintained in growth medium at 37◦C in 95% air/5% CO2 for 2 weeks in vitro before treatment. Primary neurons were cultured in serum-free media were treated with hemin (0, 12.5, 25, 50, 75,100 µM) for 18 (h). Intensity of calcium fluorescence was reduced in treated cultures with hemin (100, 86, 78.5, 60, 56, 46%, respective to the concentrations stated previously; P<0.05 for all). Hemin increased Ca2+ influx in cultured neurons. NMDAR stimulation by hemin increased the activating of NMDARs and Ca2+ influx in the cultured neurons. Therefore, heminis cytotoxic due to increase of intracellular Ca2+ influx.

P31

The Relationship between Serum Substance P Level and the Type as well as Prognostic Factors in Patients with Stroke

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Stroke is the world's largest neurological defect caused by the disruption of brain blood circulation. Apart from death, the severest damage caused by stroke, a plethora of other mental and physical disabilities can ensue the incidence of a stroke. As a result of the continued disruption of blood circulation triggered by a stroke, biochemical and physiological mechanisms affect nerve cells and cause secondary damages. One of the most diverse mechanisms of secondary damages leading to cell damage or death is the release of tachykinins including substance P (SP). The release of tachykinins such as SP provokes inflammatory responses like blood flow interruption and increased vascular permeability in brain. Considering the delay in the emergence of secondary damages, pharmacological interventions can offer an opportunity to reduce cell damage and death. In this study, serum SP levels have been measured in ischemic and hemorrhagic strokes and it was analyzed in terms of clinical variables such as type of lesion, lesion size, gaze and NIHSS. In this study, 75 persons (18 patients with a diagnosed ischemic stroke, 23 hemorrhagic patients and 34 healthy subjects as the control group) were s tudied. After examining, the clinical variables such as stroke size, NIHSS, gaze, hemiplegia type and degree of consciousness were recorded for each patient. Then, the serum SP level was measured by ELISA and the results were analyzed by SPSS. Serum SP levels were significantly higher in patients compared to healthy groups (p=0.001), but this difference was not statistically significant between hemorrhagic and ischemic patients. Similarly, prognostic factors and serum SP level were not significantly correlated (p=0.775). Serum SP level increased in stroke patients. Moreover, the results show that the type of lesion was not related to the SP level.

P32

Effect of Airway Pressure Release Ventilation Mode on Intracranial Pressure and Oxygenation in Patients with Traumatic Brain Injuries

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This study aimed at compare influences of airway pressure release ventilation (APRV) and Synchronized Intermittent Mechanical Ventilation (SIMV) on intracranial pressure and oxygenation status in patients with traumatic brain injuries. A clinical trial was carried out in 40 patients with traumatic brain injuries in the intensive care unit in Kamyab neurosurgery Hospital, Mashhad, Iran. The patients meeting the study inclusion criteria were randomly assigned into two groups; intervention (APRV, n= 20) and control (SIMV, n= 20). The ICP, CPP, MAP, PaO2, SPO2, PaO2/FIO2 in both groups were measured before and after conditioning. Analysis of data was done using independent t-test in SPSS V.22. The mean ICP remained unchanged in both groups (P= 0.421). After the intervention, the CPP, MAP, heart rate, and pulse pressure in APRV group were not significantly different compared with those in the two groups before the intervention (P> 0.05), PaO2, SPO2, PaO2/FIO2 in APRV group were significantly improved. The results showed APRV as a safe mode that can be beneficial in patients with traumatic brain injury without concerns for increased intracranial pressure.

P33

The Effect of Boswellia Serrata Extract and AKBA (Acetyl-11-keto-β-Boswellic Acid) on the Neurological Scores, Brain Edema and Brain -Blood Barrier after Severe Traumatic Brain Injury in Male Rats: the Role of IL-1β and IL-10

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Gladiolus plant is a tree from the family of Khorrasa. Boswellia serrata reduces glutamate-induced peritomerial edema. It also has potent antioxidant properties and immunosuppression, and anti-apoptosis in the central nervous system, and can be used to treat neurodegenerative diseases such as Alzheimer's disease, Huntington's disease, Parkinson's disease and dementia. However, its precise mechanism is still unknown. In this study, we investigated the effects of neural protection of the condor plant after induction of cerebral inflammation in rats. The male Albino wistar rats received different doses of Boswellia serrata (125, 250, 500 mg/kg, i.p.). All animals were intubated before surgery. In the TBI groups, diffuse TBI was induced by Marmarou method using a TBI induction device. The severe TBI was induced using a weight 450 gr.In the sham groups, all stages of induction of TBI were performed except dropping weight on the head. The disruption of Blood brain- barrier (BBB) was evaluated 6 h post- TBI. The neurologic score(VCS) and brain water content, the beam-walk –balance task (WB) were determined before trauma(Pre), on trauma time(D0), and first day (D1) and second day (D2) and third day (D3) post- TBI. 24 hours After TBI anaesthetized animals were sacrificed and the brain was removed for IL10 and IL-1B Elisa assay. Our results showed that traumatic brain injury led to significant brain edema and disrupt of blood brain-barrier and neurological defect and vestibulomotor dysfunction in the rat brain and decrease IL1B and increase IL-10 in brain tissue. Boswellia serrata (250, 500 mg/ kg) could attenuated brain edema, improved BBB and vestibulomotor dysfunction in compare with TBI control group ($P<0.001$) but in 500 dose results were better. These findings showed that Boswellia serrata has a prominent role in TBI outcome's and perhaps protect neurons through modulating inflammatory and antioxidant pathways

P34

Berberin Exerts Neuroprotective Effects by Modulating Pro and Anti-Inflammatory Cytokines in Rat Model of MCAO

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): P34

Many complicated mechanisms are involved in brain ischemia and the role of inflammatory factors in the progression of post-ischemic injury is inevitable. In present study, anti-inflammatory effect of berberine has been investigated in reperfusion injury after acute ischemic stroke. Male Wistar rats weighing 250-270 gr were randomly divided into four cohorts: healthy rats (control, n=20), sham-operated animals (sham, n=20), ischemia/reperfusion (I/R) cohort (MCAO=20), I/R+ berberine cohort (MCAO+ berberine, n=20). The animal subjected to ischemia for 45 min and berberine (40mg/kg) was interaperitoneally administrated 1 h after reperfusion. At 24 h after reperfusion, the animals were sacrificed under deep anesthesia and expression changes of pro and anti-inflammatory cytokines were determined by immunohistochemical assay. To evaluate brain edema, brain water content was measured in experimental groups 24 h after reperfusion. The infarct size was determined using 3, 5-Triphenyltetrazolium chloride (TTC) staining. significant increased levels of pro-inflammatory cytokines (IL1β and TNFα) were found in MCAO cohort compared with control and sham cohorts. Likewise, expression level of anti-inflammatory cytokines (IL10) was slightly increased following I/R in MCAO cohort. berberine conferred a neuro-protective effect via reduction of infarct size, preventing brain edema and significant down-regulation of pro-inflammatory cytokines and up-regulation of anti-inflammatory cytokines. In sum, our findings demons trate that berberine post-treatment attenuates focal cerebral IR injury by targeting inflammation process. This opens up potential avenues for future research and treatment of ischemic stroke.

P35

Berberin Exerts Neuroprotective Effects by Modulating Pro and Anti-Inflammatory Cytokines in Rat Model of MCAO

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): P35

Many complicated mechanisms are involved in brain ischemia and the role of inflammatory factors in the progression of post-ischemic injury is inevitable. In present study, anti-inflammatory effect of berberine has been investigated in reperfusion injury after acute ischemic stroke. Male Wistar rats weighing 250-270 gr were randomly divided into four cohorts: healthy rats (control, n=20), sham-operated animals (sham, n=20), ischemia/reperfusion (I/R) cohort (MCAO=20), I/R+ berberine cohort (MCAO+ berberine, n=20). The animal subjected to ischemia for 45 min and berberine (40mg/kg) was interaperitoneally administrated 1 h after reperfusion. At 24 h after reperfusion, the animals

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P36

The Interplay of Multiple Sclerosis and Menstrual Cycle: which one Affects the Other One?

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Menstruation is suggested to affect multiple sclerosis (MS) symptoms, while the effect of MS on menstruation is not studied before. Here, we aimed to compare the pattern of menstrual cycle and its symptoms between MS patients and healthy controls. This is a cross-sectional study conducted during 2015–2016 in MS clinic of Kashani hospital, Isfahan, Iran. We included female patients > 14 years with diagnosis of relapsing-remitting MS, and healthy subjects as the control group. We collected data regarding menarche age, menstrual characteris tics, history of premenstrual syndrome, the amount of menstrual bleeding, and the possible perimens trual symptoms from all subjects. Also, MS patients were asked to report changes in menstrual characteris tics after MS occurrence. The final study population contained 181 MS patients and 202 healthy subjects. The mean age in MS and control group were 36.04 ± 9.86 and 35.16 ± 11.30, respectively (P-value = 0.426). Menarche age in MS patients and control group were not statistically different (13.59 ± 1.87 and 13.29 ± 1.53, respectively; P-value = 0.087). Changing mens trual characteristics was reported in 70 MS patients (38.7%). Irregular menstrual cycle increased from 21% to 40.3% after occurrence of MS (P-value < 0.001) and was reported 24.7% in the control group. MS patients versus controls reported more symptoms before, during, and after their menstrual period (P-values < 0.001). We found no difference regarding menstrual characteristics in MS patients before onset of the disease and healthy controls. Irregular menstrual cycle was observed more after the disease occurrence while other menstrual characteristics did not change. Moreover, MS patients reported many more perimenstrual symptoms.

P37

Decreased Serum Levels of Interleukin-35 Among Patients with Relapsing Remitting Multiple Sclerosis

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The regulatory role of interleukin-35 (IL-35) in the immunopathogenesis of multiple sclerosis (MS) is sugges ted in very few studies. We aimed to measure serum levels of IL-35 among clinically isolated syndrome (CIS) and relapsing-remitting MS (RRMS) patients and evaluate the associations between this cytokine and the disease clinical course. This cross-sectional study was conducted during 2017 in MS Clinic of Kashani hospital, Isfahan, Iran. Forty patients with the diagnosis of CIS and RRMS according to McDonald criteria were included in the study, as well as 40 healthy controls. Also, data regarding clinical course of the disease was collected from cases. The levels of IL-35 in the serum of all subjects were determined by ELISA. Serum levels of IL-35 were reduced (p = 0.003) in RRMS in comparison with healthy controls. Moreover, the mean serum levels of IL-35 among new cases (diagnosed within 6 months before the study) were decreased compared to healthy controls but it were not s tatistically significant (P=0.059).The mean serum levels of IL-35 were significantly higher in new cases compared with other cases (p=0.048). We found decreased serum levels of IL-35 among RRMS patients compared to the healthy controls. We provide a view of the possible role of IL-35 in MS pathogenesis and the potential therapeutic targets in this way.

P38

The Immunoregulatory Effect of Cyclic Dinucleotides on Human Immune Cells

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In multiple sclerosis (MS) beneficial effects have been assigned to the interferon (IFN)-I subclass IFN-ß, making its administration a first-line disease-modifying treatment in MS. IFN-I responses can be induced by cyclic-dinucleotide (CDN) triggered activation of Stimulator-of-interferon-genes (STING) and have essential immunomodulatory effects. A beneficial effect of STING activation on neuroinflammation has been demonstrated in recent in vivo experiments using animal models. Here, we investigate the impact of the CDN-STING-pathway on the regulation of innate and adaptive immune responses. We first disclosed the expression of Sting via real-time PCR (rt-PCR) in murine immune cells linked to MS pathophysiology. Next, we demons trated that the high expression in some murine immune cells can also be shown in corresponding human-cell subsets. Flow cytometric and rt-PCR analysis showed that in vitro activation of immune cells by CDN leads to strong IFN responses in human peripheral blood monocytes. Consequently, we scrutinized the resulting cytokine effector profile and depicted activation and apoptosis processes in immune cell subsets. Overall, further investigations are needed to clarify the impact of CDNs on individual cell-subsets of innate-adaptive-interface and resulting interactions. By conducting ongoing studies, we aim to achieve insights into CDN and INF specific effects and potential applications for translational medicine.

P39

The Neuroprotection Effect of Erythropoietin in Cerebral Ischemia

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Cerebral ischemia causes death of millions people all over the world, annually and also suffering more people from neurological deficits and neuromuscular disorders. In our country, 250 to 300 people experience mild to severe stroke, daily. In this study we reviewed 120 original paper selected from PubMED database. Our keywords were erythropoietin, anti-inflammatory, stroke, neuropathy and cerebral ischemia. Studies have been revealed that anti-inflammatory and neuroprotection effects of erythropoietin are mediated by receptors that available in cerebral cortex, spinal cord, hypothalamus and hippocampus. These effects include the ability to repair neural inflammation, prevention of neural cell death, preservation of surviving neural cells, regulation of neurogenesis, anti-apoptosis and anti-coagulation. Erythropoietin also prevents Alzheimer's disease, Parkinson's disease, epilepsy, multiple sclerosis, and other motor diseases. All studies showed that erythropoietin has anti-inflammatory and neuroprotection properties and can decrease probability of cerebral ischemia, impressively. Today, erythropoietin is considered as an attractive and effective therapeutic approach to cerebral ischemia; one of the most common causes of morbidity and mortality around the world.

P40

The Effect of Prevention and Treatment of Cerebral Ischemia on the Basis of Neuroprotective Properties of Medicinal Herbs

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Stroke as the third cause of death in industrialized societies after cardiovascular and cancerous diseases Based on the type of artery involved, its location and size can lead to various side effects such as half-body movement disorder, sensory impairment, memory impairment, and other problems. In this regard, due to the complications of chemical drugs and their long-term use in treating the disease, and on the other hand, by proving the efficacy of herbal medicines in scientific societies, herbal medicines can be used as alternative treatments with fewer complications or supplementation of treatment Used. The aim of this study was to inves tigate the relationship between the effect of herbs and the rate of stroke reduction. This study is a review of the literature. A total of 12 research articles, review articles and meta-analyses published from 2005 to the end of 2016 in the database of Pubmed, Ovid, Elsevier, ProQuest, Google and iran medex using keys words cerebral ischemia, herbs, antioxidant titles and or abs tracts for herbs or herbal supplements, such as virgin olive oil, black cherry and watermelon, were searched and examined. A review of studies has shown that drug treatments and their derivatives can reduce the amount of brain damage, cerebral edema, sensory and motor disorders, and the consequences of cerebral ischemia (tissue damage to the pneumobra and kanon regions). A roughly common mechanism is the reduction of oxidative and titrating stress, increased nitric oxide, reduced cerebrospinal fluid flow, decreased the activity of microglia and astrocytes, and inhibited the expression of apoptotic proteins. The findings show that herbal treatments can be dramatically enhanced due to their

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neuroprotective properties The risk of lesions and abnormalities after cerebral ischemia can be reduced, but the desire of different groups of society to this range of drugs cannot be neglected.

P41

Health-Related Quality of Life in Adults with Epilepsy: A Systematic Review

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Considering the burden of epilepsy on society, inves tigation of the health-related quality of life (HRQoL) is essential for the better future targeting, optimization of current interventions and managing strategies for epilepsy. Nineteen HRQoL were investigated in this review. Increases in seizure incidence, level of depression and seizure severity were significantly related to reduced HRQoL. The mainstream of studies was cross-sectional and had a general methodologic quality that was arbitrated to be “moderate” for HRQoL studies and “poor” for health care resource. In the 7 studies, gender, age, type of seizure and duration of epilepsy did not seem to be related with HRQoL, while the predictive effect of employment and educational status and number of antiepileptic drugs (AEDs) was unclear. The relation among predictive factors and HRQoL seemed to be reliable across individuals whether refractory or seizures controlled by AEDs. In addition to seizure control, effective epilepsy controlling needs the early recognition of those most at risk of psychological dysfunction. For more researches, it is essential that multivariate statis tical strategies are predicted for HRQoL in epilepsy.

P42

The Effects of Vitamin A on Inflammatory Factors (CCL2, CCL18, CD14) in Multiple Sclerosis

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Multiple Sclerosis (MS) is a complex neurological disease in which neuro inflammation that leads to neurodegeneration _ plays a key role and its prevalence is 2 million in the world. Vitamin A is a fat-soluble vitamin that is multifunctional. One of the important functions is in immune system, both in immunological tolerance and in adaptive immune responses. 264 patients will be enrolled that suffer from MS and divided them into two groups. Group (A) will take interferon beta and Vitamin A and group (B) will take interferon beta and placebo. we will follow them for one year and every six months we will check the inflammatory factors in the serum. In this project, we expect that vitamin A regulate CCL2 (as a chemokine) which means vitamin A will increases amount of CCL2 in serum and then CCL2 will effect on TH cells (stimulate TH2 and TReg) and then IL-10 that is anti-inflammatory cytokine will increase and IL-2 that is pro-inflammatory cytokine will be inhibited by IL-10. We guess if vitamin A increases the amount of CCL2 about 50 pg/ml we can see the expected changes. Based on other researches, we expect that vitamin A regulate the inflammatory factors and then reduce the rate and intensity of relapsing in patients.

P43

The Effect of Carvacrol on Blood Pressure and Some Blood Parameters in Lead-Exposed Rats

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Lead intoxication is one of the most health hazards in humans at all ages. Lead impairs oxidant/antioxidant balance that can be partially responsible for the toxic effect of lead in the various organs of body especially cardiovascular system. The main objective of this study was to evaluate the effect of carvacrol as anti-oxidative agent on hypertension and some blood parameters in lead poisoned rats. 40 male wistar rats were used in this study and were randomly divided into 5 groups. The first group was control, the second group was lead acetate (500ppm) orally received, 3 another groups co administrated lead acetate with carvacrol (25, 50 and 100 mg/kg daily for 40 days). The systolic blood pressure was monitored weekly by tail plethysmography coupled to a computer system. Blood samples were obtained for assessment of some hematological parameters (RBC, Hb, Hct, WBC) at the end of experiment. The mean blood pressure in lead exposed group was significantly higher than control group from 21 days. Carvacrol caused a decrease in hypertension in the lead poisoned rats compared with control group. This deceasing was consistent throughout the experiment. Some blood parameters (RBC, Hb, Hct, WBC) were found to be decreased in the lead groups. These changes were prevented in the lead groups that received carvacrol. According to the result of this study, it may be concluded that carvacrol could improve some lead induced changes in the cardio vascular system.

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P44

The Antioxidant Effect of Nanomicelle Curcumin in Bisphenol A-Induced Brain Toxicity Following Sub-acute Exposure in Rats

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Bisphenol A (BPA) is used in the manufacture of polycarbonate plastics and epoxy resins; therefore, exposure to BPA is increasing every day. BPA has toxic effects on various human tissues. Curcumin, a yellow polyphenol, is the active turmeric ingredient. It is an efficacious and safe compound with multiple pharmacological activities including antioxidant, ant carcinogenic, ant proliferative, and anti-inflammatory properties. This study was designed to determine the potential protective effect of nanomicelle curcumin on BPA-induced subacute brain toxicity in rats. The wis tar rats were divided into six groups (8 rats/group). The first group served as the control (dextrose 5% + sesame oil); the second group received 50 mg/kg nanomicelle curcumin; the third group was fed 50 mg/kg BPA; the fourth, fifth, and sixth groups received 10, 25, and 50 mg/kg nanomicelle curcumin, respectively, supplemented with 50 mg/kg BPA, after one hour. At the end of the study period (4 weeks), MDA level and GSH content were measured in the cerebellum, cortex and hippocampus. This study revealed that the dose of 50 mg/kg of BPA significantly increased malondialdehyde in the cerebellum ($P< 0.001$), cortex ($P< 0.001$) and hippocampus ($P< 0.01$). In addition, BPA decreased glutathione content in the cerebellum ($P< 0.001$), cortex ($P< 0.001$) and hippocampus ($P< 0.01$) as well. However, nanomicelle curcumin (50 mg/kg) significantly improved these toxic effects of BPA in rat brain tissue. The results provide evidence that nanomicelle curcumin has preventive effects in subacute exposure to BPA (50 mg/kg) induced toxicity in rat brain tissue.

P45

The Effects of Nigella sativa on Sickness Behavior Induced by Lipopolysaccharide in Male Wistar Rats

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Neuroimmune factors contribute on the pathogenesis of sickness behaviors. Nigella sativa (NS) has anti-inflammatory, anti-anxiety and anti-depressive effects. In the present study, the effect of NS hydro-alcoholic extract on sickness behavior induced by lipopolysaccharide (LPS) was investigated. The rats were divided into five groups (n=10 in each): (1) control (saline), (2) LPS (1 mg/kg, administered two hours before behavioral tests), (3-5) LPS-Nigella sativa 100, 200 and 400 mg/kg (LPS-NS 100, LPS-NS 200 and LPS-NS 400, respectively). Open- field (OF), elevated plus maze (EPM) and forced swimming test (FST) were performed. In OF, LPS reduced the peripheral crossing, peripheral distance, total crossing and total distance compared to control ($p<0.01$- $p<0.001$). The central crossing, central distance and central time in LPS-NS 100, LPS-NS200 and LPS-NS 400 groups were higher than LPS ($p<0.01$- $p<0.001$). In EPM, LPS decreased the open arm entries, open arm time and closed arm entries while increased the closed time compared to control ($p<0.001$). Pretreatment by NS extract reversed the effects of LPS ($p<0.05$- $p<0.001$). In FST, LPS increased the immobility time while, decreased the climbing and active times compared to control ($p<0.05$- $p<0.001$). The results of the present s tudy showed that the hydro-alcoholic extract of NS reduced the LPS-induced sickness behaviors in rats. Further investigations are required for understanding the responsible underlying mechanism(s).

P46

Optic Neuritis as the First Manifestation of the Primary Antiphospholipid Antibody Syndrome

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Optic neuritis is commonly associated with diseases such as Multiple Sclerosis, infections, taking special drugs, etc. but recently we have observed a patient that presented retro bulbar optic neuritis as a manifestation of antiphospholipid syndrome. A 35 y/o young woman with the history of chickenpox admitted to the Qhaem hospital (Mashhad, Iran) with the c/o progressive pain and blurred vision in the left eye. the brain MRI was normal. the VEP test demonstrated severe left optic neuritis and the right optic nerve was also mildly affected. the concentration of glucose, urea, electrolytes

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and CBC were WNL, but the presence of autoimmune antibodies in her blood proposed the probability of an autoimmune disease. she received Corton and Val acyclovir orally. After receiving Corton pulses her vision was moderately improved and she was released with some drug prescriptions. Also she was to perform vasculitis tests.13 days later, after consulting with the rheumatologist about the results of the vasculitis tests, she was referred to the neurology department again. she was undergoing 5 sessions plasma pheresis. Then, she was recharged with moderate general good feeling. According to this patient and other similar cases, for the patient that is affected by vasculitis and also have thrombocytopenia, but does not fulfill the criteria for lupus, antiphospholipid syndrome may be considered as a possible diagnose.

P47

Effects of Adipose Derived Stem Cells Transplantation on Locomotor Activity and Imbalance in Parkinson Model of Rats

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Parkinson disease is a neurological disorder accompanied by degeneration of dopaminergic system and neuronal loss. Recently studies have focused on s tem cells therapy, therefore the goal of this study was to investigate the effect of adipose derived stem cells grafting, on locomotion and imbalance in Parkinson model of rats. Twenty-four male Wistar rats weighing 200-250 g were divided into three groups including: sham, MPTP, MPTP-stem-cell. Rats were cannulated by stereotaxic apparatus in medial forebrain bundle (MFB) under anesthesia. Then MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) (1µm/2µl) were injected bilaterally into the MFB to induce Parkinson model. Rotation (following apomorphine injection) and Ladder Rung Walking Task were conducted to confirm model of Parkinson, then stem cells from passage 3 (5 ×103 cells/mm3) were infused into the MFB. After 6 weeks Ladder test was repeated. The results showed that administration of MPTP significantly caused motor disability (p=0.025) and treatment of stem-cells significantly improved motor activity and imbalance (p=0.05). Our findings indicate that grafting of stem cells derived from adipose tissue improves behavioural dysfunctions probably by regeneration of dopaminergic neurons.

P48

Protein Changes Resulted in Sub-Chronic Neurotoxicity of Bisphenol A in Rat Brain

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Bisphenol A (BPA) is one of the most widely used chemicals in the plastic industry, which enter the human body through occupational and food contact. In this s tudy, the protein changes in rat cerebral cortex was evaluate in order to evaluate the neurotoxicity of BPA. 24 adult male rats were randomly selected and divided into four groups (n=6) and each group respectively received 0, 0.5, 5 and 50 mg/kg of BPA for 4 weeks orally. To determine the oxidative status, reduced Glutathione (GSH) and Malondialdehyde (MDA) were measured in brain cortical tissue. After extracting the protein of each sample, the proteins transferred to the acrylamide gel of two-dimensional electrophoresis and from the obtained protein map, 10 points – with at least 10% or more volume difference with control group - were sent for mass spectroscopy analysis. The lipid peroxidation in both doses of 0.5 and 5 mg/kg was significantly ($P < 0.05$) greater than the control group. Based on the results of mass spectroscopic analysis and data from the Mascot database, 10 changed proteins were identified as below: Pyruvate kinase PKM (Pkm), Alpha-enolase (Eno1), Aconitate hydratase (Aco2), and Creatine kinase B-type (Ckb) -involved in the metabolism of neurons-, Phosphatidylethanolamine-binding protein 1 (Pebp1), 14-3-3 protein eta (Ywhah) and Guanine nucleotide-binding protein subunit beta-1 (Gnb1) –which play different roles in cell signaling. Dihydropyrimidinase-related protein 2 (DPYSL2) and Glutamine synthetase (Glul) -which are important in the proper functioning of the neurons- and a structural protein; the Neurofilament light polypeptide (Nefl). Different reports indicate that changes in the level of these proteins are related to various neuropsychiatric disorders such as Alzheimer's disease, Parkinson's disease, depression, schizophrenia, and brain tumors. Further studies are needed to examine the role of BPA in these diseases.

P49

The Evaluation of Aqueous Extract of Glycyrrhiza Glabra on Nerve Recovery in the Rat after Sciatic Nerve Injury

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Peripheral nerve injury requires a long recovery period, and recovery, once attained, usually is incomplete. Inflammatory procedures may inhibit functional recovery after nerve injury and produce cell death in both the central nervous system and the peripheral nervous system. Since the glycyrrhiza glabra extract has anti-inflammatory effects, it could reduce the severity of injury. The aim of this study was to evaluation of aqueous extract of G. glabra on nerve recovery in the rat after sciatic nerve injury. 24 male wistar rats were randomly divided into four groups: control and G. glabra extract with 50, 100 and 150 mg/kg doses groups. Sciatic nerve was exposed to compression for 60 second using locker pincers. At days 7, 14, 21, and 28 nerve regeneration and functional recovery were evaluated using the sciatic functional index (SFI). Result of present study shows that at day 7 and 14 there were no significant differences between all groups in their SFI. At day 21 SFI was significantly improved in 100 mg group. Also, SFI differences between control group and 50 and 100 mg groups were statistically significant at 28-day pos t-injury. This study revealed that Aqueous extract of G. glabra is able to promote sciatic nerve regeneration and improve the function of a crushed sciatic nerve and the extract with 150 mg/kg dose had the largest impact. However, to confirm the present results and determine the exact mechanism more studies will be necessary.

P50

Selective HCRTR2 Antagonism Increases Embryonic Mouse Cortex Neural Stem Progenitor Cells Proliferation

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In multiple sclerosis Oligodendrocytes are obliterated by the immune system. neural stem/ progenitor cells (NS/P Cs) have the capacity to differentiate into mature myelinating oligodendrocytes. In embryonic mouse cortex oligodendrocyte progenitor cells (OPCs) are more abundant than the ganglionic eminence. Doing gene set enrichment analysis using DAVID and Panther websites it was shown that Gpr37l1 is highly expressed in oligodendrocyte progenitor cells (OPCs) in comparison to oligodendrocytes. The selective orexin 2 recptor (HCRTR2) antagonist jnj-10397049 has been shown to inhibit this orphan GPCR. In this study we sought to scrutinize NS/P Cs survival after the inhibitor on gpr37l1 and HCRTR2 by jnj-10397049. primary cortex NS/PCs were derived from embryonic mouse 13.5 as described before. cytotoxicity effect of varying doses of JNJ10397049 was screened using MTT assay. The expression of gpr37l1, hcrtr2, PDGFRalpha and Cnpase expression was analyzed using real time PCR. MTT analysis demonstrated that JNJ10397049 at 15 and 10 micromolar dramatically increases proliferation of neural stem cells by 2.62 and 2.43 respectively. Gpr37l1 and orexin2 receptor are more expressed in embryonic mouse cortex NS/PCs than embryonic mouse ganglionic eminence by 3.45 and 4.57, respectively. PDGFRalpha and Cnapse genes are also highly expressed on cortex NS/PCs by 112.36 and 76.56, respectively in comparison to ganglionic eminence NS/PCs. Here it was shown that Orexin 2 receptor and GPR37L1 can be valid drug targets in demyelination diseases by inducing proliferation of NS/PCs. Further study is under process to confirm and expand these results.

P51

Anti-Inflammatory Curcumin Effect on Neuronal Number in the CA1 Area Following Global Cerebral Ischemia

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Global cerebral ischemia (GCI) leads to inflammation and neuronal death in CA1. Curcumin with neuroprotective and anti-inflammatory properties is a potential candidate for suppressing cell death. The aim of this study was to determine the effects of curcumin on neuronal number in the CA1 area following GCI. 28 Sprague-Dawley male rats were randomly assigned into four groups including sham, control, and curcumin 50 and 100 mg/kg (n= 7/group). Two treatment groups were orally received curcumin for 28 days. Control group was received PBS, Sham group did not receive anything. Cresyl violet staining following GCI was performed in 40µm paraformaldehyde perfused sections. Then the volume "v (CA1)" was estimated using the Cavalieri method and the total number of neurons in the CA1 area was determined using the optical dissector

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method. There were no significant differences in the volume of CA1 between studied groups. Total number of neurons significantly reduced in control group compare with sham group ($p<0.01$). Also there was no significant difference between curcumin 50 mg/kg and control group in total number of neurons. But, curcumin 100 mg/kg significantly increased number of neurons in comparison with curcumin 50 mg/kg ($p<0.05$) and control group ($p<0.01$). We found two effects curcumin dose dependently: the first, curcumin cause to prevent neuronal death, the second, curcumin increased neuronal number of CA1.

P52

The Survey of Correlation IL6 (-174)-IL10 (-1082/-819) Genes Polymorphism and Plaques in Women with Multiple Sclrosis

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Multiple sclerosis (MS) is a type of inflammatory disease in which the myelin sheath is attacked by the immune system in the central nervous system. Demyelin lesions , tissue damage, and neurodegenerative disorders in white matter are associated with decreasing cognitive decline in MS. Interleukin 10 is an anti-inflammatory cytokine .Interleukin 6 is a multi-functional cytokine that is used in the immune system . The aim of this s tudy was to determine the association between the polymorphism of genes 10 and cerebellar plaques in women with MS. This case-control study was performed on 20 female patients with multiple sclerosis as a case group and 20 women without MS lesions, as the control group diagnosed as having no disease according to a neurologist diagnosis. For genotyping, in the case and control group, 10 cc blood samples were taken. DNA extraction was performed using phenol chloroform. Genotyping of DNA was done by SSP-PCR method. Then, the brains of MRI images of these people who were taken by FLAOR method were used to count the number of plaque. The results indicated that the IL6 genotypic release in the case group compared to control (0.818) and IL10 (-819) and IL10 (-1082) in the case group was (0.890) and (0.997) There was no significant relationship. No significant association was found between IL-6 and IL-10 and plaques. This study did not show any association between plaques and genotypes IL10 (-819), IL10 (-1082), and IL6 in women with multiple sclerosis.

P53

The Effect of Cognitive Rehabilitation Program on Hope Status in the Elderly

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Along with increasing age of people, mental health problems have become an important concern worldwide. The aim of this study was to examine the effects of cognitive rehabilitation program on hope of the elderly. In this field trial, 70 retired elderly people were recruited via convenience sampling method and randomly allocated into intervention and control groups (n=35 per group). The intervention group participated in 12 one-hour sessions of cognitive rehabilitation program, whereas the control group received no intervention. Data were collected using a demographics form and Snyder et al.'s Hope Scale (1991). It was analyzed by SPSS software using paired t-test and independent t-tes t at a significant level $p<0.05$. The mean age of subjects in the intervention and control groups was 61.03 ± 3.81 and 61.02 ± 2.37, respectively. There was no significant difference between the two groups ($p = 0.98$, $t = 0.001$). The post-intervention mean score of hope was significantly higher than the pre-intervention stage ($p <0.001$), but there was no difference in the control group ($p = 0.84$). The mean score of hope after intervention was significantly greater in the intervention group than in the control group ($p <0.001$) while the score before intervention was similar in the two groups ($p = 0.09$). The cognitive rehabilitation program, including attentive and memory exercises, was effective in those who had somewhat lower hopes. Cognitive rehabilitation program can be a useful instrument for healthcare specialists to improve hope status in the elderly.

P54

The Oxidative Stress Assessment of Echium Amoenum on Mice Brain

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Echium Amoenum (Boraginaceae) is one of the important medicinal herbs in traditional medicine. It has

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known for its variety effects such as demulcent, anti-inflammatory and analgesic, especially for common cold, anxiolytic and sedative, this plant contains small quantities of pyrrolizidine alkaloids that are toxic and chronic consumption may have adverse effects on the body's organs. in this study, 60 mice were chosen and divided into four groups. Group (1) as control group (without injection), group (2) received 12.5mg/kg, group (3) received 25mg/kg, group (4) received 50mg/kg body weight plant. Boraginaceae was injected into mice for a month. Then, the mice were decapitated and brains were collected to evaluate oxidative stress. The level of lipid oxidation was significant in groups. Brain tissue is rich in polyunsaturated fatty acid. Therefore, the level of lipid oxidation is usually more than other tissue. Changes in Ion reducing antioxidant capacity assay (CUPRAC) were not significant. The level of glutathione (GSH) increase in high dose. Echium Amoenum may don't trigger toxicity in brain in low and intermediate doses.

P55

The Effect of Dimethyl Sulphoxide on Sciatic Nerve Regeneration in Rats with Eggshell Membrane Guide Channel

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Autograft is gold standard treatment for peripheral nerve repair up to now. Eggshell membrane (ESM) as nerve guide channel effectively enhances nerve regeneration. Dimethyl sulphoxide (DMSO) has anti-inflammatory and anti-oxidant properties. The aim of this study was to evaluate the effect of DMSO+eggshell membrane guide channel on sciatic nerve regeneration in rats. Thirty two adult male rats were randomly divided into sham surgery, autograft, ESM + normal saline (NS), and ESM + DMSO groups. A 10 mm segment of left sciatic nerve was removed. In the ESM groups, the cut ends of the nerve were telescoped into the nerve guide channel, and then DMSO or NS injected into them. In the autograft group the nerve segment used as an autologous nerve graft. Then all animals were evaluated by sciatic functional index (SFI), withdrawal reflex latency, histology, and gastrocnemius muscle weight. The mean of SFI and withdrawal reflex latency were improved in all groups. On the day 30 post-operation, the mean SFI of DMSO group was greater than the autograft and NS groups. The withdrawal reflex latency was not statistically significant in experimental groups. The number of myelinated axons in DMSO was greater than autograft and NS. The mean of muscle weight in DMSO group was significant more than autograft and NS groups. These findings demonstrate that ESM+DMSO effectively enhance nerve regeneration and promote functional recovery in injured sciatic nerve of rat.

P56

A Case Report on a New Aicardi-Goutieres Syndrome Inducing Gene

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Aicardi-Goutieres syndrome (AGS) is an inflammatory genetic disease inherited in an autosomal recessive manner. Common features of this disease are encephalopathy, splenomegaly and hepatomegaly, muscle stiffness, irritability, unstoppable crying, seizures and dilation in growth. According to previous studies, primary genes responsible for this Syndromes are as followed: TREX 1, RNASEH2A, RNASEH2B and RNASEH2C. Moreover, mutation in ADAR and SAMHD1 genes are assumed to play part in AGS. In this case we found a new gene mutation probably responsible for this syndrome. A 4 years old female with encephalopathy, one ear hearing impairment, strabismus, and hypertonic upper and lower limbs with tapering fingers was admitted to our genetic clinic. According to these clinical features and supplementary testing, she was diagnosed with AGS. Further molecular genetic testing indicated no homozygous mutations in common genes responsible for AGS in despite that both of her parents had a mutation in RNASEH2C and she carries only one mutated copy of RNASEH2C but a homozygous mutation in LAMA 1 gene, which encodes Laminin alpha 1 chain, was found. Previous studies demonstrated that LAMA 1 mutation could lead to motor neuron impairment and optical defections. It is necessary to emphasize that both of her parents had abnormal LAMA1 gene. With regard to our testing results, RNASEH2C mutation and LAMA 1 c.1957C>T mutation was concluded to be responsible for AGS in this case as a compound heterozygote. As a result, LAMA 1 can be introduced as a new AGS inducer. The findings of present research suggest the family should be subjected to PND in any pregnancy. After this report it is recommended to check LAMA 1 gene along with other responsible genes as a candidate for this syndrome.

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P57

Herbal and Complementary Medicine for the Treatment of Multiple Sclerosis

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Multiple Sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system. Signs and symptoms of MS vary widely and depend on the amount of nerve damage and which nerves are affected. A literature search was conducted using medical and health science electronic databases (PubMed and SID) up to January 31, 2018. Of the 150 records identified, 25 articles were eligible and reviewed using herbal medicine, phytotherapy, multiple sclerosis as keywords. Studies have been carried out on five medicinal herbs, including curcumin, cinnamon, ginseng, aloe vera and cannabis. Although the mechanism of herbal medicines is still not completely clear, most of these plants have anti-inflammatory and antioxidant effects. While mos t studies have highlighted the positive effects of these plants, few of these papers also pointed to the lack of their benefits. MS is a disease that usually affects young people, especially in their productive ages, and this can be the most severe complication of this disease. In addition, current drugs also have little effect on reducing the complications of the disease, despite their severe side effects. Therefore, Complementary and alternative medicine (CAM) is an area of great public interest and activity, both nationally and globally. Herbal medicines are the most commonly used complementary medicine worldwide, especially in East Asia.

P58

Visual Working Memory Performance Based on Saccades in Children with and without Specific Learning Disorder: An Eye-Tracking Study

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Some of the previous studies show that children with SLD have deficits in visual processing and working memory. Hence, the aim of this research was to inves tigate problems of visual working memory based on behavioral neuroscience method, using an eye tracker device. The method of present study was ex-post facto s tudy. The participants included couple of twelve children with SLD (mean age=10.92) and without SLD (mean age=12.50). For measuring visual working memory, CORSI task was used and eye-tracking was used for recording saccade duration, saccade frequency, saccade amplitude, and saccade latency and saccade velocity. The findings highlighted that there is a significant difference in block span, total score and memory span between children with/without SLD ($P< 0.05$). There was a clear difference in saccade amplitude and saccade duration between two groups and these indexes were poor in SLDs. Based on these findings, further studies of neural mechanisms of visual working memory in SLDs are needed for better clarification of such deficits.

P59

Effect of Umbelliprenin on Behavioral Responses of Neuropathic Pain and the Expression of Inflammatory Factors in Chronic Constriction Injury Model of Neuropathic Pain in Male Rats

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Despite extensive investigations, the main mechanisms underlying neuropathic pain development are still not fully understood and there is no effective treatment for that. So intensive research is being done for finding new, efficient analgesic drugs. 56 male Wistar rats (230±30) were divided into 7 groups (n=8); control, sham, CCI and 3 Umbelliprenin groups (25, 50 and 100 microgram/rat). CCI model was used to induce chronic neuropathic pain. Umbelliprenin was intrathecally injected from day before to 2 days after the surgery. Mechanical and thermal allodynia and thermal hyperalgesia were evaluated by von-Frey, acetone drop and hot-plate tes ts respectively. Lumbar enlargement of spinal cord was collected for protein content and gene expression analysis. Statistical analysis indicated that allodynia increased in CCI group from POD 4 and hyperalgesia from POD 2 significantly in compare to the control group ($P<0.05$). Furthermore, 50 ($P<0.01$) and 100 ($P<0.001$) doses significantly attenuated the induction of allodynia and hyperalgesia in compare to CCI group, and this effect was significant from POD 7-14. One-way ANOVA indicated that Umbelliprenin was able to reduce TNFα protein content and IL-1β gene expression. The results show that administration of Umbelliprenin before injury can prevent or delay the onset of neuropathic

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pain symptoms. Furthermore, it can reduce cytokine expression and protein content in lumbar spine of CCI rats. Our view is that these results could be useful in finding new drugs to treat or alleviate the symptoms of neuropathic pain.

P60

Mesenchymal Stem Cells Encapsulated in a Self-Assembling Nanopeptide Scaffold Attenuate Neuroinflammation and Behavioral Function in a Model of Traumatic Brain Injury in Rats

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Traumatic brain injury is one of the major causes of brain function impairments and surgery is involved in the treatment program of many cases although it cannot rescue the brain functions completely and is confined to reduce the second injury .in this study we aimed to inves tigate the effects of mesenchymal stem cells encapsulated in RADA1- GGS IKVAV, surgically injected into the lesion site. 36 male Wistar rats underwent an acute model of TBI. Subjects were divided into 5 groups, each consisting of 6 to 9 rats: Sham (receiving no treatment), PBS, GSIKVAV, Mesenchymal Stem Cells (MSCs), GSIKVAV+ MSCs. MSCs were stained with BrdU. Flow cytometry was done to characterize MSCs. Several Behavioral tests were conducted: Open Field (OF) and Elevated Plus Maze (EPM) to assess anxiety-like behavior and modified Neurological Severity Score (mNSS) to evaluate the sensory- motor function. Subjects were euthanized at day 30. IHC was carried out to measure MSCs' viability and differentiation. Also, Wes tern blotting was performed to check for inflammatory factors including toll like receptor3, 4, tumor necrosis factor α and glial fibrillary acidic protein. There was a significant decrease ($P < 0.05$) between MSCs and MSCs + GSIKVAV groups in mNSS score. In addition, the number of entries to the open arm in the EPM test and total distance in OF test was significantly higher ($P < 0.05$). Our data suggest that using MSCs in combination with GSIKVAV can rescue cognitive function. These findings suggest that new assembly peptides can be a new and potential therapy for TBI patients.

P61

Pandard Syndrome, Disorders and Treatments Available: An Overview Article

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Pendard syndrome is a genetic disorder that is usually associated with hearing loss in children and thyroid s tatus called goiter, and sometimes affects the balance of the person. Researchers estimate that 7 to 8 percent of the total congenital hearing loss is Pandard's syndrome. A sign that a person may have mutated the SLC26A4 gene is a family history of hearing loss in the early days. Another sign is a family member with goiter and hearing loss. The mutation in the SLC26A4 gene can be determined by genetic testing using a blood sample. In this article, we are trying to find out more about the latest articles on the disease so that the latest findings on the disease can be readily available to the reader. The systematic search of pub med and med science databases was done to obtain more information about the syndrome and identify the published articles as well as the therapeutic methods tested on this syndrome. A total of 20 articles in this field were reviewed. Among them, about 15 articles have been presented and studied the disease and various symptoms and causes of it, and 5 articles have introduced methods for preventing symptoms and functional disorders in activity. The thyroid gland and the inner ear of the affected person. There is no specific treatment for this syndrome, but it is possible to inform the patient and his family about therapeutic options to help and improve the condition of the patient. Children with this syndrome should be supported at an early stage in order to learn how to use communication skills at an advanced age. People with this syndrome should refer to the hearing specialis t in terms of auditory assessment and ENT. Cochlear implant surgery can also treat existing anomalies.

P62

Agmatine Protects Against Intracerebroventricular Streptozotocin-Induced Water Maze Memory Deficit, Hippocampal ApoptosisandAkt/GSK3β Signaling Disruption

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Intracerebroventricular stereptozotocin (STZ) treatment has been described as a suitable model for sporadic Alzheimer's disease (sAD). Centrally administered STZ decreases insulin and insulin receptors in the brain and interrupts PI3/Akt signaling pathway and GSK-3β. Additionally it raises Bax/Bcl-2 ratio and prompts hippocampal apoptosis. Agmatine, a polyamine derived from L-arginine decarboxylation, is recently shown to exert some neuroprotective effects. This study aimed to assess if agmatine reverses STZ-induced memory deficits and Akt/GSK-3β signaling disruption and apoptosis in the hippocampus. Adult male Sprague-Dawely rats weighing 200-250 g were used in this s tudy. The canules were implanted bilaterally into lateral ventricle. STZ was administered on days 1 and 3 (3 mg/kg). Agmatine treatment (40 or 80 mg/kg) was started from day 4 in an every other day manner and continued till day 14. The animal's learning and memory capability was assessed on days 15-18 using Morris water maze. After complement of the behavioral studies the hippocampi was isolated and the amounts of hippocampal cleaved caspase 3 (the landmark of apoptosis), Bax/Bcl-2 ratio, total and phosphorylated forms of GSK-3β and p-Akt were analyzed by western blot. The results showed that agmatine in 80 but not 40 mg/kg reversed the memory loss induced by STZ. Wes tern blot analysis revealed that STZ induced elevation of caspase-3; Bax/Bcl-2 ratio and disrupted Akt/GSK-3β signaling in the hippocampus. Agmatine prevented apoptosis and Akt/GSK-3β signaling alteration induced by STZ. This study disclosed that agmatine treatment avert not only STZ-induced memory deterioration but also hippocampal apoptosis and Akt/GSK-3β signaling interruption.

P63

Automatic Detection of Glioblastoma Multiforme Tumors Using Magnetic Resonance Spectroscopy Data Based on Neural Network

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Inflammation has been closely related to various forms of brain tumors. However, there is little knowledge about the role of inflammation in glioma. Grade IV glioma is formerly termed glioblastoma multiform (GBM). GBM is responsible for over 13,000 deaths per year in the America. Magnetic resonance imaging (MRI) is the most commonly used diagnostic method for GBM tumors. Recently, use of the MR spectroscopy (MRS) technique has been widely considered. The advantage of using MRS with MRI is that MRS can show biochemical biomarkers non-invasively. In the analysis of MRS data, the segmentation of GBM and normal regions is very important (especially in the borders) and requires very careful operation. The purpose of this study is to distinguish between GBM and normal regions using a neural network to improve the diagnosis of neuroscientists, neurologists, radiologists and neurosurgeons. Four patients, including 2 men and 2 women with GBM tumors, were studied according to the radiologist's comments .Applying a 1.5Tesla Siemens scanner, MRS data were acquired. Choline and n-acetyl aspartate metabolites were identified. In order to measure the concentration of metabolites, MRS data were first analyzed using TARQUIN software. Then, using MATLAB software, the calculated concentrations were classified into normal and GBM groups using a neural network. The results showed that sensitivity and specificity of classification are 78% and 87% respectively. By categorizing the data obtained from TARQUIN software and using neural networks, it is possible to determine the GBM and normal regions automatically in order to investigating the role of neuro inflammation in GBM tumor metabolites.

P64

Increased Proportion of Tc17 and Th17 Cells and their Significant Reduction after Thymectomy may be Related to Disease Progression in Myastenia Gravis

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Myasthenia gravis (MG) is an autoimmune disease mediated by auto-antibodies against the neuromuscular junction. The thymus has an important role in the pathogenesis of MG because most of the patients have thymic pathology and thymectomy (TE) can reduce the severity of the disease. In the present study, the frequency of Th17 and Tc17 cells were studied in MG patients (pre and 6 months post-TE) and healthy controls. We recruited 12 MG patients from the Shariati Hospital, Tehran, Iran, and the Alzahra Hospital, Isfahan, Iran, and 12 age- and sex-matched HC from the outpatient service of our institution (Department of Immunology, Isfahan Medical School, Isfahan, Iran) from April 2016 to May 2017. The frequency of Tc17 cells in pre-TE patients was significantly higher than HC ($p < 0.05$) and after thymectomy Tc17 cells significantly decreased compared to the pre-TE ($p < 0.05$). The frequency of Th17 cells in pre-TE patients was significantly higher than HC ($p < 0.05$) and after thymectomy Th17 cells significantly decreased compared to the pre-TE ($p < 0.05$). Our findings indicated a possible role of Tc17 and Th17 in MG pathogenesis.

P65

Speech Recognition Based on Bbrain Signals by the Quantum Support Vector Machine for Inflammatory Patient ALS

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People communicate with each other by exchanging verbal and visual expressions. However, paralyzed patients with various neurological diseases such as amyotrophic lateral sclerosis and cerebral ischemia have difficulties in daily communications because they cannot control their body voluntarily. In this context, brain-computer interface (BCI) has been studied as a tool of communication for these types of patients. In this study, the reliability of electroencephalography (EEG) signals in discriminating between different covert speech tasks is investigated. Twelve participants, across two sessions each, were asked to perform multiple iterations of three differing mental tasks for 10 s each: uncons trained rest or the mental repetition of the words “no”, “yes” and “rest” A Quantum Support Vector Machine was used to classify all three pairwise combinations of “no” or “yes” and “rest” trials and also for ternary classification. In Results An average accuracy of 0.94% ± 2.6 was reached in the classification of covert speech trials versus rest, with all participants exceeding chance level (0.95%). The classification of “no” versus “yes” yielded an average accuracy of 0.93 ± 0.6 with ten participants surpassing chance level (0.95). Finally, the ternary classification yielded an average accuracy of 0.93% ± 0.4. with all participants exceeding chance level (0.96%). The proposed QSVM algorithm provided significantly higher accuracies compared to some of the most common classification techniques in BCI. To our knowledge, this is the first report of using QSVM for the classification of EEG covert speech across multiple sessions. Our results support further study of covert speech as a BCI activation task, potentially leading to the development of more intuitive BCIs for communication.

P66

A New Approach for a Rodent Model of Pos t-Traumatic Stress Disorder (PTSD)

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introduction:One of the most important chalenges in s tuding the anxiety disorders like PTSD is ethical limits in order to make the animal anxious. Sometimes this anxiety should last for a long time such as rodent models of PTSD, and this needs aggressive proceses on rats.We found a new approach for a rodent model of PTSD, wich seems to be more efficient and ethical. Materials and Methouds: 36 adult male Wistar rats weighing 200±20 were divided into two groups of experimental and control. The experimental group were exposed to a male adult cat for 5 minutes, one by one. The cat was kept hungry for 14 hours and the rat`s cage was smeared up with cat`s food. the control group have not been exposed to the cat. After 7 days, the EPM and the Open-field tes t was performed and the blood samples were sent to laboratory for corticosteroid tests. Results: The results of the EPM test in conjunction of the open-field test showed that the anxiety in the experimental group was significantly higher than the control group. The cortisol level was also significantly higher in the experimental group. Conclusion: In this study we showed that long-lasting manifestations of PTSD such as increased anxiety and higher cortisol can persist by only a single

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5 minuetes cat exposure, which is a significantly shorter time in comparison to the previous methods.

P67

The Effect of Melatonin on EDSS and Fatigue in Multiple Sclerosis

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Multiple Sclerosisis (MS) is a common demylinating disease of CNS that inflammation and stress oxidative processes have an important role in clinical courses and progression of it. Suppressing the antigen-presenting capacity of glial cells seems a convinient way of reducing inflammatory activity in MS. The aim of the present s tudy was to determine the anti-inflamatory effects of Melatonin on fatigue and EDSS in MS. In a double-blind, placebo-controlled exploratory study, 62 patients with relapsing remitting MS were randomised to oral Melatonin 3 mg or placebo daily for 8 weeks. Scale of Fatigue in MS and disability was studied by assessing FSS (Fatigue Severity Scale) and EDSS (Expanded Disability Status Scale) on weeks 1 and 8. 47 patient in intervention group and 15 patient in placebo group completed the study. In patient, treatment with Melatonin produced a significant decrease in FSS vs placebo patienta.Although in control group،the average of FSS was 35.866 that after intervention، was 41.Because the score of placebo patient is near or upper than 36 ،this s tudy suggested that Melatonin has a significant effect on improving and decreasing fatigue in MS. Also in this s tudy the average of early EDSS was 2.96 in intervention group and 4.86 in placebo group and after treatment with melatonin was 2.8 and 4.53 in intervention and placebo group respectively. This study suggested that melatonin has a weak effect on improvement of disability and EDSS in MS. Our study shows that use of melatonin along side of First-line drugs such as mitoxantrone، IFN – B or glatimer acetate can improve fatigue in patient with MS.

P68

Effects of Saffron Extract and its Active Constituent Crocin on Development of Neuropathic Pain in a Rat Model of Chronic Constriction Injury

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Neuropathic pain is caused by a lesion or disease affecting the nervous systems, and is generally manifested as spontaneous pain, hyperalgesia, and allodynia. Previous study indicate that saffron has anti-inflammatory and antioxidant properties, we investigated whether saffron and crocin, a major constitute of saffron, would infleuence on behavioral responses of pain induced by chronic cons triction injury (CCI). Adult male Wistar rats (200 to 250 g) were randomly assigned into 6 groups: Sham, CCI, CCI+ Saline, CCI+Saffron (30mg/kg) and CCI +Crocin (15) and CCI +Crocin (30). CCI was induced by setting four loose ligatures around the left siatic nerve of the rat. Two weeks after nerve lesion, injections of saline, saffron or crocin were started and continued every 24 hours until the day 26 post-surgery. Pain behavioral responses including mechanical allodynia (von Frey filament tes ting) and thermal hyperalgesia were measured at days 14, 17, 20, 23, 26, and 40 after CCI. CCI induced a long-lasting hyperalgesia to nocious heat and mechanical allodynia. Treatment with saffron and crocin (30 mg/kg) decreased thermal hyperalgesia and mechanical allodynia from the days 20-23 after surgery that lasted until the day 40. The lower dose of crocin (15 mg/kg) only decreased mechanical alodynia from the day 23 after CCI that lasted until the day 40. These findings indicate that treatment with saffron and crocin after CCI may have a therapeutic effect, suggesting that these subs tances may offer new strategies for the treatment of this highly debilitating condition.

P69

Effect of curcuma longa (Curcumin) in Neuropathic Pain Behavioral Response to CCI Model in Rat

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Treatment of neuropathic pain is still a major challenge because of its noresponsiveness to most available pharmacotherapy. Curcumin has been reported to play an active role in the treatment of various neurological disorders, such as neuropathic pain. In this study, the role of curcumin has been evaluated in pain behavioral responses of chronic Constriction injury (CCI) in rat. In this experimental study male Wistar rats (200–250 g)

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were used. Animals have been categorized as random based on groups of CCI, CCI with vehicle injection and CCI with 30 and 60 mg/kg Curcumin injection. For induction of CCI, Bennett & Xie (1988) method has been used applying CCI by 4 loose ligatures. Fourteen days after creation of neural injury, IP Injection of Vehicle and Curcumin have been started and continued to 26th day as daily. Animal behavioral responses have been measured using mechanical allodynia (Von Frey) and thermal hyperalgesia in 14 to 40th days After CCI. Data analyzed by one-way ANOVA and tukey test. Results indicated that CCI increases pain behavioral responses as significantly (P<0.05). Curcumin injection (30mg/Kg) leads to decrease of mechanical allodynia from 20th day and thermal hyperalgesia from 23th day. These effects have been continued to 40th day. Curcumin injection (60 mg/kg) leads to decrease of mechanical allodynia and thermal hyperalgesia only in 26th day. Based on findings, probably, curcumin can be effective on resulted neuropathy pains of CCI.

P70

Comparison of Hemp Seed Oil Effect on Expression of Cannabinoid Receptor 1 and 2 in Experimental Autoimmune Encephalomyelitis

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Multiple sclerosis (MS) is an autoimmune disease of central nervous system with inflammatory basis. Experimental autoimmune encephalomyelitis (EAE) is common animal model used in experiments the mos t for investigating the multiple sclerosis due to many similar aspects. Hemp seed oil possess potential anti-inflammatory properties. In our research we investigated and compared the effect of hemp seed oil containing natural cannabinoids and poly unsaturated fatty acids on expression of cannabinoid receptors (CB) 1 and 2. Female C57bl/6 mice in three groups (8 in each) randomly allocated as follows: non-EAE (A), EAE treated with hemp seed oil (B) and EAE control (C). After one week of acclimatization in circadian rhythmic standard experimental condition mice were immunized, save group A. The day before induction (day zero), ip administration of hemp seed oil initiated and continued for 28 days. Clinical score and weight was recorded by a blind expert through the study and analysed by SPSS and ML-win where P value <0.05 considered statis tically significant. Findings demonstrated a significant difference in clinical scores in group B compared to C (p values < 0.001). Moreover, expression of both CB1 and CB2 promoted significantly in group B in comparison to C (all p values < 0.001). This increase in CB2 expression was statistically more significant than CB1 expression. Numerous immunoregulatory, anti-inflammatory and anti-oxidant properties of hemp seed oil appraised for its poly unsaturated and/or essential fatty acids, anti-oxidants, vitamins and cannabinoids containment. Here in, hemp seed oil diminished clinical debilities probably through reduction in inflammation as confirmed by disease score descend. Furthermore, activation of CB1 and CB2 expression suggest pivotal role of these receptors in disease control.

P71

The Effect of Pretreatment with Ibuprofen and Dexamethasone on the Effect of Nerve Block Lower Alveolar Marked in Irreversible Pulpitis Teeth

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Successful local anesthesia is the bedrock of pain control in endodontics. Pain control is essential to reduce fear and anxiety associated with endodontic procedure. It is belivied that premedication with non-streroidal anti–inflammatory (NSAID) drugs before administrating inferior alveolar nerve block (IANB) increases the success of anesthesia. The purpose of this study was to evaluate the effect of premedication with dexamethasone (SAID) and ibuprofen (NSID) on frequency of success of IANB in patients with symptomatic irreversible pulpitis. 75 emergency patients in severe pain diagnosed with symptomatic irreversible pulpitis of a mandibular posterior tooth randomly divided in 3 groups. Patients received, in a double-blined manner, identical capsules of either 4mg dexamethasone or 400mg ibuprofen or starch powder 1 hour before the administration of the IANB. Access was begun 15 minutes after the completion of IANB. Success was defined as no or mild pain (Visual analogue scale recording) on access. The success rate for the IAN block was 80.8 % for the dexamethasone group and 73 % for the ibuprofen group, with no significant difference (P=0.337) between the 2 groups. The success rate was 38.5 % for placebo group that significantly lower than dexamethasone and ibuprofen groups (P=0.008). Preoperative administration of 4mg dexamethasone or 400mg ibuprofen 1 hour of the IAN block result in a significant increase in anesthetic success in mandibular

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molars with sympthomatic irreversible pulpitis.

P72

Hesperetine Nanoparticles Ameliorate Glial Activation and Reduce Demyelination Llevel of Rat Optic Chiasm in Lysolecithin-Iinduced Demyelination Model

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Multiple sclerosis (MS) is one of the most autoimmune neurological and inflammatory disease in worldwide. Demyelination and disturbance of action potential conductance are regarded as main signs of MS disease. Hesperetin (Hst) is one of the flavonoid that have neuroprotective properties. The present study attempts to evaluate the effects of hesperetin or its nanoparticle on myelin repair and glial activation in lysolecithin (LPC) -induced demyelination model. Local demyelination was induced by administration of LPC (1%, 2µL) into the rat' optic chiasm. Animals have received oral administration of Hst or nano-Hst at dose of 20 mg/kg for 14 and 21 days post lesion. Visual evoked potential (VEPs) records were performed on days 0, 7, 14 and 21 post lesions. Immunostaining against Iba 1 (microglia marker) and GFAP (astrocytes marker) were carried out for evaluation of myelination and astrocytes activation. Electrophysiological evidence emphasize that oral administration of hesperetin and nano-hespretin could reduce the P1-N1 latency and increase the amplitude of VEPs waves compared to the saline and Hst groups ($p \leq 0.001$). Immunostaining showed that myelin repair was improved in animals which have received nano-Hst treatment; In addition, nano-hesperetin and its nanoparticle effectively reduced the expression of GFAP in optic chiasm ($p \leq 0.001$). The extent of demyelination was reduced in animals under treatment of hesperetin) $p \leq 0.01$ (or nanohesperetin($p \leq 0.001$). Our results showed hesperetin and nano-hesperetin treatment significantly enhances myelin restoration through endogenous sources of glial progenitor cells following local injection of LPC.

P73

Nano-Phytosome of Curcumin Improve Behavioral Impairment on Carrageenan-Induced Acute Inflammation Model in Mice

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Inflammatory disorders alone or as a consequence of neurological disease affecting patients in life. Experimental models of inflammation are use to evaluate the production of inflammatory mediators at site of inflammation. Curcumin is one of the flavonoids possesses potent anti-inflammatory activity. However, because of low water solubility curcumin, its clinical application has been limited. The present study attempts to assessment the effects of curcumin and nano-phytosome of curcumin on improve of behavioral impairment and reduce inflammation cytokines in carrageenan-induced inflammation model. Animals have received oral administration of curcumin or nano-phytosom of curcumin at dose of 15 mg/kg for 7 days before injection of carrageenan. Acute inflammation was induced by injection of carrageenan (1%,) into the subplantar region of left paw in mice. Tail pinch test and hotplate test (for evaluated the threshold of neuroinflammation pain) were performed on ½ h before injection and ½ h, 2 h, 24 h after injection of carrageenan. The results of behavioral testes showed enhance of antinociceptive effects in the animals recieved curcumin($p \leq 0.01$) and nano-phytosome of curcumin ($p \leq 0.001$) compaired to other groups. These results suggested that curcumin and its nano-phytosome improvement behavioral impairment and reduce inflammation cytokines following local injection of carrageenan.

P74

Low-Intensity Aerobic Training for Along with Blood Flow Restriction on Amount of Protein BDNF in Soleus and EDL Muscles as well as the Sciatic Nerve in Aged Male Rats

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Neurotrophins are a group of nerve growth factors with their protein structure. These proteins play an important role in the growth and metabolism of many cells and maintain nerve, muscle, synaptic potentials, and also helps reduce depression and apoptosis. The aim of this s tudy was to evaluate the effect of 10 weeks low-intensity aerobic training and limited blood flow to the amount of protein neurotrophic factor derived from the brain (BDNF) in the soleus, extensor long fingers muscles (EDL) as well as sciatic nerve in rats the elderly. 60 elderly male Wistar rats (23-24 months) were chosen which their weigh were 355 to 481 grams. They were divided into 6 groups randomly, blood flow restriction (BFR), exercise with blood flow restriction (BFR + Ex), sham (Sh), sham with exercise (Sham + Ex), control (Ctl) and exercise (Ex). They were trained with a low aerobic exercise (15 m / min) for 1 hour, 5 days a week for 10 weeks then were sacrificed 48 hours after the las t training session. The samples of muscle and the sciatic nerve were separated immediately and they were put in a solid nitrogen. and Then, they were preserved at -80 ° C. The protein samples were detected by Western Blotting method. The statistical analysis was conducted with SPSS software version 18. Data normal distribution determined by Shapiro – Wilk test. Comparisons were performed among different groups by one-way ANOVA and post hoc Tukey's test. P values less than 0.05 were considered as statistically significant. The results have shown that BDNF protein just decreased in the EDL muscle for BFR + Ex group compared with Ctl and Sh groups ($P<0.05$) significantly. In addition, in soleus muscle BFR + Ex group decreased in compare with all groups (groups Ctl, Sh and BFR $P< 0.001$ and Ex and Sh + Ex ($p<0.05$) significantly. moreover, Ex, Sh + Ex groups in comparison with Ctl and Sh have decreased significantly ($P< 0.001$). On the other hand, the sciatic nerve of BFR + Ex group, in comparison with all groups, increased significant ($P< 0.001$). This study has indicated that this type of exercise can effect on the BDNF protein in slow and fast witch muscles. Furthermore, BFR+Ex, as a training method, can influence on nerve and muscle positively.

P75

Expression of GDNF Genes in the Cerebellum of Rat Neonate Born to Mother with Diabetes

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Diabetes Mellitus as a common metabolic disorder in women of reproductive age is rising throughout the globe. Diabetes in pregnancy has various adverse outcomes on different organs development including the central nervous system (CNS) and it can cause learning deficits, behavioral problems and motor dysfunctions in the offspring. The cerebellum is a part of brain that coordinates voluntary movements and controls balance and also participate for motor learning, and language processing. Neurodevelopmental assessment of the child born to diabetic mothers has displayed a short and long-term neurocognitive and neurobehavioral abnormalities in the offspring. Even though neuronal death has also known the main leading cause of diabetic CNS and peripheral neuropathies, the exact mechanism of neuronal death in diabetes type I mellitus has not been completely understood yet. Neurotrophic factor family (NTFs) consists of: nerve growth factor (NGF) family, glial cell line-derived neurotrophic factor family ligands (GDNF) and some cytokines. Several processes in neuronal cells such as survival, migration, neurite outgrowth, formation of synapses and neuronal plasticity are controlled by NTFs. In the majority of experimental studies the important role of GDNF and its receptor components (GFRα1 and Ret) in the survival of different populations of neurons in the central and peripheral nervous systems have been proved. Beside the expression of GDNF in developing Purkinje cell and granule cell layers in many researches has shown. In spite of the fact that subsequent studies demonstrated molecular layer interneuron (MLIs) are essential for normal cerebellar function and motor learning, signals controlling survival and mechanisms migration of Purkinje cells (PCs) from the ventricular zone to form the PC plate during embryonic development of the cerebellum are incompletely unknown. In previous studies reported that the neurotrophic receptor GFRa1 is transiently expressed in developing PCs and loss of GFRa1 delays PC migration. Regarding above mentioned facts, since cerebellum is one of the important parts in the brain for memory/learning processing; on the other hand expression GDNF and GFRα-1 is essential for the development and PCs migration, this study aimed to investigate mRNA expression and distribution pattern of GDNF in different layers of cerebellum in early pos tnatal development in diabetics' rat off springs.

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P76

Comparison the Effect of Coriandrum Sativum and Salvia Officinalis Hydroalcoholic Extract on Learning and Memory in Mice; the Role of oxidativeS

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In this study, the efficacy of Coriandrum and salvia extracts in spatial and passive avoidance memory in mice was studied. 56 male mice (25-30g) divided randomly into 8 groups: negative control, positive control and extract administered (50, 100 and 200 mg/kg of both plants), and treated for 25 days. negative and positive control group received normal saline and Ritalin respectively. After training, passive Avoidance memory was measured with shuttle box and spatial memory was measured with Morris water maze and Y maze, twenty-four hours and one week after the last injection. Finally, blood samples were collected, MDA and SOD in serum were measured using ELISA kits of Zell Bio, Germany. Data were analyzed by using ANOVA and Tukey test. The $P<0.05$ was considered significant. Data analysis showed both Coriandrum and salvia hydroalcoholic extract(200mg/kg) improved passive avoidance memory ($P<0.01$) and spatial memory ($P<0.05$) in mice in compared with control groups.after one week this effect was seen in salvia but wasn't seen for Coriandrum.and in both group (100 and 200mglkg) amount of MDA was significantly. decreased, but no significant change was observed in the SOD content between them. It seems that the beneficial effects of these plants on memory related to oxidative stress indices and reducing the level of lipid peroxidation of the serum but other mechanisms maybe have effect in longer efficacy in salvia rather than Coriandrum and more studies is needed to determine the mechanism this effect.

P77

NADPH Oxidase Type 4 Inhibits Immune Cell Trafficking into The Central Nervous System During Neuroinflammation

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Transendothelial trafficking of immune cells into the central nervous system (CNS) and disruption of the blood brain barrier (BBB) are pathophysiological hallmarks of neuroinflammatory disorders like multiple sclerosis (MS). Accumulating evidence suggest that oxidative s tress plays a major role in the pathogenesis of MS, whereas a specific influence of oxidative stress on BBB dysfunction in MS was unclear so far. Here, we identify NADPH oxidase type 4 (NOX4) as a specific and direct modulator of BBB integrity. Deficiency of NOX4, but not NOX1 or NOX2, rendered mice more susceptible to experimental autoimmune encephalomyelitis (an animal model of MS) and was accompanied by a remarkable enhancement of BBB disruption and CNS inflammation. Murine and human in vitro analysis revealed that lack of NOX4 amplifies leukocyte trafficking by modified endothelial cells. Further, reduced endothelial NOX4 expression was found in CNS tissue of individuals suffering from MS indicating an important role of NOX4 also in humans. Our study demonstrates, for the firs t time, that NOX4 is an important and direct regulator of BBB integrity. NOX4 activation can decrease BBB damage and cell invasion during neuroinflammation and may offer a novel strategy for the treatment of MS.

P78

Depression and the Relation of These Symptoms with Plasma Cortisol Level in Individuals Dependent and Independent to Methamphetamine, a Cross-Sectional Study

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Methamphetamine is a psychostimulant drug that is highly addictive and causes epigenetic changes that is associated with symptoms of depression. The aim of the present study is comparing age and gender differences in individuals dependent and independent to methamphetamine and examining the relation of depression with the level of cortisol. In a cross-sectional study, 55 methamphetamine users with diagnosis of depression (29 men and 26 women) and also 65 non-users depressed patients (30 men and 35 women) among who referred to three stimulant treatment centers in Tehran were selected using purposeful sampling method and were assigned into four groups. Evaluating the level of plasma cortisol hormone was done using radioimmunoassay method (RIA) and depression symptoms were evaluated using Depression Ques tionnaire. Data were analyzed using chi-square test,

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multivariate variance analysis and Pierson correlation. The results showed the level of cortisol in two groups of male and female Meth users was higher than two groups of non-users (P<0.05). Also, there is a significant relationship between the level of cortisol and depression in Meth users (P< 0.05). This relationship is stronger in women than men. Also, age index in users had lower than two groups of non-users (P<0.05). The findings of the present study can be useful in the process of preventing and treating addiction. Using chemotherapy in creating changes in cortisol levels with the aim of controlling usage relapse can be an appropriate path for future researches in this field.

P79

The Efficacy of Cefazolin Plus Macrolide (Erythromycin or Clarithromycin) Versus Cefazolin Alone in Neonatal Morbidity and Placental Inflammation for Women with Preterm Premature Rupture of Membranes

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Although the use of broad-spectrum antibiotics in women with preterm premature rupture of membranes (PPROM) is recommended to prolong pregnancy and decrease short-term neonatal complications, the optimal regimen remains undetermined. The objective of this study was to compare the efficacy of cefazolin plus macrolide (erythromycin or clarithromycin) versus cefazolin alone in reducing neonatal morbidity and placental inflammation for women with PPROM. This prospective study included singleton pregnancies with PPROM (23-33 weeks gestation). The primary outcome was neonatal composite morbidity and the secondary outcomes were the incidence of abnormal brain sonography and infant neurological outcome at one year of age. The presence and the stage of acute histological chorioamnionitis and funisitis were also reviewed blinded to all clinical information. 120 women were randomly assigned to cefazolin (n = 40), cefazolin plus erythromycin (n = 40), or cefazolin plus clarithromycin (n = 40). The neonatal composite morbidity, the incidence of abnormal brain sonography, and infant neurological outcome at one year of age were similar between the comparison treatments (combination of cefazolin plus erythromycin or clarithromycin) and cefazolin. However, the presence and stage of histological funisitis showed significant difference between cefazolin plus clarithromycin versus cefazolin alone (p = 0.314). This study is the first clinical trial of the use of cefazolin with either clarithromycin or erythromycin compared to cefazolin alone in the management of PPROM in which the primary and secondary analyses showed no difference among the three antibiotic regimens. The only noted difference was from a lesser degree of histological funisitis associated with clarithromycin exposure. Our data suggests that clarithromycin may be an alternative worth considering with potentially beneficial effects compared to erythromycin in PPROM.

P80

The Effects of Progesterone Receptors' Antagonist RU-486 on BrainEdema, Intracranial Pressure and Neurological Outcomes after Traumatic Brain Injury

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In previous studies, the neuroprotective effect of proges trone in diffuse traumatic brain injury has been shown. This study used mifepristone (RU-486), a potent proges terone receptor antagonist, to evaluatethe hypothesis that the neuroprotective effect of progesterone in traumatic brain injury is mediated by the progesterone receptors. The ovariectomized rats were divided into 6 groups. Brain injury was induced by Marmarou's method. Progesterone was injected 30 minutes after traumatic brain injury, and RU-486 was injected before traumatic brain injury and also before proges terone treatment. The brain water content(BWC) and Evans blue dye content (EBC) were measured 24 and 5 hours after traumatic brain injury, respectively. The neurologic outcomes and intracranial pressure (ICP) were assessed before, 4, and 24 hours after traumatic brain injury. BWC and EBC were less in progesterone -treated group comparison to vehicle group. RU-486 eliminated the effects of progestrone on brain edema and blood brain barrier permeability. ICP was increased significantly after trauma, and progesterone decreased intracranial pressure at 4 and 24 hours after traumatic brain injury in comparison to vehicle. This inhibitory effect was also eliminated by treatment with RU-486. RU-486 also inhibited the progestrone induced increase in neurologic outcomes following traumatic brain injury. The results suggest that a genomic pathway of progesterone receptor have probably a role in the neuroprotective function of progesterone following traumatic brain injury.

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P81

Detection of Epileptic Seizures Using EEG Signal Processing

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Epilepsy is the most common brain diseases that cause many problems in the daily life of the patient. In most attempts to automatic detection, the attack used an EEG. In this paper, The complete data set consists of five sets recorded from normal and epileptic patients. Each set containing 100 single-channel EEG segments. Here we used first and last sets (A and E). Set A consisted of segments recorded from healthy subjects while they were relaxed in an awake state with open eyes While set E contained seizure activity taken from patients whom had the resection of one of the hippocampal formations and were under seizure control the empirical mode decomposition method used to analyze the random signals better than previous methods. The feature used for classification have been obtained from EEG signal decomposition into frequency sub-bands by Discrete wavelet transform (DWT) for decreasing dimensions of DWT coefficients, some statistical features are calculated for each frequency sub-bands. we compared the classification results of using reduction technique, each frequency sub-bands. Experimental results show that the proposed method can serve as a promising alternative for Automatic diagnosis system in the future. In this paper, we designed a computer diagnostic sys tem that helps experts to increase the accuracy of the diagnosis of the epilepsy.

P82

Comparison of the Effect of Stress & Exercise on Plasma Corticosterone & Hippocampal Apoptosis in the Female PTSD Rats

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PTSD is a condition that develops after an individual has experienced a major trauma and associated with inability to extinguish these fear memories, increase anxiety behaviors & hippocampal atrophy, HPA axis dysfunction & increase the risk of neurodegenerative disease. Studies showed that apoptosis is a neurodegenerative process that has important role in that ,although volume & function of hippocampus reduce by the chronic effect of glucocorticoids. Hippocampal s teroid receptors control the HPA axis. Glucocorticoids involve in stress response, they also have activity in immune system ,control of physiologic & basal level of metabolism and memory consolidation. The SSRIs have been considered as a first-line medication choice but the response of rates rarely exceed 60%. Exercise is advocated as a behavioral intervention to alleviate neurological deficits. Adult female Wistar rats divided to SPS & sham groups. We used to (SPS) model as an animal model for PTSD. After 14 days SPS & sham group rats divided to two sub groups: 1) Exercise group: moderate treadmill exercise for 4 weeks & 5 days in each week. 2) Control group without any intervention. At the end of the intervention, rats from each group were decapitated, trunk blood was collected & serum was used for the corticosterone assay. The hippocampi were also removed and the mRNA expression of pro-apoptotic (Caspase 3) proteins was determined by using RT-PCR method. Statistical analysis Showed that corticosterone levels significantly increased in PTSD rats and exercise can alleviated this factor in these animals, expression of Caspase 3 mRNA increased in the SPS rats, and exercise reduced expression of Caspase 3 mRNA in this animals. This study have shown that moderate treadmill exercise can be reduce the rate of apoptosis in hippocampus PTSD rats by regulating corticosterone secretion.

P83

Central Neuropathic Pain Development in Experimental Autoimmune Encephalomyelitis C57BL/6 Mouse Model Induced by QS-21 Adjuvant

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Central neuropathic pain (CNP) is considered as a complicated sensory disturbance which many multiple sclerosis (MS) patients suffer from. Although monophasic experimental autoimmune encephalomyelitis (EAE) mouse model is a gold standard model in preclinical

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research of MS, severe movement deficit could confound pain behaviors evaluation over the disease course. In this study, complete Freund's adjuvant (CFA) was subs tituted with an acylated triterpene glycoside saponin adjuvant named quillaja saponin-21 (QS-21) to establish EAE model for CNP development. Twentyfour, 5-7 weeks old female C57BL/6 mice were randomly divided into three groups. Two groups immunized with MOG35-55 peptide emulsified with CFA and QS-21 adjuvant. The last group received PBS as negative control group. Thermal hyperalgesia as a CNP clinical manifestation through hot plate test and clinical signs were assessed for 60 days' post immunization (p.i). On days 21 and 60 p.i mice were sacrificed and TCD4+, TCD8+, IL-17+ cells in total splenocytes population by flow cytometry and lymphocyte infiltration and demyelination of brain samples by histopathological staining were evaluated. EAE was established in MOG+QS-21 and MOG+CFA groups as mild relapsing-remitting and monophasic models, respectively. Thermal hyperalgesia developed in the bilateral hindpaws on the onset of clinical symptoms in MOG+CFA and MOG+QS-21 groups and it was maintained until study completion in MOG+QS-21 group. TCD4+, TCD8+ and IL-17+ cells population in MOG+QS-21 and MOG+CFA groups increased significantly ($P<0.05$) on days 21 and 60 p.i compared to PBS group. Although, inflammatory cells infiltration were increased significantly on days 21 and 60 in MOG+QS-21 and MOG+CFA groups, however demyelination was seen on days 21 and 60 only in MOG+CFA group compared to PBS group ($P<0.05$). QS-21 adjuvant is capable of establishing mild relapsing-remitting EAE model for CNP development with severe neuro-inflammation and no significant demyelination in white matter.

P84

Effect of Insulin-Like Growth Factor 2 (IGF2) as a Microglia-Derived Anti-Iinflammatory Ccytokine on Improving Memory Impairment Following Hippocampal Intracerebral Hemorrhage in Rat

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Insulin-like growth factor 2 (IGF2) as a microglia-derived anti-inflammatory cytokine has a pivotal activity in memory consolidation. However, there is limited evidence on brain cell-originated IGF2 expression, regulation and function in pathological condition and neuro-inflammation. Hence, the present study was conducted to investigate the effect of IGF2 on improving the memory impairment in a rat model of hippocampal intracerebral hemorrhage. 24 male Sprague Dawley rats randomly assigned into three groups. To stablish a rat model of intracerebral hemorrhage, 100 µl of blood autologous was injected into the left hippocampus. The animals received intrahipocampally the IGF2 upon 30 minutes after injecting the blood, followed by tes ting for behavioral parameters, including neurological deficit score, passive avoidance test, wire hanging tes t and novel object recognition at two weeks after the injection, then hippocampus volume was estimated using the Cavalieri method. The result indicated that retention and recall capability improved, was IGF2 injected into hippocampus compare with control group ($P<0.05$). Also, neurological deficit score significantly increased following IGF2 injection ($P<0.05$), but there was no significant difference in locomotor deficits measured by wire hanging test between groups. Moreover, hippocampal volume increased ($P<0.01$) and infract volume decreased ($P<0.01$) in IGF2 group compared to the control group. Our results showed that the IGF2 injected into the hippocampus promoted learning and memory and also IGF2 as a microglia-derived anti-inflammatory had a positive influence on infract volume resulted from brain secondary damage and neuroinflammation after ICH.

P85

Association of Disability with Urinary and Sexual Dysfunction in Patients with Diagnosed Multiple Sclerosis

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Multiple Sclerosis (MS) is one of the most common diseases of the central nervous system it is often seen in adults who may be sexually active. This disability can be associated with sexual-urinary problems that affect all aspects of their lives. The aim of this study is to evaluate the association between disability and urinary-sexual dysfunction in women with MS. This study is a descriptive-correlation study of 78 women

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with MS who were recruited with the method of "access based on target .included participants were 78 women suffering from MS who referred to Neurology clinic of Ghaem Hospital 2013.Data were collected using data information sheets for demographic variables and urinary parameters. Krutzke scale for measuring developed disability and female sexual function index (FSFI) were also used to collect information. Data were analyzed using descriptive statistics and appropriate s tatistical methods and SPSS -19 were used the p-value of (0.05) was considered statistically significant. Patients had participated mild disability (52.6%) and moderate (47.4%). The most and the least prevalent parameters were satisfaction domain and pain sexual with the frequency of (43.6%) and (10.3%), respectively. The most and the least prevalent parameters were urinary frequency and urinary incontinence with the frequency of (30%) and (2.5%), respectively. Pearson's correlation reported meaningful negative linear correlation between the levels of disability with sexual dysfunction. (r=-0.61 P=0.001). Based on regression analysis, it was determined that the level of disability, the high age, the years of diagnosis and the type of Relapsing-Remitting disease had the greatest effect on sexual dysfunction. The prevalence of urinary – sexual dysfunction in multiple sclerosis affects all aspects of life, so the evaluation should focus more on these disorders and provide context for the implementation of rehabilitation programs for patients.

P86

Investigation of the Relationship between ZNF804A and DISC1 Polymorphisms in RS61886494 and RS12133766 Positions in Schizophrenic Patients

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Schizophrenia is a mental disorder characterized by abnormal social behavior and failure to understand reality. The aim of this study was to determine the relation between FOLH1 and DISC1 genes polymorphism in patients with schizophrenia in Iran. In this study, 50 patients with schizophrenia and 50 healthy controls were evaluated. PCR-RFLP was used for FOLH1 gene and Tetra-ARMS for the DISC1 gene for evaluation of single nucleotide polymorphism in both groups of patients and control. For enzymatic digestion of PCR products, the positions of RS61886494 and RS12133766 were used for enzyme MseI and BseLI and incubated for 37 hours at 16°C. The frequency of CC, CT and TT genotypes for FOLH1 gene in RS61886494 region was 92%, 8%, and 0%. In the DISC1 gene, the frequency of GG, GA, and AA genotypes in the RS12133766 region was 84%, 8%, and 8%. For FOLH1 gene in RS61886494 region, the frequency of CC and TT genotypes in patients was 2% and 6% lower in healthy people, while the CT genotype in patients was 8% higher in healthy people. Interes tingly, TT genotype was not observed in patients and healthy genotype CT was not observed. Regarding the DISC1 gene, the results showed that the frequency of GG and AA homozygote genotypes in the patients was higher in the RS12133766 region when the heterozygote GA was high in healthy people and was not observed in patients with this heterozygote.

P87

The Role of the Long Non-Coding RNA Sequences (LncRNAs) in Neurological Disorders

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Precise interpretation of the transcriptome sequences in the several species showed that the major part of genome has been transcribed; however, just a few amounts of the transcription sequences have open-reading frames which are conversed during the evolution. So, it is unlikely that many of the transcribed sequences code the proteins. Among the all human non-coding transcripts, at least 10000 are approximated to be less 200 nucleotides and are considered as long non-coding RNAs. Overall analysis of the mammalian transcriptome shows that long non-coding RNAs may form the large part of the cellular transcripts. In recent years, there had been an increase in researches for determining the role of lnRNAs at the development and disease. Studies have showed that lncRNAs play an important role in controlling the development of the central nervous system (CNS). Brain development of higher vertebrates is associated to the increase in the levels and complexity of lncRNAs in the developing nervous system. It is known that limited rates of expressed lncRNAs in CNS are important for the neuronal differentiation. Conclusion: LncRNAs contribute in creating neurological disorders by playing a role in mRNA splicing. Advancement in the sequencing technologies and promotion of the useful non-coder elements lead to the rapid detection of the expressed lncRNAs in the vertebrates and invertebrates. Systematic interpretations of the time and spatial pattern of expression in the developing nervous system provide a background for the hypotheses which are related to the lncRNA function.

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P88

The Relationship between the TNFα of the Microglia Cells and the Parkinson Disease

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TNFα is one of the inflammatory cytokines which plays an important role in activating the Caspase and inducing the apoptosis. This cytokine which is secreted in inflammations, induces the NO production in macrophages and leads to differentiation of the macrophages to the epithelioid and giant cells. One of the most important factors which stimulates the TNFα secretion, is LPS and that antigen (LPS) is the mitogen of the Ti-1 in the B lymphocytes and as a result, many of macrophages such as microglia cells proliferate. Microglia is the smallest non-neuronal cells in the CNS and responds to the inflammations in the CNS. One of the factors in developing the Parkinson disease is TNFα which is secreted of the microglia cells and des troys the dopaminergic neurons in substantia negra. In Parkinson disease, microglia cells leave the resting form and achieve the plastic amoeboid morphology (In the resting form, microglia cells have homeostatic roles and eliminate the neurotoxins and dead cells). Finally, the levels of the Caspase 3 and 8 increase and the condition is prepared for the apoptosis of the neuron and in both chronic and acute forms, the levels of the TNFα increase in the cerebrospinal fluid (CSF).

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The Relationship Between the TNFα of the Microglial Cells and the Multiple Sclerosis

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TNFα is an inflammatory cytokine and the caspase and apoptotic processes can be activated by TNFα There are two classes of TNFα (solTNF and tmTNFα) which are important from the receptor aspect, so that solTNFα and tmTNFα can attach to the TNFR1, but tmTNFα jus t attaches to the TNFR2. Microglia cells are resident immune cells of the central nervous system (CNS) and they respond to the injury and infection and also remove the cellular debris. In the neurological disorders, the microglia cells are activated and they secrete cytokines such as TNFα Some neurological disorders are associated to the TNFα of activated microglial cells and one of the common diseases is multiple sclerosis (MS). The TNFα which is secreted by microglial cells, functions through various mechanisms and causes the multiple sclerosis. One of the ways would be that TNF increases the caspase 3 and 8 and stimulates the neuronal destruction. Furthermore, this cytokine changes the levels of the proteins such as tau protein and impairs the synaptic formation. In this article, we will investigate the association between this cytokine and MS.

P90

The Relationship between the TNFα of the Microglia Cells and the Alzheimer Disease

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The Neuroscience Journal of Shefaye Khatam, 2018; 6(S1): P90

TNFα is one of the most important inflammatory cytokines which induces caspase and apoptotic process. There are two forms of TNFα: soluble TNFα (solTNFα) and transmembrane TNFα (tmTNFα). These inflammatory cytokines have two receptors namely TNRF1 and TNRF2. The attachments of the cytokines to those receptors induce the TRAF and activate the NK-Kb. TNFR1 is found in most of the cells and solTNFα and tmTNFα have the ability to attach to the TNFR1, but the TNFR2 is just found in the microglia and endothelial cells and reacts with the tmTNFα. Microglia is one of the smallest cells in the CNS which has the ability of phagocytosis and is one of the first cells which responds to the inflammations and exogenous factors. In general, there is a balance between the levels of the solTNFα and tmTNFα, but in the CNS disorders, in according to the damages in the neurons and disturbances in the cell conditions, that balance is interrupted and one of the cytokines is secreted more than normal level. One of the mos t important chronic neurodegenerative damages that is stimulated by TNFα, is Alzheimer disease. In the Alzheimer disease, activated microglia cells increase the levels of the TNFα which inclines the extracellular b-Amyloid, tau protein, intracellular neurofibrillary tangles and microgliosis that these changes lead to synaptic dysfunctions and neuronal destruction.

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P91

Auto Graft Transplantation of Adult Human Neural Stem Cells in Treatment of Traumatic Brain Injury as a Hypothesis

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Traumatic brain injury (TBI) leading to 5 million deaths annually is 1 of the 5 major causes of morbidity and mortality worldwide. In Iran, accidents are the main cause of death in youth as well as a dominant factor in reducing quality of life. In developing countries TBI incidence as one of the worst consequences of these accidents is growing due to wide use of motor-vehicles. Therapeutic strategies for TBI are limited to supportive care such as reduction of intracranial pressure and maintaining blood pressure. Currently there are no specific effective treatments available. Cell replacement strategies have become a major focus of innovative therapies over the last 15 years. Neural stem cell (NSC) transplantation in treatment of various neurological and neurodegenerative diseases (stroke, TBI, spinal cord injury, brain tumors, Parkinson's disease, etc.) have showed promising effects including promoting tissue regeneration, replacing the lost neural cells and improving functional deficits. Several studies suggest anti-inflammatory and neuroprotective effects of human NSCs derived from human fetus and embryonic stem cells after TBI. However, to our knowledge efficacy of adult NSC transplantation in treatment of TBI has not been studied previously. Transplantation of auto graft adult NSCs derived from the patient's brain can be considered as a potential treatment to minimize TBI side effects and its extensive economic burden to hospitals including emergency department visits, hospitalizations, and utilization of intensive care units. This method has minimal autoimmune responses therefore ample use of immunosuppressant medication is not required.

P92

Boswellia Serrata Ameliorates Neuro-Inflammation Caused by Periodontitis: A Narrative Review

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Neuro-inflammation is the inflammation of nervous tissue due to oxidative stress, aging and autoimmunity and the major cause of Alzheimer's, Parkinson's and Huntington's disease. Periodontitis, the inflammation of the gums and supporting structures of the teeth, is a chronic peripheral immuno-inflammatory condition, affiliated with gram- negative, anaerobic bacteria that cause low-grade systemic inflammation by release of pro-inflammatory cytokines into systemic circulation and activation of microglia; So recent studies advocate for links between neuro-inflammatory diseases and periodontitis; Because neuro-inflammatory diseases begin to develop many years before clinical diagnosis and treating systemic inflammations like periodontitis may have an inhibitory effect on the pathogenesis of neuro-inflammation. Boswellia Serrata, a traditionally used herbal medicine, has anti-inflammatory and antibacterial properties; mainly noticed on Aggegatibacter actinomycetemcomitans, associated with aggressive forms of periodontitis. It can inhibit lipoxygenases, leading to its anti-cancer and anti-inflammation activity and is used to treat asthma and arthritis. Based on previous studies, it has no remarkable side-effects compared to NSAIDs; So it can be a good target for future anti-inflammatory drugs. Neuro-inflammation can be prevented or delayed by treating systemic inflammations like periodontitis. The current procedures for treating periodontitis are not completely efficient and it is important to use alternative and body-friendly, natural treatments. Boswellia Serrata is among natural anti-inflammatory, antibacterial substances which can be used in oral hygiene products, such as toothpaste, mouthwash, dental floss and even chewing gums and drinking water.

P93

Coriandrum Sativum Prevents Neuro-Inflammation by Mitigating Periodontitis: A Narrative Review

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Neuro-inflammation, biochemical and cellular responses of the nervous system to injury, infection or neurodegenerative diseases, is a major risk factor for Alzheimer's, Parkinson's and Multiple Sclerosis. Periodontitis is a serious bacterial gum infection that damages the soft tissue and teeth supporting bones, causing teeth loss. It also renders systemic inflammation by secretion of pro-inflammatory cytokines and microglia activation and subsequently, causes neuro-inflammation. Mechanical removal, systemic anti-infective therapy and local anti-infective therapy are used to treat periodontitis. Based on recent studies, local application of herbal gels has a considerable positive effect on bacterial plaque removing and treating periodontitis. Coriandrum Sativum is a traditionally used medicinal plant. Its extract has tannin that has anti-inflammatory,

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antibacterial and antioxidant properties and is used in cosmetics. Many studies indicate no remarkable side-effects for this herb, compared to synthetic medicaments. Due to decreasing in anti-oxidative and antibacterial activity of saliva during periodontitis, it is important to applicate such herbs to prevent progression of inflammation. Neuro-inflammatory diseases initiate many years before they can be clinically diagnosed. By treating systemic inflammations like periodontitis, Neuro-inflammation is preventable or can be delayed. Current methods used for treating periodontitis are not completely helpful and studies show better results when herbal medicines are used concomitantly. Coriandrum Sativum is one of these herbs to be used in oral-hygiene products such as toothpaste, mouthwash, dental floss and even chewing gums and drinking water.

P94

Nigella Sativa Attenuates Neuro-Inflammation by Alleviating Periodontitis

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Neuro-inflammation is a reaction to brain injury involving the activation of glial cells and release of inflammatory mediators, such as cytokines and chemokines which has a key role in neuro-degenerative diseases. Periodontitis is a microbiome-related dental inflammation caused by gram- negative bacteria that leads to connective tissue destruction and loss of teeth. Previous studies demonstrated that periodontitis can cause neuro-inflammation by inducing systemic inflammation due to secretion of pro-inflammatory mediators and glial cells' activation. Thus, treating periodontitis can prevent or delay neuro-inflammation. Herbal medicines are alternative targets for future periodontitis drugs, due to their less adverse side effects and more efficacy in comparison to allopathic, synthetic drugs. Nigella Sativa (Black Seeds) is a traditionally used herbal medicine, cultivated in South Europe and Middle Eastern Mediterranean region. The seeds of N. Sativa and its active constituent, Thymoquinone, are proved to have anticancer, antidiabetic, antimicrobial and antioxidant traits. N. Sativa is also claimed to have anti-inflammatory effect, by inhibiting of some inflammatory cytokines. Furthermore, it has showed no remarkable toxicity or adverse side effects in previous s tudies. Neuro-inflammation can be prevented by treating systemic inflammations such as periodontitis. Suitable administration of herbal medicines in combination with chemotherapeutic drugs may lead to effective treatment of inflammations and preventing drug resistance. Nigella Sativa is one of these herbs that can be used in oral health products, chewing gums and drinking water. In this case, mental health and a high quality of life is guaranteed.

P95

Targeting of Microglial M1/M2 Polarization through Stem Cells Therapy as a Promising Candidate in Traumatic Brain Injury (TBI)

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Traumatic brain injury is a serious global health problem with irreversible high morbidity and disability and Because of its unknown pathophysiological mechanisms, efficient therapeutic approaches to improve the poor outcome and long-term impairment of behavioral function are still remains lacking. The microglial cells are the resident macrophage cells of the brain and have M1/M2 phenotype, for expression of pro-inflammatory and anti-inflammation cytokines, respectively. The results have been shown that pharmacological inhibiting of M1 phenotype and activating M2 phenotype of microglial cells could relieve cerebral injuries and increase neurological function recovery after Traumatic brain injury. Mesenchymal Stem Cells (MSCs), a type of multipotent stem cells, are regarded as promising therapies in several CNS diseases clinical trials. In animal models, transplantation of stimulated MSCs could promote the activation of microglia via transforming the classic M1phenotype into alternative M2 phenotype to inhibits the release of pro-inflammatory cytokines and raise tissue repair after traumatic brain injury(TBI). In this review, we summarized the beneficial effects of MSCs on TBI damaged tissues and their function in regulating the immune system to maintain the CNS. Although, lab trials studies have also confirmed that MSCs are able to promote positive outcomes in TBI models, however, there are still some unanswered ques tions regarding MSCs-based therapy due to complex ethical and safety bconcerns.

P96

Progress in the Treatment of Alzheimer's Disease by Gene Therapy

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Alzheimer's disease (AD) is a progressive neurological disorder characterized by the aggregation of two proteins, amyloid-b and hyper phosphorylated tau, and by neuronal and synaptic loss. The progress of gene-modified cells and stem cells is a particularly promising therapeutic method for AD. Gene-Modified Cell-Based Therapy for AD prior to transplantation can be beneficial for increasing cell survival and making them more effective. Furthermore, adapted cells could be used for the transfer of factors that can ameliorate neurological complaints. Because of the loss of cholinergic neurotransmitters in AD, some scientists were interested in developing gene-modified cells that can produce acetylcholine (Ach). Primary fibroblast cell line genetically engineered to express choline acetyltransferase to make Ach after transplantation into the hippocampus of rats. Another example for the simplification of gene therapy in AD is the over expression of neprilysine (NEP), an Ab degrading protease that has been exposed to ameliorate extracellular amyloids. Transgenic mice (APP/PS1) injected with lentiviral vector expressing NEP presented a decrease in Ab deposits, and MSCs overexpressing the NEP gene proved the ability to degrade Ab peptides in vitro. Similar results were found in vivo with transgenic mice that were transplanted with primary fibroblasts transfected with a lentivirus carrying NEP. Currently, no treatment has been established that can stop or reverse the development of AD. Though challenges such as immune rejection and cell survivability need to be addressed. The usage of autologous cells from patients for the generation of iPSC or gathering autologous MSCs may circumvent some of these challenges.

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Physical Exercise as an Effective Factor in Alzheimer Disease

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Alzheimer's disease (AD) is a progressive disease that destroys memory and other important mental activities. Scientists have found that remaining relatively active can lead to better brain activities in those at risk of developing AD. In some Meta-analyses of prospective investigations, a significantly reduced risk of dementia related to midlife exercise have been proven. Most s tudies have been performed on animal models about the effects of exercise on brain β-amyloid deposition, showed that the level of amyloid plaques are reducing significantly. In another study about the serum BDNF levels and exercise have recognized major transient increases of circulating BDNF with short-term aerobic exerciseThis information recommend that aerobic exercise is related to a decreased risk of cognitive impairment and dementia. A convergence of data from both human and animal studies proposes that aerobic exercise may reduce development of neurodegenerative processes and age-association loss of synapses. This may occur by a direct effect on neurodegenerative disease procedures or simplification of neuroprotective neurotrophic factors.

P98

Vitamin-D Deficiency as a Potential Risk Factor in Multiple Sclerosis

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Multiple sclerosis (MS) is a multifactorial disorder caused by the effects of several genes in combination with environmental factors. This disease is characterized by myelin loss, varying degrees of axonal pathology and inflammatory lesions. It is an important cause of disability in young adults, seen to be more prevalent in the woman, and affects 2.5 million people worldwide. Great efforts are being made in identifying the role of Vitamin-D in MS, where Vitamin D deficiency seems to contribute to disease activity and Vitamin-D supplementation investigations have proved this issue. Two significant prospective studies showed a protective effect of Vitamin-D in MS. A case-control study reported that high serum concentrations of 25-hydroxycholecalciferol associated with decreased MS risk. Recent prospective s tudies confirmed these results and reported that levels of Vitamin-D over 75 nmol/L were related to a decreased MS risk. Numerous observational investigations have consistently proved a relation of low serum levels of Vitamin-D with increased MS risk and supported the results from the prospective studies. Vitamin-D intake was found to relatively decrease the risk of MS in a large prospective study (n= 187,563). Vitamin-D status associated inversely with escalation risk in relapsing-remitting MS and recommended a beneficial outcome on MS disease activity. This effect was also seen in patients on interferon-β therapy, where the lowest rate of new lesions was found in patients with Vitamin-D levels over 100 nmol/L. Whether and how Vitamin-D contributes to the pathophysiology of MS is unknown. Further insight into the role of Vitamin-D, in neuro inflammatory diseases, especially as it relates to the immune system, neuroprotection, and inflammation, will help shed light on the causal pathophysiology of these conditions and may aid the design of better treatment strategies for future.

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The Role of C- Reactive Protein in Obesity and Neuropathic Diseases

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Acute phase C-reactive protein (CRP), raised in obesity and inflammation, is a main binding protein for leptin. It is assumed that CRP contributes to leptin confrontation by preventing leptin from crossing the blood-brain barrier (BBB). Here we defined how CRP cooperates with the BBB and whether it deters leptin from attainment CNS targets. CRP was constant in blood, but did not permeate the BBB in trace quantities. Though, it increased paracellular permeability at an upper dose. CRP did not permeate hCMEC/D3 cells nor change zona occludin-1 or cyclooxygenase-2 expression. Investigations showed that a middle dose of CRP had no outcome on leptin transport across the BBB after co-therapy. Therefore, acute interactions among CRP and leptin at the BBB level were insignificant and did not elucidate the leptin resistance seen in obesity. The interactions of CRP and the BBB increased paracellular permeability at a high dose that permits its entry into the CNS and aids to induce reactive gliosis and damage CNS activity.

P100

A Review of Postoperative Cognitive Dysfunction and Neuroinflammation Processes Induced by Anaes thesia

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Although anaesthesia is generally effective, safe and also an indispensable preoperative clinical approach, there is a really growing concern about usage, form of usage and dosage of it. Postoperative cognitive dysfunction (POCD) is one of the common postoperative complications that often affects elderly patients and includes a range of domains, may involve oxidative s tress and neuroinflammation. The etiology of POCD has not been known till now, but it was shown that the type of surgery and type of anaesthesia (intravenous vs. volatile anesthetics) don't have significant effect on the incidence of POCD, and as a multifactorial disorder it's better to consider factors such as surgery, anaesthesia, in general, also the consequence of them, and patient-related predisposing factors. Also it was shown that the consequent long-term cognitive deficits, as a potentially harmful factor to the human brain, should be avoided. The detailed molecular mechanisms of POSD is also still largely unknown, while the neuroinflammation has been increasingly denoted as one of the core mechanisms for the pathogenesis of POCD. The mast cells-neurovascular unit communication and the inductive effluence of extracellular RNAs on neuroinflammation are some of possible mechanisms. Even though anaes thesia is an essential and generally safe preoperative clinical approach, because the adverse findings on the relation of it with POCD and neuroinflamation, the person that introduces the anaesthetics should be more aware. Because the exact mechanism and the reason of POCD still has not been significantly proved, more research is needed to regulate neuroinflammation and its relationship to cognitive performance.

P101

Neuroinflammation and Cognitive Dysfunction as a Side Effect of Abdominal Surgery

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One of the most common postoperative complications is Postoperative cognitive dysfunction (POCD), and it's usually a geriatric's disease. Although many studies have done, the exact molecular mechanisms of POCD is still largely unknown; nevertheless, neuroinflammation has been increasingly denoted as one of the core mechanisms for the pathogenesis of POCD. As a hypothesize, surgery-induced neuroinflammation was suggested to mediate POCD and also play an important role in pathobiology of neurodegenerative diseases, stroke, and neuropsychiatric disorders. The abdominal surgery as a different form of disease is symptoms, molecular imparity and area of disorder in the brain attracted lots of attentions. One of the probable mechanisms that shows that explains why abdominal surgery leads to POCD is Mast Cells-Neurovascular Unit Communication. There is a significant communication between the immune sys tem and the central nervous system (CNS). Mast cells, as the first responders in the CNS, can other responses beyond the activation; in addition they can modulate inflammatory processes in initiate, strengthen and prolong multiple CNS pathologies by their secreted mediators; and surgery, generally, induces degranulation of them. Furthermore, surgery can induce neuroinflammatory responses, and pro-inflammatory cytokines, including TNF-α, IL-1β, IL-4 and IL- 6. Extracellular RNAs that released from necrotic cells, as another candidate, were observed to initiate the inflammatory responses in

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different pathological conditions and neuroprotective and edema protective effects of ribonuclease was sugges ted in acute stroke. Evidence indicates that surgery may lead to neuroinflammation and POCD, but because the exact mechanisms of it is unknown till now, more research is needed to regulate neuroinflammation and its relationship to cognitive performance.

P102

The Association of the Anti-GAD Antibodies to the Neurological Conditions

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Glutamic acid decarboxylase (GAD) is an enzyme which converts the glutamic acid to the neurotransmitter gamma-amino butyric acid (GABA). GABA is an inhibitory neurotransmitter that inhibits or weakens the neuronal stimulations. Presynaptic GABAergic neurons in the central neurons system (CNS) and the cells in the islets of Langerhans in the pancreas generate GAD. There are two isoforms of GAD namely GAD-65 and GAD-67. Antibodies against GAD (anti-GAD-Ab) are related to some neurological conditions that these antibodies usually attack the GAD-65 isoform. Neurological conditions such as stiff-person syndrome, epilepsy, limbic encephalitis and cerebellar ataxia are associated to the antibodies against GAD. Furthermore, those antibodies are synthesized in the IDDM (Insulin-Dependent Diabetes Mellitus). There are various mechanisms for the function of these antibodies in different conditions but they mainly function by inhibiting the postsynaptic transmission in Purkinje cells, creating the neuronal dysfunction and reducing the GABA by GAD destruction that these mechanisms demonstrate the relationship between the neurological conditions and the antibodies against GAD.

P103

The Association between Antibasal Ganglia Antibodies of Streptococcal Infection and Neurological Conditions

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The outbreak of the post-streptococcal neurological disorders related to the antibasal ganglia antibodies is broadening. In addition to the disorders such as chorea and obsessive-compulsive disorder which have been recognized previously, the movement and behavioral abnormalities are the other aspects of post-streptococcal neurological disorders. Streptococcus is a positive-gram and coccus bacteria which causes disease in human. Antibasal ganglia antibodies (ABGAs) are related to the group A-beta haemolytic streptococcal infections (GABHSs). The neurological conditions such as Tourette syndrome (TS), obsessive-compulsive disorder (OCD), acute disseminated encephalomyelitis (ADEM) are associated to the increased levels of ABGAs that we can use the levels of ABGAs as a marker for recognition the origin of those neurologic conditions. It has been showed that the process of generating neurological conditions by ABGAs is an antibody-mediated process. Furthermore, There are several mechanisms for the function of the AGBAs but the underlying mechanism is that the antigens of streptococcus infections imitate molecularly the basal ganglia so that when the antibodies attack those antigens, in fact the basal ganglia are invaded by those antibodies.

P104

Obesity-Induced Neuroinflammation: Focus on Hypothalamic Inflammation

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Obesity is a Health issue around the world. Obesity is not limited to body weight, generally associated with low grade inflammation and with a cluster of disorders generally referred to as “metabolic syndrome”. Regarding obesity and relapse, long-term concentration was set on the hypothalamus. Most recently,obesity- Originated neuroinflammation has been shown to affect other brain structures such as the hippocampus,cortex, brainstem, oramygdala. Additionally, obesity is accompanied by an increase in central disturbances such as depression and cognitive impairment. The hypothalamus is a key brain region in the regulation of energy balance. The hypothalamus is a key brain region in regulating energy balance. Especially controls food intake and both energy storage and expenditure through integration of humoral, neural and nutrient-related signals. The hypothalamus cells and glial cells act jointly to orchestrate. We discuss the effects and mechanisms of obesity- originated neuroinflammation, with a specific emphasis on extra-hypothalamic structures, as well as the consequences of neuroinflammation for some cerebral functions. Therefore, the existence of a causal link between hypothalamic inflammation and deregulations of feeding behavior, such as involuntary weight-loss or obesity, has

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been recommended. Among the inflammatory mediators that could induce deregulations of hypothalamic control of the energy balance, chemokines represent interes ting candidates. Chemokines, primarily known for their chemical absorption role of immune cells to the inflamed site, have also been recommended capable of neuromodulation.

P105

Inhibition of Vasculogenic Mimicry in a Three-Dimensional Culture in Glioblastoma

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Glioblastoma is one of the most common primary brain tumors (80% of patients) that has a poor prognosis due to malignancy. Glioblastoma has an annual incidence of 5.26 per 100 000 population or 17 000 new diagnoses per year and so as the population aging, the number of patients is expected to increase. There is a growing body of literature investigating the tumor microenvironmental mechanisms which lead to metastasis. It has previously been believed that the tumor ensures its growth through angiogenesis. Recent evidence suggests a new approach for tumor nutrition which is the act of tubulogenesis by tumor cells mimicking endothelial angiogenesis in the condition of hypoxia. This procedure is called vasculogenic mimicry. In addition to searching for oxygen and vital nutrients supporting tumor growth, vasculogenic mimicry can result in metastasis due to tumor cells migration into blood vessels. This approach has been detected in several cancers such as melanoma. In recent years there has been an increasing interes t in vasculogenic mimicry, nevertheless there is not sufficient research discussing mentioned mechanism in glioblastoma. The aim of this study is to investigate tumor cells behavior resulting in vasculogenic mimicry in glioblastoma in a three-dimensional culture in order to simulate natural brain environment to get exact and detailed results. Based on recent researches reviewing controlling ways of vasculogenic mimicry in melanoma and other cancers, it is expected to find interrupting mechanisms for tumor nourishment and metastasis through mentioned approach.

P106

Interleukin-1 Beta; A Forgotten Piece of MS Puzzle Target Therapy

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Multiple sclerosis (MS) is an inflammatory autoimmune disease which is presented by environmental factors and genetic predisposition, increasingly affecting a large number of people worldwide. CNS inflammation is a local tissue response to stimulants and is characterized by induction of cytokines, chemokines and vascular permeability. Our goal in this study is to understand the micro-environment and immunopathogenesis in neurobiology of MS. A group of cytokines such as Interleukin-1 (IL-1), play an important role in MS pathogenesis. In spite of the fact that IL-1β is a pro-inflammatory cytokine and a mediator responding to inflammation of the nerves, but it's role in chronic pathophysiologic conditions isn't well known. IL-1β is mainly secreted from endothelial, T cells, fibroblasts, as trocytes and microglial, meanwhile it is secreted from monocytes, B and T cells as autoimmune mediator in MS patients. So, increment in serum, CSF and CNS lesions level of IL-1β titration in patients is of importance compared with healthy ones. IL-1β is like a sword of two edges, it is secreted from gelial cells in the hypothalamus and activates the neurons, reducing plaque volume. On the other hand, its neuronal excessive expression in MS patients causes severe clinical symptoms, apoptosis of the neurons and the loss of axons and myelin sheath. According to failure in several therapeutics based strategy, MS progression has been remained as a dilemma, leading clinical researchers look for novel agents for target therapy in MS immuonopathogenesis and microenvironment.

P107

Using Nano Particles as a Novel Application for Alzheimer's Disease; an Effective Endeavor for Drug Delivery

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As the most common cause of dementia among the elderly results in cognitive and behavioral impairment, Alzheimer's disease (AD) is characterized with aggregation of senile plaques (Beta-amyloid protein), cortical atrophy and ventricular enlargement. Unfortunately, conventional methods like acetyl cholinesterase inhibitor drugs, are not so effective owing to restrictive mechanisms imposed at the blood–brain barrier (BBB), poor solubility, and low bioavailability.

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So, researchers show a tendency towards using Nano technological methods involving application of nanoscale drug delivery system through polymeric nanoparticles, microemulsion, solid lipid nanoparticles, nanostructured lipid carriers, nanoemulsion, and liquid crystals. As drug delivery agents, Nanoparticles are solid colloidal particles ranging in size from 1 to 1000 nm that mask the BBB limiting characteristics. This system may slow drug release in the brain, decreasing collateral damage and peripheral toxicity. They have high drug loading capacities that is capable of targeting towards the mutagenic proteins of AD. Biodegradable nanoparticles such as PLGA, PLA, chitosan gelatin, polycaprolactone and poly-alkyl-cyanoacrylates have been used frequently as drug delivery vehicles due to its grand bioavailability, better encapsulation and less toxic properties. These carriers can deliver drugs that proved to have anti-Alzheimer effect, such as: clioquinol derived from quinoline known to solubilize the A-Beta plaques in vitro and inhibits the A-Beta accumulation in AD transgenic mice in vivo, D-Penicillamine conjugated to NPs seem to reverse the metal-induced precipitation (specially Cu2+) and decreases the beta amyloid protein concentration. A robust collaboration between specialis ts and medical nanotechnology researchers opens promising windows to AD dilemma.

P108

Microglia in Traumatic Brain Injury

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Microglia is one of the first innate immune components. These cells account about 5 to 10% of the entire adult brain cells and are activated by trauma. Complex-mediated inflammatory responses occur through cellular and molecular events during and after the traumatic brain injury (TBI). In-lesion area astrocytes, microglia, and damaged neurons begin to secrete cytokines and chemokines. Microglia has the potential to polarize the M1-like and M2-like phenotypes. Several studies have been shown that the use of different therapeutic methods effect on the polarization of microglia phenotypes. Intracranial transplantation of human neural stem cells (hNSCs) decreased microglial activity through M2/M1 ratio in the cortical-controlled injury model. This switching of phenotype was associated with an increase in the expression of the anti-inflammatory interleukin-4 receptor α and a decrease in the expression of the proinflammatory interferon-γ receptor β, and ultimately most hNSCs differentiated into neurons. Microglia has proposed as a target cell in the process of treatment after head trauma. Different phenotypes of microglia have different effects on the tissue and brain function. Knowing how microglia works on neurodegenerative and TBI diseases are crucial for determining therapeutic s trategies.

P109

Neuroinflammation and Forgotten Dimensions of Persian Medicine: from the Basic Concepts to Biopsychologic Interventions and Their Probable Mechanisms

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Based on the teachings of ancient philosopher/physicians in Greece, Persia and Islamic lands inflammation starts with a non-self hot dystemperament which can involve any type of corporeal structures of the body (tissues, primary and secondary fluids, subtle & fine bodies). Such Non-self hot dystemperaments in neural tissues might happen due to the somatic and/or psychological causes, and seems to have correlations with both the biologic factors especially the original personal temperament, and the life style especially diet, sleep pattern, and mental-emotional events chiefly continuous anger and grief repression. According to the Persian medicine literature, inflammations in the brain can primarily begin from the brain itself or may originate from other sites of the body especially from gut, liver, spleen, and uterus which all interact with the brain. On the other hand stress-induced neuroinflammations attributed closely to the heart. To assess and judge about this explanatory model for neuroinflammation, one should firstly understand the basic concepts as elucidated in the main works of Persian medicine literature, but practically it is so helpful to know that regarding these basic concepts and teachings, various nutraceutical and pharmaceutical protocols can be designed to subside the neuroinflammations and heal their subsequent events. It seems this ways works through regulating the immune system, anti-oxidant and anti-inflammatory actions, improving the hepatic detoxification process, remodeling of extra cellular matrix, improving the psychoneuroimmunologic and psychoneuroendocrine axes, and promoting the bioenergy fields. To discover the new solutions for neuroinflammation it is necessary to merge the teachings of Hikmat-based medicine with modern findings in various fields especially neurosciences, health psychology, and psychosomatic medicine in a systemic convergent approach. Top research ideas derived from the Persian medicine

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manuscripts can play important roles to find novel scientific perspectives and new therapeutic ways with more efficacy and safety.

P110

Nitrous Oxide and Neuroinflammation

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Nitrous oxide was an anaesthetic gas that was used for many years. There were many anaesthetic drugs in operations with many side effects. Nitrous oxide had some side effects too. But it was a safe gas in many times. In hospital personnel who worked in operating rooms exposure to this gas was important. The objective of this study was the introduction of nitrous oxide and neurologic side effects. In the review paper in texts, magazines and guidelines the result was found. The researcher tried to finding new and applicable results for prevention of effects on operating room personnel. The nitrous oxide had neuroinflammation effects and could be caused demyelination of nerves and myeloneuropathy. It had symptoms similar to Vit B12 deficiency. Operating room personnel must be protected from exposure to nitrous oxide with proper ventilation such as scavenging systems in the rooms. The mask of the patient must had not leak. Correct respirator must be recommended for these personnel with specific media cartridge for this gas. Control of nitrous oxide and Vit B12 in the blood was recommended too.

P111

Effect of Human Neural Stem Cells on Neural Hyperactivity in Kindeling Rat Models

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The excessive electrical activity of neurons is reported in many diseases including: Parkinson's disease, Alzheimer's disease, and Epilepsy. Electrical overactivity in hippocampus accelerates the depletion of neural stem cell (NSC) and impairs the neurogenesis in hippocampus. It is believed that neurogenesis in hippocampus improves the cognitive functions. In this experiment, we use kindled model of rats to represent the hyperactivity of neurons, using the repeated weak excitation of brain structures that progressively increases sensitivity to the same stimulation. At the end, we will compare the NSC group, vehicle group (which get the resolvent of NSCs), and control group by considering their immunohis tochemistry and western blotting samples of each group. Cognitive function and neurogenesis in the hippocampus will be evaluated. Our data probably show that the NSCs can improve the function of the hyperactivated brain and can reduce the complications and impairments due to the hyperactivated neural diseases by change in rat's EEG and field potential record.

P112

P112: Tumour Associated Macrophages and Vasculogenic Mimicry: A New Insight in Glioblas toma Treatment

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Glioblastoma is one of the most common brain tumors in adults with poor prognosis, aggressiveness, and treatment resistance. Vasculogenic mimicry (VM) consists of generating vascular-like channels by tumor cells, independent of endothelial angiogenesis. Studies showed in glioblastoma, the proportion of VM to all vascular channels is associated with poor prognosis and higher invasiveness levels. Tumor-associated macrophages (TAM) play a homeostatic role in glioblastoma maintenance and growth by producing immunosuppressive microenvironment and pro-angiogenic factors. In comparison with low-grade glioma, the number of macrophages in glioblastoma is higher in correlating with a tumour vascular density. Up-regulation of VM markers and increased interleukin 6-type (IL-6) production were observed in tumor–macrophage coculture. Although it's indicated that TAM induces VM formation through IL-6, but more studies is needed to clarify the signaling pathways between TAM and VM formation. It can make new insights in glioblas toma treatment in the future.

P113

Inflammation and its Role in Neurological Diseases with an Emphasis on MS

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Inflammation is an important factor in the pathophysiology of neurological diseases and the physiological response of the immune system against internal and external harmful stimuli. Inflammation is the natural response of the body to damage, which leads to the removal of debris from dead cells and infections from damage and tissue repair. In this study, neuropathic inflammation and its role in the pathophysiology of neurological diseases, with an emphasis on MS illness, has been studied. The method of doing this research is descriptive. In MS, the cells of the myelin and myelin are damaged, resulting in damage to the lower nervous system, which is called axonal injury. The three main characteristics of MS are inflammation, myalgia, and gliosis (scarring). The result of this study shows that MS has not been treated conclusively until now, and only the current treatments for MS are reducing microbial inflammation and some medications and treatments. Symptoms to improve symptoms and slow the course of the disease. The identification of endogenous nerve stem cells in the central nervous system of the human is a new strategy for the repair of brain damage.

P114

Rehabilitation for Inflammatory Disorder in the Brain

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The diversity of neurological sequelae that may occur after an inflammatory disorder in the brain (brain abscess, encephalitis, or meningitis) provides a range of challenges to the rehabilitation team. The therapist must identify the problems underlying the individual's movement dysfunctions without the template of the cluster of "typical" problems available with some other neurological diagnoses. Each client presents a combination of problems that is unique to that client and that requires the creative design of an intervention program. The following discussion of the therapeutic management of individuals recovering from an inflammatory disorder in the brain focuses on the process of designing an intervention plan to address the specific dysfunctions of the individual client. Because the management of the clinical problems is built on an understanding of the underlying pathological condition and because therapists may not be as familiar with these disease processes, an overview of the inflammatory disorders of the brain is presented.

P115

Abstract title: A Novel High Tech Approach to Monitor the Pharmacotherapy of Alzheimer; a Narrative Review

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Alzheimer's disease (AD) is multisystem and multifactor disease with a long no-symptom stage. We propose that a more effective approach to use fMRI as a still emerging, repeatable, non- invasive neuroimaging tools that can be very useful for evaluating, diagnosis, treatment and drugs- development. We studied 30 articles which published between 2008-2017 that included the effects of different biomarkers and tools for diagnosing AD and assessing, improving and detection of Alzheimer's medications. Attractive alternatives to the animal and human experimental modelling approaches are the "multi-scale", "multi-level" computational modeling approaches to AD drug discovery and therapy. 6 articles were about the animal models while we should try accepting the limitations of animal models and focus more on humanize research. Peer-reviewed publications were identified through search in PubMed, Google scholar and SCI-HUB by using the search terms "fMRI", "Alzheimer", "cognitive side effects", "drug", "pharmacological neuroimaging". The search was limited to articles published in English. Relevant articles were chosen based on clinical experience and the expertise of the authors. FMRI measures hold promise for multiple clinical applications. Generally, models especially pharmacological fMRI showed that drug repositioning is a cost-effective way to develop disease-modifying treatments over shorter timescale and future models should provide a theory of how increasing Ach levels using cholinesterase inhibitors and N-methyl-d-aspartate antagonists (NMDA) impact neural and behavioral processes in AD. Models should also inves tigate how memantine (NMDA antagonists) can reduce toxicity of beta-amyloids as reported in experimental studies.

P116

The Relationship between Spinal Cord Injury and Neuroinflammation and Treatment Methods

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Spinal cord injury (SCI) is usually caused by a physical factor, especially like burst fracture. Its

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primary phase involves displacement and physical accidents for the spinal cord, which have two factors of depth and speed of impact. In this phase, most damaged cells are oligodendrocytes in white matter. The secondary phase involves a cascade of cellular and molecular events that progresses rapidly and can cause neuroinflammation.in this phase, The leukocytes and microglians that are in injured region, accelerating the development of neuroinflammation by making unknown species of oxygen reactions. Our goal is to investigate the factors that reduce or improve neuroinflammation. Immunoglobulin G can decrease the activity of leukocytes and microglians and rate of neuroinflammation. Also, the use of useful antioxidants can be effective in reducing neuroinflammation. It seems that presence of immunoglobulin G with an antioxidant can reduce the amount of neuroinflammation caused by SCI seriously. For example, research has shown that combination of palmitoylethanolamid (an endogenouse lipid-neuroprotection) and luteolin (anti-oxidant) can reduce neuroinflammation.also,Ethanol is extracted from black chokeberry (Aronia melanocapa L.) has anti-inflammatory effects. Other studies have also shown that transplantation of bone marrow mesenchymal stem cells (BMCs) and cerebral dopamine neurotrophic factor can inhibiting the process of neuroinflammation and decearse production of proinflammation.in addition,it can be effective in regenerating and repairing damaged neurons in SCI. Perhaps a combination of stem cell transplantation and antioxidant effects can help to prevent the development of neuroinflammation and regeneration damaged neurons.

P117

A Study of Prevalence of Primary Headaches in Patients with Multiple Sclerosis

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Primary and chronic headaches are one of the most common problems in patients with multiple sclerosis, seen in more than half of them. Despite the fact that pain is not the main symptoms of MS, many of the patients suffer from various types of pain, including a headache. Headache is one of the MS-related pain syndromes. The study is based on the consideration of the prevalence of primary headaches in MS patients. The data from the current study, including the MAGIRAN, SID, and Google Scholar datacenters, were collected from the database, and the related queues were subtracted from the study. Studies have shown that primary headaches in patients with MS can be attributed to disease, side effects of medications, genetic and environmental factors, the presence of MSPP in the upper respiratory tract, age, gender, duration of disease, physical and social function, and complications of the disease, such as depression and anxiety. Migraine is the most frequent primary headache in MS patients and is prevalent among females. MS patients with headache have larger number of sites than MS patients without headaches and MS patients with migraine have had more lesions in the cerebrospinal fluid, cerebellum, frontal and temporal vesicular thalamus. Due to the high prevalence of headache in MS patients, a clinical study is required in all patients, since both headache and MS affects the quality of life and daily functioning of the patients. As a result, simultaneous attention to these two forms can play an important role in medicines prescription and other common therapies of the disease.

P118

Metformin; A Review to its Anti-Neuroinflammatory Properties

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Neuroinflammatory disorders include a wide spectrum of disorders in which immune system injures components of the nervous system. Despite the advances in therapy, lack of efficient curative therapies for these disorders not only affects the quality of life but also places huge burden on the society. These limitations have necessitated therapeutic interventions for developing more efficient regimen. Recently, emerging new therapeutic approaches like targeting underlying pathways offered some respite and drawn much attention. Accordingly, adenosine monophosphate-activated protein kinase (AMPK), signal transducer and activator of transcription3 (STAT3), P65/NF-κB, mTOR, and PI3K/Akt pathways have been reported to be affected in neuroinflammation, and, thus could be considered as novel molecular targets for therapy. Among the available drugs, metformin has shown a great potential in targeting aforementioned pathways. Studies declared that metformin could suppress neuroinflammation in neurological deficits such as Alzheimer disease, brain injury, and neuropathic pain. Besides, preclinical and clinical studies indicated that metformin not only improves inflammation through affecting metabolic parameters, but also exerts direct anti-inflammatory effects. In this view, metformin could be defined as an available, cost-effective drug with great potency to target multiple signaling pathways in neuroinflammation. However, requiting metformin in clinical stage demands enough knowledge about the mechanisms and pathways involved in its anti-

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neuroinflammatory activities and comprehensive review of its in-vivo utility. This review discusses the published evidence of anti-neuroinflammatiory properties of metformin and its clinical implication in neuroinflammatiory disorders with the objective of paving the way for further clinical application and better management of diseases.

P119

Animal Models of Epilepsy: The Impact of some Chemoconvalsants on Animal Models

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We summarize some of the most frequenthly used rodent animal models of temporal lobe epileps and the impact of chemoconvulsants on them. Temporal lobe epilepsy is the most common epilepsy in humans in which seizures spread to the neighboring cortiase and hippocampal neuron loss and other neuropathological take place. Temporal lobe epilepsy and the other form of epilepsy cannot acquired in chinical studies whit human, as result the use of oppropriate animal models is essential. Rodent must display a similar “chinical history” as the human conterpart including an intial latent period between the injury and the occurrence of spontaneous seizures chronic manifastation of spontaneous seizures and his topatological change deemed characteristic of temporal lobe epilepsy. Chemoconvulsants: 1) Kinic acid: Kinic acid was one of the first compounds used to model temporal lobe epilepsy in rodonts, injecded rodents show recurrent seizures. Kinic acid has the advantage of causing habitually hippocampus -restictes injury. 2) Pilocarpien: In the human halt spontaneous seizures in the pilocarpien model, systemic or intracerebral injection of pilocarpien causes seizures that build up into a limbic. In addition, there are several network and neurochemical similarities between human temporal lobe epilepsy and the pilocarpine model. Pilocarpine wich can also produce lesions in neocaritical area cognitive and memory deficits’ commonly are found in temporal lobe epilepsy patient’s, are also present in pilocarpine rats. Chemocanvulsants allow rapid inves tigation of epileprogenic mechanisms and screening at the expense of high mortality of subject and spontaneous seizures.

P120

Efficacy and Safety of Dimethyl Fumarate Treatment in Relapsing-Remitting Multiple Sclerosis

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Multiple sclerosis (MS) is a chronic autoimmune disorder of central nervous system. This demyelinating disease affects more than 2.3 million people world wild. Most of patients are young adult. The most common type of MS is relapsing remitting multiple sclerosis (RRMS). However there is no cure, available modifying therapies has revolutionized the care of patients with RRMS. Interferon (IFN) beta has been considered as the first line treatment of RRMS. Some reasons including lack of efficacy and safety concern cause to switch to an alternative disease-modifying therapy (DMT). Delayed-release dimethyl fumarate (DMF) has been recently approved as DMT and demonstrated significant efficacy in patients with RRMS. It activates the nuclear factor-related 2 (Nrf2) pathway which cause augmenting the oxidative capacities and reduction of inflammation. This review has focused on elucidating the efficacy and safety of Dimethyl Fumarate for Relapsing-Remitting Multiple Sclerosis. In most studies the relapsing ratio had been decreased for at least 6 months using of DMF. Expanded Disability Status (EDSS) and radiological activity had improved in case group versus placebo in most studies. DMF has significant efficacy in patients with previous IFN treatment. The severe side effect of DMF occur in approximately 5% of patients but in mos t clinical trials Absolute Lymphocyte count (ALS) were generally stable throughout the observational period. So DMF appeared to be safe and efficient in most clinical trials.

P121

The Effect of Stress on Neuroinflammation in Multiple Sclerosis

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Multiple sclerosis (MS) is a chronic autoimmune disorder of central nervous system. This demyelinating disease affects more than 2.3 million people world wild. Most of patients are young adult. There are many possible triggering factors including infections, toxin, immunization, trauma, sunlight exposure and hormonal variable in pathogenesis of MS. One of the important trigger is stress. There are some substantial evidence that

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indicates stress can precipitate or worsen symptoms and sign of inflammation in general and more specifically in multiple sclerosis. This review has focused on elucidating the effect of stress on neuroinflammation in multiple sclerosis. The mechanism of stress in MS is not completely understood. There are some proposed theories such as secretion of Corticotropin Relapsing Hormone (CRH) and neurotinensin (NT). These inflammatory factors activate microglia and mast cells leading to maturation and activation of T17 autoimmune cells. Disruption of Blood Brain Barrier (BBB) cause T17 cells to enter in to the CNS. Finally brain inflammation will be worsened. The pathway of stress neuroinflammation can be considered as a therapeutic target for M.S patients. Some evidences indicate Glatiramer Acetat, stress management therapy, Diazepam, Alprazolam and CRH antagonists can reduce brain inflammation So the mechanism of stress in pathogenesis of M.S can be novel therapeutic target.

P122

Small Molecules as Chemical and Pharmacological Tools for Neuroinflammatory Diseases Treatment (with Emphasis on Multiple Sclerosis)

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Multiple Sclerosis (MS) is a neuroinflammatory disease resulting in degeneration of the myelin sheaths and death of oligodendrocytes. So far, several strategies have been introduced to control the disease. Treatment with small molecules is one of the strategies that have recently attracted the attention in the scientific community. These molecules that target epigenetic and other cellular processes offer powerful tools for disease treatment from several ways including modification of cell function by inhibition/activation of specific proteins and also reprogramming somatic cells and manipulating their fate to a desired cell type. This process ultimately leads to demyelination cessation and remyelination stimulation. Studies show that specific small molecules such as Src family kinase inhibitor PP2, and Chir99021 chemically have been effective in modulating disease progression and also its treatment. There are several studies that have reported successful efforts of using these molecules for MS control and treatment. In this article the authors will review recent studies that have been published in this research area. We have searched the PubMed databases comprehensively and accurately to find peer reviewed articles with Small molecule and multiple sclerosis keywords. We studied them carefully and selected the most prestigious and the most recent of them to ensure that all knowledge on this topic is discussed. Also we put meeting abstracts under precise consideration to ensure that all references have been investigated. In this review we emphasized on the great opportunity of using small molecules for MS control and treatment. There are several studies that have used small molecules to inhibit demyelination and induce remyelination that indicates the growing attention to this research area. Nevertheless, there are several challenges in using these cells for treating MS but we hope that efforts of this growing research community will completely solve all the problems and someday this therapeutic approach will take a great step in neuroinflammatory disease treatment.

P123

Stimulating In Vivo Remyelination (IVR): A New Approach for Multiple Sclerosis Treatment

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Multiple sclerosis (MS) is one of the most common neuroinflammatory disorders that causes disability in the young adults. In this disease immune-driven demyelination and following that inefficient remyelination occurs. Therapies for this disease are limited, especially those to enhance myelin repair. Cellular reprogramming using defined genetic factors is a way to produce remyelinating Oligodendrocyte Precursor Cells (OPCs). These cells can be differentiated to Oligodendrocytes (OLs) to produce myelin sheets around naked axons. There are some theories indicating this approach has significant risks with respect to abnormal expression and genetic mutations. Therefore, researchers have focused on cell reprogramming by non-viral and non-integrating compounds. Recently, Proteins-mediated in vivo reprogramming and small molecules effective on neural cell reprogramming and transdifferentiation have attracted wide attention in the scientific community. There are several studies that have reported successful efforts of reprogramming neural cells and differentiation of this cells to the desired neural ones including OPCs and eventually OLs. In this article the authors will review recent studies that have been published in this research area. Recently, IVR is attracting attention of translational researchers aiming for medical applications. There are several studies that used IVR approach to induce regeneration in the central nervous system that indicates the growing attention to this research area. Despite challenges ahead, we hope

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that efforts of this growing research area will solve the problems ahead and some day may apply these therapeutic approach for treatment of MS patients.

P124

Listeriosis in Central Nervous System

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Listeriosis is one of the CNS inflammatory diseases which is happened majority in the form of sporadic. Lis teriosis is ingested with the food. Infection occurrence in an exposed person depends on the number of organisms ingested, the virulence of the organisms strain and the individual condition. It is known that Listeriosis has decisive importance as an infection which the cellular immunodefense mediated by the T lymphocyte is dis turbed. Even patients without previous disease worth indicating may be affected. The attribute of the many CNS manifestations are illustrated via the case his tories (meningoencephalitis, acute meningitis, brain abscess, brain stem encephalitis, chronic recidivating encephalitis, meningoencephalitis with infected cerebral infarct). Neurological signs, combined with CSF findings atypical for bacterial CNS disease, must not be taken and may point to listeriosis even though they aren't specific for CNS listeriosis. The conclusive evidence is the proof of the Listeria in the blood or CSF or the proof of antibody titre changes in the serum. New CSF diagnos tic procedure like CSF lactate determination and the recognition of IgG-positive B lymphocytes are suitable in differentiating between viral and noninflammatory CNS disease; the most important for consistency are repeation of CSF examinations. The therapy of choice in CNS listeriosis is high-dosage amoxycillin or ampicillin treatment combined with gentamycin. Estimation of chances of the listeriosis in CNS depends on the prior to disease in each case. The high mortality is at least in part due to delayed diagnosis.

P125

An Overview of the Effect of Inflammation Induced by Temporal Epilepsy on the Hippocampus and Amygdala Based on Nerve iImaging

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Temporal epilepsy is a common neurological disorder that begins before adulthood. Two -way factors in causing epilepsy and continuing attacks can be inflammation that is caused by immune system and infection. The hippocampus and amygdala are part of a limbic sys tem that relies on memory and emotional regulation. The purpose of this study was to review the effect of epileptic-induced inflammation on the hippocampus and amygdala based on neuroimaging. In a vast library search, the keywords "neuropathic inflammation, temporal epilepsy, amp; hippocampus amygdala" were searched for the pedagogical, pedagogical, science and medical sciences databases, as well as Google Scholar in a five-year period. 50 related articles were identified in English, review articles showed that, contrary to the common view that the cause of temporal epilepsy is the onset of the hippocampus, chronic inflammation resulting from trauma and infection can be an important component in epilepsy. In a recent study on animal and human models that were performed through neuronal imaging, a range of hippocampus and amygdala malformations was observed, which significantly explained the poor performance of memory and learning among affected children than peers. Inflammation, as an effective factor in epilepsy, causes specific biochemical changes in the neurotransmitter of TNFX and decreases glutamate and neuronal levels in the cystic gyrus. Given the available evidence, neuroimaging as an inflammatory diagnostic tool can lead to early epilepsy treatment.

P126

Post-Traumatic Stress Disorder and Inflammation

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Post traumatic stress disorder, a special disease that also accompanies with histological changes such as inflammation. In this paper we decided to review the relation between PTSD and inflammation. Stressful events causes immune system dysfunction by suppressing natural killer cells and altering levels of cytokines. Also in this condition, cytotoxic T lymphocytes results in under strained pro-inflammatory cytokines (PICs). These pro-inflammatory cytokines play an important role in the disease generating and their overproduction lead to nitric oxide (NO) and reactive oxygen species (ROS). Elevated ROS can cause cell death and tissue damage also there are some special cellular mechanism that intervenes in this process and prevent of cell death, tissue damage, but their exact mechanisms is still unknown. PIC upregulation is due to activation of inflammasome more over than leukocytic responses. Inflammosomes are protein complexes that cooperate with pattern-recognition receptors (PRRs). When inflammosomes activates, it converses pro-caspase 1 into its active

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form which lead to PIC production and intervenes the inflammatory response. These cytokines can across the blood brain barrier and reach to the central nervous sys tem an activate microglials which causes producing more cytokines and this causes a positive feedback loop. A study which has done to investigate the cytokines which are in common with inflammatory cytokines. In this study, the participants divided into two groups the control and the experiment. Studying their peripheral blood revealed that interleukin 1β, interferon γ were higher than the control group. Tumor necrosis factor α in PTSD patients who did not take any medication, were increased in comparison with the controls. It would be interesting to you to know that interleukin 1β, TNFα and interleukin 6 are also depression cytokines and it would be a reason for accompanying PTSD by depression. It can be concluded that more investigations is needed to detect the histological mechanism of psychological diseases such as PTSD, moreover than its psychological diseases.

P127

The Role of Genes in ASD

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Autism Spectrum Disorder (ASD) is a heterogeneous group of neurological disorders that is determined solely by their behavior. In this disease, a large part of the neurological disorder and neural controls disorder are observed. Researchers believe that over expressions changes in many genes are the cause of autism. Our goal is to investigate the genetic factors affecting ASD and its treatment by umbilical cord. Most genes that cause the disease have only little effect on the disease, but in general, their interaction with other known or unknown genes, or some of the environmental factors, determines the ultimate cause for a person who has Autism. Studies shows that copy number variation of the UBE3A gene and over expression of the gene, product E6AP protein is a common cause of autism spectrum disorders (ASDs). During brain development, dendritic growth and remodeling play crucial roles in neuronal connectivity and information integration. Laboratories experiments indicates that overexpression of E6AP in primary neurons in autism mouse brain leads to significant loss of dendritic arborization. This effect is mediated by the ubiquitination of XIAP by E6AP, subsequent activation of caspases, leading to local degeneration and retraction at the tips of dendritic branches. These findings demons trate dysregulation in neuronal structural stability as a major cellular neuropathology in ASD. For the treatment of autism, using of CD34 (stem cell) of umbilical cord and MSC is effective.

P128

Relationship of Childhood Brain Tumors and Hair Dye Usage During Pregnancy

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Brain tumors, which is one of the destructive forms of human being's cancers, are the second most common children's cancers. Brain tumors may have an inherited (small percent), acquired reasons due to environmental factors. Nowadays advances in cosmetic industry have increased our ability in the field of youth and beauty. Hair dye products are such innovations. Recent studies showed considerable controversy concerning about the safety of hair dyes (specially looking at risk of brain tumors and cancers). Several studies represent the absorption of toxicant contents of hair-coloring products in sufficient amounts through the scalp. The carcinogen's removal from hair dyes and appropriate labeling would help reduce the potential risk of brain tumors. The observations showed a widespread use of hair dye which is varies between 66-74% (showing increasing desire of women during pregnancy). Here is a summary of the effect of this increase on brain tumors. Studies have been presented that there was an association between maternal hair dye use and childhood brain tumors (in three of four case studied there was a correlation) and also neuroblas toma risk during childhood period. Even there was an increase in the risk of brain tumors in children of women who start to use hair dyes one month before pregnancy. Finally, it seems the reliability of using hair dye during pregnancy has still been controversial in case of brain tumors cause to the rational approach to recommend avoiding frequent or long use as much as possible.

P129

Use of Stem Cells to Regenerate Degenerative Optic Nerve

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Stem cells are undifferentiated cells that have the ability to convert to different types of cells and after dividing, they can produce their own cells or other cells. Axons

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of the retinal ganglion cells, from the optic nerve. These cells lose the ability to regenerate themselves before birth. Optic nerve degeneration can result from various causes including increased intraocular pressure, compromised vascular supply and physical trauma. There are currently no effective treatment for this disease. Scientists believe that every internal organ of the body has its own stem cells. In the Retina, the stem cells of pigmented epithelium of the eye have been identified which can, to some extent replace the ganglion cells after the optic nerve damage. they are not able to fully repair the nerve. Therefore, researchers are looking for a way to Retinal stem cell transplantation to compensate for defects. There are batches of genes that can convert conventional cells into stem cells using specific genetic agents. These cells called induced pluripotent stem cells (IPSCs). The purpose of this study was to review use of stem cells to regenerate degenerative optic nerve. Stem cell technology is now an important way to treat and replace lost cells. The use of these cells in rats has been successful in the visual acuity damage model. Research has shown that IPSCs can be differentiated into various retinal cells, but whether these cells can functionally replace retinal lost cells, must be further researched.

P130

The Role of Rho-Kinase (ROCK) in Microglia/ Macrophage Polarization in Neuroinflammatory Diseases

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Macrophage/microglia with heterogonous phenotype and function under physiological and pathological conditions are the main cell lineage involved in inducing immune responses in neuroinflammatory diseases which exhibit combined inflammatory and anti-inflammatory functions. An increase in the expression of iNOS triggers M1 phenotype that secrete high concentrations of inflammatory cytokines, while an elevation in the expression of Arg-1 triggers M2 phenotype which forms anti-inflammatory cytokines. Rho-kinase (ROCK) is a serine/threonine kinase and it expresses in both central nervous system and the periphery. ROCK inhibitors have been reported frequently to decrease the infiltration of leukocyte in some models of inflammation, including ischemic injury; the ROCK inhibitors changes M1 to M2 in neuroinflammatory diseases. studies have found three possible mechanisms for M2 polarization by ROCK inhibitors, as follows: iNOS inhibition or Arg-1 enhancement, Change in multiple cytokines production lead to M2 activation likewise increased IL-10 proving M1 shift to M2 microglia. The present review aimed to investigate the role of Rho-kinase on M1 and M2 microglia and the effect of Rho-kinase inhibitors in shifting M1/ M2 phenotypes which is significantly correlated with the neuroinflammatory diseases. The disease phases and severity might be involved in the microglial phenotype changes. Promising therapeutic purposes can be obtained by understanding the stage-specific switching of M1/M2 phenotypes.

P131

Cannabidiol: A Promising Treatment for Intractable Epilepsy

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Epilepsy is a chronic disease affects CNS in various ages. WHO estimates 50 million people are suffering from epilepsy worldwide which make it a serious prevalent problem among neurological diseases. Intractable epilepsy (IE) affects about 20-30% of epileptic patients who failed to be seizure - free after antiepileptic drug (AED) consumption. Although many AEDs now are available for epilepsy treatment, discovering new pharmaceutical components is needed to overcome intractable epilepsy. The antiepileptic potential of Cannabis sativa extractions has been recognized from past. Cannabis has many active components, including cannabidiol and Δ9-terahydrocannabinol (THC). Cannabidiol showed anticonvulsant effects in many studies with mild side effects. The mechanism by which Cannabidiol exerts its anticonvulsant properties is still unclear but may include effects on equilibrative nucleoside transporter, orphan G-protein-coupled receptor GPR55, transient receptor potential of vanilloid type-1 channel, 5-HT1a receptor, and α glycine receptors. Also it's demonstrated the cannabinoid CB1 receptor is the primary site of action for cannabinoid-induced effects on the CNS. CB1 receptor Activation damps neurotransmission and produces an overall reduction in neuronal excitability. Not only anticonvulsant effects but also, cannabidiol showed antioxidant, anti-inflammatory, neuro-proliferative and re-myelinating effects in many preclinical and clinical studies. Further studies is needed to clarify the cannabidiol effects on various CNS disorders. It can make ways to introduce new drugs for neurological diseases, including IE.

P132

Efficacy of Crocin as Anti-Inflammatory Agent in Multiple Sclerosis Patients

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Inflammations form an integral part of the innate immunity against pathogenic infections. NF-κB represents a family of inducible transcription factors, which regulates a large array of genes involved in different processes of the immune and inflammatory responses. It is now clear that NF-κB signaling pathway is involved in the regulation of inflammation, contributing to the initiation and development of inflammatory diseases. One of inflammatory disease is multiple sclerosis, which is the CNS generally considered to be an autoimmune disease involving the pathogenic action of CNS-specific CD4+ T cells, particularly Th1 and Th17 cells. Normally, inflammation is beneficial to the host and can be resolved in a timely manner. Saffron in filaments is the dried, dark red stigmata of Crocus sativus L. flowers and it is used as a spice, food colorant, and a drug in medicine. Crocin is the chemical primarily responsible for the color of saffron. In additional to reduce the TNFR1 protein expression, decrease of p-IκB protein levels and thus confirming an early inactivation of NF-κB so it's useful as anti-inflammatory drugs.

P133

Targeting NF-Kb Signaling Pathway as Potential Therapeutic with Curcumin in Treatment of Multiple Sclerosis

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Curcumin is active component of turmeric and isolated from the rhizome of turmeric, a phenolic natural product. One of inflammatory disease is multiple sclerosis, a multifocal chronic autoimmune inflammatory disease of the CNS, which is also known as a perivascular demyelinating disease. Studies have been shown that neuro-inflammation can have both harmful and beneficial effects on the neuronal and glial cells function, transcription factor nuclear factor-kappa B¬(NF-κB) has a determinant role in controlling this process. The involvement of NF-κB signaling pathway in multiple sclerosis has been suggested by genome-wide association studies. Selective anti-NF-κB therapeutic strategies could be beneficial for minimizing damages during acute and chronic inflammation. Genes that can be induced by NF-κB signaling include those that encode important molecules such as TNF-α, IL-1, IL-6, SOD, Bcl-2, and inhibitors of apoptosis proteins. Curcumin, have been shown exhibit anti-inflammatory and anti-mutagenic in addition to anti-carcinogenic activity. Downregulation expression of the NF-κB-regulated gene products such as COX-2, TNF, 5-LOX, IL-1, IL-6 and others, inhibition multiple pro-inflammatory pathways.

P134

Central Nervous System and Blood Biomarker in Stroke, CNS Bleeding, Epilepsy, and Traumatic CNS Injury; MicroRNAs

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A Central nervous system (CNS) hemorrhage is bleeding in or around the brain and spinal cord. Reasons of CNS hemorrhage include high blood pressure, cancers, drug abuse, abnormally weak blood vessels that leakage, and trauma. Regression of CNS bleeding was confirmed to be relatively repetitive in patients with severe FV, FX, FVII and FXIII deficiencies. Generally in CNS hemorrhage, radiological evaluations are necessary, for example a magnetic resonance imaging (MRI) scan or computed tomography (CT) scan. The MRI or CT scan highlight different features and location of CNS bleeding. Several patterns of MicroRNA (miRNA) expression occur in blood and CNS 24 h after CNS hemorrhage, kainite seizures, brain ischemia, and even surgeries. A number of miRNAs were considerably regulated more than 1.5-fold in blood and brain after each CNS damage. Several miRNAs were down regulated or upregulated in both CNS and blood after a given damage; and a few miRNAs, containing mir-155, mir-362- 3p, miR-298, etc, were down regulated or upregulated in both CNS and blood after several variety damages. The 'cell cycle' was among the top-ranked roles for miRNA regulated in both CNS and blood, and for mRNAs and miRNAs that changed in CNS and blood one day after injury.

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The miRNAs induced in blood related to the 'cell cycle' may relate to the blood inflammatory response and the proliferation of white blood cells (WBCs) to acute CNS injury. Cell cycle re-entry in neurons has been confirmed in a lot of CNS diseases, including stroke, CNS bleeding, epilepsy, and traumatic CNS injury.

P135

The Role of Amyloid Beta-Peptides and Tau Protein in Alzheimer's Disease

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Alzheimer's desease is the most common age-related neurodegenerative disorder, and cognitive problems such as defects in learning and memory are of its symptoms. Among the factors involved in the pathogenesis of the disease are biochemical disorders in protein production, oxidative stress, decreased acetylcholine secretion and inflammation of the brain tissue. Extra-neuronal accumulation of Beta-amyloid and hyperphosphorylated Tau neurofibrils degredate the dendrites and destroy the synapses, which ultimately results in memory loss in Alzheimer's patients. Amyloid Beta is an important molecule in the pathogenesis of Alzheimer's disease, which progressively accumulates in the mitochondrial matrix and directly associated with the mitochondrial toxicity, which leads to the production of ROS and the oxidative stress that result in the neural dysfunction and eventualy the death of neurons. Beta-amyloid fibrils as plaque activate the microgelia, which results in the release of inflammatory cytokines and des truction of neurons. One of the most important effects of beta-amyloid is the damage to synaptic activity and inhibition of stimulant synapses, which in fact causes disruption in the the learning and memory sys tem. It seems that the production of abnormal forms of Beta-amyloid peptides and Tau proteins is one of the main causes of Alzheimer's disease and observing the amyloid plaques in the cortex and the hippocampus in the early stages of the disease and their spread to other areas of the brain at the higher stages are the warnings for this disease. Therefore, it is possible to delay its progress to the debilitating stages by modulating the Beta-amyloid level and preventing the formation of abnormal forms of peptide chains. This review study aimed to investigate the role of Beta-amyloid peptides in the pathogenesis of Alzheimer's disease.

P136

The Role of Th1 Lymphocytes in the Pathogenesis of Multiple Sclerosis (MS)

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Th1 lymphocytes produce cytokines such as IL-2, IFN-γ, and TNF-α, TNF-β and GM-CSF and play an important role in the increase of delaying sensitivity and defense against intracellular pathogens. IFN-γ is the most important Th1 cell cytokine that induces the production of IgG, activation of macrophages,enhancing phagocytosis, and also increasing MHC class I and class II molecules. Increasing serum level of Th1 cytokines have also been observed in MS patients. studies have also shown an increase in the serum level of IFN-γ in mice with EAE. It has also been proven that in humans, exacerbation of MS disease is often accompanied by the increase of myelin-specific Th1 cells in the CSF and according to pathological observations, in thrombolytic plaques, the accumulation of Th1 cells and the production of IFN-γ is directly linked to the demyelination process, which also proves the pathogenicity of Th1 cells. Moreover, the treatment of multiple sclerosis with IFN-γ increases the severity of the disease; while treatment with an anti-IFN-γ antibody improves the disease. Th1 cells cytokines activate macrophages, and activated macrophages cause damage to myelin and subsequently oligodendrocytes and can also produce other inflammatory cytokines that can exacerbate tissue damage. Macrophages and activated microglia cells secrete a number of cytokines such as IL-1, IL-6, IL-12, IL-23 and TNF-α, that high concentrations of these cytokines may damage the oligodendrocytes and neurons. According to studies, Th1 lymphocytes seem to play an important role in immuno-pathological reactions in MS. Preventing the entry of Th1 cells into the CNS, differentiation of native T-cell into Th1 cells, and also activation of Th1 cells, and in the other hand targeting cytokines secreted from Th1 cells or their receptors can significantly reduce the process of demyelination in MS. This review study aimed to inves tigate the role of Th1 Lymphocytes in the Pathogenesis of Multiple Sclerosis.

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Stem Cell Therapy in Alzheimer's Disease

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Alzheimer disease (AD) is a progressive neurodegenerative brain disorder which plays an important role in neural cell destruction and as a result it causes memory loss in the patients. This disease is also the most common type of dementia which doesn't completely respond to medical treatments so no certain cure is available. Recent studies show the advantages of using stem cells (SCs) in treatment of AD. This cells has been used in animal models of AD and they have been effective in control and also treating AD symptoms in this animals. Embryonic stem cells, mesenchymal stem cells, and neural stem cells are the most common SCs that have been used in AD treatment. This cells could be transplanted to the animal body intravenously and locally. There is a hypothesis that the transplanted cells probably stimulate neurogenesis damaged pats of Alzheimer's patient brains that causes improvements in patient's cognitive functions. Another hypothesis says that this cells divide into neuronal precursor cells, neurons and glia in damaged areas and integrate into the brain circuits. SCs also could be used as a carriers for effective therapeutic compounds including Neprilysin, Plasmin and Cathepsin B which will reduce the Beta-amyloids in mice brains. With the advancements of the technology of SCs and the ability of transforming SCs to different types of neurons of central nervous system, many successes in AD treatment are foreseeable. There are a huge number of researchers who try to delay the disease progression and also regenerate the damaged neural cells by this cells. Despite challenges ahead, we hope that efforts of this growing research area will solve the problems ahead and some day may apply these therapeutic approach for treatment of AD.

P138

Effect of Exercise on Improvement of Patients with Multiple Sclerosis

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Multiple Sclerosis (MS) is a prevalent neurological and non-traumatic disease which is most common in young and adult women. Multiple Sclerosis is a disabling disease can affect various aspects of life. s tudies have reported that complement and alternative treatments can have positive effects on people with MS. Exercise presents an important behavioral approach for counteracting the declines in CNS structure and associated function among people with multiple sclerosis. Aerobic exercise improves walking ability in people with multiple sclerosis and increases neurological recovery and neurotrophin in nonhuman animals. Non-aerobic exercise has no effect on the memory of people with MS, but aerobic exercise can be utilized as a behavioral approach to improving the memory of people with MS. Purpose of this review was to study the effects of exercise on the improvement of patients with multiple sclerosis. According to the results, it seems that exercise in a variety of ways (aquatic exercise, aerobic exercise, non-aerobic exercise,…) can have positive effects on people with multiple sclerosis and we can use an exercise plan as a complementary treatment for multiple sclerosis patients in clinical therapies.

P139

Effect of Paeonia Lactiflora Root Extract on Epilepsy

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Epilepsy is a complex neurological disorder that affects around 1%of the world's population.it affects the neural cells of the CA1 and CA3 regions of the brain's hippocampus that causes behavioral disorders. The use of medicinal herbs for the treatment of epilepsy has long been common, but these effectors have been less successful. According to glutamate theory, the cause of epilepsy is the accumulation of glutamate produced from GABA metabolism in the extracellular domain and consequently the inability of the GLT-1 protein in the transfer of glutamate and as a result of impaired function of the nerve. During epileptic seizures, the amount of glutamate in the cell increases. The experiments show that the albiflorin in this extract can be enhanced by inhibiting serotonin and norepinephrine absorption. Purpose of this study is checking the effect of the root extract of Paeonia lactiflora on epilepsy. Albiflorin and pentagalloylglucose can increase the flow of calcium from the gap junction of astrocytes, producing some ATP to the extracellular space, and the activity of the neurons at the same time increases the attack. Long-term exposure to Paeoniflorin can also prevent cell proliferation by increasing the expression of the A20 gene. As a result, the plant can be effective in preventing or reducing the severity of attacks.

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The Roles of Micro RNA in Multiple Sclerosis Disease

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As one of the most common neurological disabilities in young adults, Multiple sclerosis (MS) has characteris tics of inflammation, demyelination, neuro-axonal damage, and progressive prolonged disability. The disease is clinically divided into three general categories according to response to treatment: Relapsing-Remitting MS (RRMS), Primary Progressive MS (PPMS) and Secondary Progressive MS (SPMS). So, focus of clinical researches has been concentrated on some molecular based strategies such as microRNAs (miR). They are small non-coding RNAs with post transcriptional gene expression function that are located inside exosomes, and could play a role in prognosis, assessment of response to treatment or even estimation of aptitude for disease in healthy patients. Hence, it is hoped that they can be used for timely diagnosis, type of the disease determination, and the risk of future illness. Although clinical examination, imaging, CSF laboratory assessment and electrophysiology are measured out for MS dilemma, there are currently no definitive tests for MS evaluation. Serum miRNAs have been identified as powerful biomarkers, not only detect MS patients from healthy controls, but accurately also detect RRMS from progressive forms of the disease. Indeed, the dysregulation of exosomal miRNA in MS patients and their different expression in different subtypes of the disease, has led to their diagnostic features. miR- 370-3P, miR-432-5P were clearly distinguished between the two different groups (RMMS and PPMS clinical subdivisions). miRNAs could greatly improve early detection and determination of disease categories, as well as the conversion of RRMS to SPMS.

P141

The Role of Microglia in Cortical Spreading Depression in Migraine

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Migraine is a disorder that afflicts nearly one tenth of the population. Involving both nervous and vascular system, it has been found as a prominent factor of disability. The migraine attacks may be initiated in the brainstem or may begin peripherally in the meninges while the role of cortical activation preceding an attack is also debated. Although available treatments, more s tudies on migraine pathogenesis is needed to introduce more effective treatments. Microglia realized to have a central role in innate immunity within the central nervous system, by generating inflammatory responses. They develop from myeloerythroid progenitor cells in the yolk sac to make a colony of tissue macrophages in the brain. In patients suffering from migraine with aura, cortical spreading depression (CSD) waves are caused by increased neurological activity, that spread slowly across the cortical surface at a certain rate, leading to transient loss of signaling capabilities. This can happen due to different factors like increasing neuro-transmitter production, such as glutamate, which boosts NMDA activity resulting in an ionic imbalance. Following this, as an immune response, the microglia cells increase the intensity of the wave by giving inflammatory responses through producing substances, like alpha-TNF, and this in turn may lead to induction of CSD. Microglia plays an important role in CSD cyclic pattern in migraine pathogenesis, by producing inflammatory factors. Further studies should be planned to clarify signaling cascades between microglia and CSD for making new therapeutic procedures in migraine treatment.

P142

Inflammation or Neurodegeneration: Which one has Remarkable Role in Multiple Sclerosis?

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Multiple Sclerosis (MS) is a complex disease resulting from the occurrence of intermingled episodes of neuroinflammation and degeneration. However, this concept has recently challenged by several observations suggesting that in this disease neurodegeneration might occur independently of inflammation. The evidence that active neurodegeneration in MS is invariably associated with inflammation is provided. The attack of myelin starts inflammatory processes, which triggers other immune cells and the release of soluble factors like cytokines and antibodies. Further breakdown of the blood–brain barrier in turn causes a number of other damaging effects such as swelling, activation of macrophages, more

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activation of cytokines and other destructive proteins. Inflammation can potentially reduce transmission of information between neurons in at least three ways. The soluble factors released might stop neurotransmission by intact neurons. These factors could lead to or enhance the loss of myelin, or they may cause the axon to break down completely. Neuroinflammation could also found in deep gray matter with pathological and clinical relevance. Therefore, control of inflammation with anti-inflammatory therapies must take into account as one of the main purposes of MS treatment parallel with other immunomodulatory and immunosuppressive treatments. Future therapeutic options for this disease discussed based on recent knowledge of the mechanisms of inflammation and neurodegeneration.

P143

The Neuroprotective Effect of Chloroquine in Animal Model of Traumatic Brain Injury

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Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality in young adults and children, and is a leading public health problem worldwide. In TBI, neurological impairment is caused by immediate brain tissue disruption (primary injury) and post injury cellular and molecular events (secondary injury) that exacerbate the primary neurological insult. However, the destructive molecular events that follow TBI evolve over several days, and therefore there is a window of opportunity during which therapeutic strategies may improve outcome. The antimalarial drug, chloroquine (CQ), has been reported as an autophagy inhibitor in a variety of disorders, including Alzheimer's disease and brain ischemia. To the best of our knowledge, no studies to date have examined the potential for CQ to provide neuroprotection in animal models of traumatic brain injury (TBI). Chloroquine (CQ) has long been used in the treatment and prevention of malaria, and less commonly has been employed in the treatment of autoimmune diseases, due to its immunosuppressive properties. In summary, this study demonstrated that neuronal autophagy was inhibited by post injury treatment of CQ in a rat model of TBI. Furthermore, CQ attenuates secondary brain edema and improves cognitive functioning. These findings emphasize that CQ administered immediately following injury, could be neuroprotective against the damaging consequences of TBI, and we anticipate that this study has shed light on the potential use of CQ as a neuroprotective agent in the treatment of cerebral injuries.

P144

Therapeutic Application of Mesenchymal Stem Cells in Spinal Cord Injury Treatment

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Spinal cord injury (SCI) is a neurologic disorder that have a significant impact on quality of life, life expectancy, and economic burden. SCI leads to irreversible neuronal loss and ultimately leads to paralysis. Mesenchymal s tem cells (MSCs) are a promising source for cellular therapy because they have possessed the capacity of self-renewal and differentiation to several distinct mesenchymal lineages. Mesenchymal stem cells can be derived from a diverse range of tissues but bone marrow, umbilical cord blood, adipose tissue and peripheral blood are the major sources of MSCs. MSCs can reduce inflammatory responses, and cell death following the mechanical trauma. Mesenchymal stem cells are suitable for reducing and minimizing many pathophysiological consequences of SCI. When MSCs are injected at injury site, they secrete a variety of cytokines and growth factors, such as neurotrophic factors, insulin-like growth factor (IGF), vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF-2), and transforming growth factor (TGF). In addition, MSCs increase serum interleukin IL-10 and decrease tumor necrosis factor (TNF). T cells change from pro-inflammatory Th1 cells to anti-inflammatory Th2 cells in the presence of MSCs. Nowadays MSC is novel therapeutic approach in the treatment of spinal cord injury. This promising approach to the treatment of SCI in its nascent stages is facing several challenges. However, further research is needed to better understand of the mechanism of action and the behavior of stem cells in lesion after transplantation to determine the most effective pathway and the best time frame for post-traumatic application.

P145

Vitamins Level Change in Spinal Cord Injury

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Spinal cord injury (SCI) is damage to the spinal cord that leads to sudden loss of motor and autonomic function and sensory under the level of the injury. studies showed that

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individuals with SCI has a clear tendency to vitamins level change. The aim of this study was to review the vitamins level in spinal cord injury. Vitamins C level decrease in the injured spinal cord patient. Vitamins C have major antioxidant functions and play certain roles in the secondary injury response to the direct initial spinal cord injury. Vitamin D insufficiency is common in SCI individuals owing to the presence of many contributing factors including limited sun exposure and intake, use of medication and endocrine perturbations. Vitamin E has major antioxidant functions and this vitamin deficiency was shown in persons with SCI. Vitamin B complex helps to alleviate degeneration in the nervous system and vitamin B1 (thiamine), vitamin B6 (pyridoxine) in combination with vitamin B12 are clinically adminis tered. These vitamins levels have varied with progression of spinal cord injury. Vitamin B12 level in the injured spinal cord have been shown decreasing. Although, effectiveness of oral vitamin B12 treatment has not yet been confirmed in persons with SCI. It is recommended that physicians consider vitamins deficiency in their patients with SCI, particularly in those with neurologic and/or psychiatric symptoms. These symptoms may be reversible if treatment is initiated early.

P146

Role of Essential Trace Elements in Parkinson's Disease

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Parkinson's Disease (PD) is a chronic, progressive, neurodegenerative disorder with motor and non-motor signs and symptoms. PD is caused by idiopathic degeneration of dopamine-producing cells in the subs tantia nigra, located in the midbrain. Recently, Trace elements have been recognized to play an important role in the development of Parkinson's disease (PD). The aim of this review was to assess the role of essential trace elements in Parkinson's disease. Serum zinc and copper have been found did not differ between the PD patients and control group. Its suggested that serum levels of zinc and copper do not play any role as risk factors for PD. Selenium is an important trace element which works as an anti-oxidant substance. studies suggest that Selenium could be involved in the pathophysiology of PD and that the mineral, if used in appropriate doses, could protect against this disease. Iron and cadmium has been suspected to contribute to PD because they are known to promote oxidative damage. Studies showed that in PD patients Fe concentrations were significantly increased. It's have been found that the serum zinc levels were associated with the risk of Parkinson's disease, and low serum zinc levels may be an important risk factor for PD. the parkinsonism and other neurological effects can be resulting from chronic Mn exposure. This study highlights the implication of essential trace elements in Parkinson disease and provide us with the knowledge how the mineral effect on PD.

P147

Postoperative Cognitive Dysfunction and Neuroinflammation Associated with Cardiac Surgery

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One of the incapacitating postoperative morbidities that was seen in all types of surgery is Postoperative cognitive dysfunction (POCD). The elder patients and also cardiac surgery patients (CSPs) are at particular risk. Additionally it was shown that the severity, extension of impairment and the incidence rate of POCD is more in CSPs. POCD is a dementia-like symptoms disorder is characterized by symptoms such as memory impairment, loss of concentration, an inability to plan, and difficulty to switch between tasks. In the CSPs not only the impairment of spatial memory like other kinds of surgery can occur, but also spatial learning and object recognition impair can be observed. Several mechanisms have been suggested for this disorder. Many of studies show the neuroinflammation process involvement. Some possible mechanisms that lead to neuroinflammation are postoperative increase in systemic and hippocampal pro-inflammatory cytokines and macrogelial and mas t calls activation. In mast cells neurovascular unit communication, Mast cells, as the first responders in the CNS, can initiate, strengthen and prolong other responses upon activation. Also they can modulate inflammatory processes in multiple CNS pathologies by their secreted mediators. Also the association between cardio-pulmonary bypass (CBP) and microembolism with POCD and relationship between anesthesia and POCD have remained unknown. It was proved that all types of surgery may lead to neuroinflammation and POCD, especially cardiac surgery. Several probable mechanisms and also the relation between CBP and anes thesia with POCD was discussed, but because they are s till unknown more study is needed.

P148

Facial Nerve Paralysis Secondary to Odontogenic Infection

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Peripheral facial nerve paralysis is the most common form of motor cranial neuropathy. Several factors can cause Bell's palsy such as vascular ischemia, intracranial lesions, iatrogenic damage, etc. Treatment relies on diagnosing the causing factor, varying from steroids to surgical techniques. Since there has been but few reports of facial nerve paralysis caused by dental infection, odontogenic factors may easily be neglected leading to an incorrect diagnosis and inappropriate treatment. Facial nerve paralysis with a dental origin needs further studies not only to determine different causes (such as: impacted molar teeth infection, post surgical infection of extracting impacted molars, root canal treatment, etc.) but also to best manage the situation using a specific treatment protocol.

P149

Statin and Vitamin D as a Prophylactic Medication for Migraine

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Migraine is a primary headache disorder that is characterized by severe headaches and impairment of autonomic nervous system function. This neurovascular disorder ranks as the eighth cause of disability in the world. Migraine is basically an inflammation problem caused by activation of the trigeminal neurovascular complex. Neuropeptides like serotonin, calcitonin gene-related peptide (CGRP) and NO that release from trigeminal fibers cause neurogenic inflammation. Migraine patients usually use abortive drugs for interrupting attacks and prophylactic drugs for preventing. Anticonvulsants, beta blockers, and tricyclic antidepressants are commonly used as prophylactic medications. Although their effects have been proven, they have significant side effects. Therefore, trying to discover new drugs is required. Statin and vitamin D have immunomodulatory effects and also are effective in reducing pain. Statin that is known to be a cholesterol-lowering agent also have cholesterol-dependent effects. Many of these pleiotropic effects are probably applied by inhibiting of isoprenoids which are involved in intracellular signaling pathways. Statin with improving endothelial dysfunction can reduce the risk of vascular disease in these individuals. Regarding vitamin D status, statin has different effects on migraine. This way in people with higher levels of vitamin D is significantly linked to lower prevalence of migraine. Vitamin D supplements can reduce inflammatory factors but there is no evidence that there is a relationship between vitamin D deficiency and migraine. According to the studies reviewed, this way is effective in reducing the period of migraine, the dose of abortive drugs and duration of taking them. No serious side effects have been observed for this medication. Therefore, statin and vitamin D due to their anti-immunomodulatory effect can be studied as a prophylactic treatment for migraine.

P150

The Effect of Periodontitis on Migraine Chronification

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Migraine is a neurovascular disorder that is characterized by unilateral, pulsatile headaches. Migraine due to its individual and social effects is known as a major cause of disability in the world. The main complication of this disease is chronification that is known as chronic migraine (CM). It seems that several factors contribute to migraine chronification such as age, female gender, obesity and depression. Periodontitis (PD) is a common progressive inflammatory disease in the adult population that can cause destruction of surrounding connective tissue and increased loss of alveolar bones. During PD that occurs as a result of interaction between periodontal pathogen and host response, some factors such as interleukin-1 (IL-1), IL-6, C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-alpha) which are produced locally are systemically distributed. This chronic inflammatory condition can be associated with overexpression of neurogenic biomarkers such as calcitonin gene-related peptide (CGRP), substance P (SP), neurokinin A (NKA) in CM. Hypertension, hypercholesterolemia, insulin resistance, stroke and coronary artery disease are a number of comorbidities that are associated with CM and PD. On the other hand, adipocytokines (e.g., leptin) have important role in various physiologic processes such as endothelial function, immune response and inflammation. Several studies have shown that some of them are involved in CM. High level of leptin not only contribute to pathophysiology of migraine, but also its chronicity through systemic inflammation. Chronic increasing in leptin concentration in patients with PD can worsen the inflammatory process of migraine. In conclusion, PD through increased endothelial dysfunction, systemic inflammation and trigeminovascular system activation could be involved

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in process of migraine chronification. Besides that, the altered concentration of adipocytokines may be a biomarker of CM which can be considered as a new therapeutic role for migraine. Although PD is mitigated as a potential factor for CM more evidence is needed to examine the effect of periodontal treatment on CM.

P151

The Effects of Boswellia Serrate on Central Nervous System

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In the process of neuronal inflammation, an increased in inflammatory cytokines (IL-1β, IL-6 and TNF-α) from immune cells (leukocytes and macrophages), brain cells (microglia, astrocytes and neurons) and in hippocampus, amygdala occurs. Raise the level of cytokines result in reduced in production of molecules that are related to plasticity, especially BDNF, IGF-1 and VEGF. Microglia activation lead to suppression of neurogenesis, differentiation of NPCs, decrease in long-term potentiation (LTP) and induction of learning and memory impairment. Also, the phenomenon of nerve inflammation with an increase in the level of TNFα cause inhibition of astrocytes in the removal of glutamate and led to neuronal death. Elevated in TNFα level result in increased activity of the iNOS enzyme that is available in astrocytes in the CA1 hippocampus and it is responsible for increase in oxidant molecules and depression of LTP. Furthermore, the increase in inflammatory reaction mediators result in subsequent neurotoxic consequences. Indeed, inflammatory factors deliberate as a predisposing agent for neurodegenerative disease. Boswellia serrate from Burseracea family, it's resin (Frankincense or Ollibanum) and the main cons tituent of this resin (boswellic acid) play important role in suppression of neuronal inflammation with inhibition of 5-lipoxygenase, Prostaglandin E2 formation and expression of inflammatory cytokines and chemokines. Boswellia serrate and its derivatives with anti-inflammatory properties have therapeutic effects on memory retention, decrease in brain edema, facilitation in nerve impulse, improve the pathogenesis of neuroinflammatory disease like Alzheimer's disease.

P152

Neurotoxicants and Mechanisms Neurodegenerative in Acrylamide

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Many chemicals with broad industrial, pharmaceutical and agricultural application produce a neurotoxic syndrome in humans and experimental animals involving weight loss, skeletal muscle weakness and ataxia. Neurotoxicity is defined as a structural change or a functional alteration of the nervous system resulting from exposure to a chemical, biological or physical agent. Neurotoxicity including Neuronopathia, Axonopathia and myelopathies. The causes of Neuropathies Are Doxorubicin, Methyl Mercury. Axonopathies causes of Gamma-Diketones, β′-Iminodipropionitrile, Acrylamide and Myelopathies causes of Hexachlorophene Tellurium, Lead. Acrylamide (ACR) as a chemical industry is a poison in foods prepared at high temperatures and is the most important neurotoxic agent. Humans that workers in factories are more susceptible for peripheral neuropathies of these toxic agent at high doses. The first symptoms are observed in Pacinian corpuscles, muscle spindles and the nerve terminal. These side effects is result from additions of neurofilaments at the nerve terminal. Developing of Para nodal swellings, cause myelin withdrawal. In additional, Acrylamide lead to sensory axonopathy, Axonal degeneration and peripheral neuropathy. In study mention that, mild ataxia, typical ataxia and hind limb weakness were appeared with 10 mg/kg and 20 mg/kg respectively. The GAP-43 protein marker, is tested for assess of neuronal function that is related to hippocampal neuronal growth, promoting axonal elongation and retaining axonal morphology. Also GAP-43 protein may modulate the transmission of neural signals because of extensively distribution at the axonal terminate. ACR exposure cause inhibition of expression of GAP-43, so may result in disturbance in axonal growth, synaptic terminal vesicles, mitochondria, synaptic inhibition and eventually, terminate the retrograde and anterograde axonal transport. In more recent studies were suggested that ACR, even in its low-dose, can lead to neurological symptoms and nerve terminal degeneration like axonopathy.

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P153

Evaluating the Effect of Lactobacillus Acidophilus Probiotic Supplementation on Sensory-Motor Recovery After a Traumatic Brain Injury

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Traumatic brain injury (TBI) is a common cause of death which affects millions of people around the world. TBI is also associated with various neurological impairments. After the primary mechanical injury at the moment of a TBI event, several cellular and molecular processes are activated within the brain tissue as the secondary injury. An important mechanism involved in the secondary injury of TBI is Neuroinflammation. Therefore, neuroinflammation offers a promising avenue for therapeutic intervention with the aim of preventing progressive neurodegeneration and improving the neurological recovery after TBI. A number of studies have shown the efficacy of probiotics in modulating inflammatory responses; however, it is still unclear if probiotics have the ability to regulate neuroinflammation, and more research is needed to determine the effect of probiotics on neuroinflammation and neurological recovery. If proven beneficial, probiotics offer a non-invasive, safe and cheap therapy for regulating post-TBI neuroinflammation. Multiple studies have demonstrated the ability of probiotics in decreasing inflammatory cytokines while also increasing the anti-inflammatory cytokines. Therefore, we hypothesize that using probiotics after TBI may have the capacity to modulate neuroinflammatory response and as a result, improve the neurological recovery.

P154

Role of Exosomes as Novel Biomarkers in Diagnosis and Prognosis of Glioblastoma

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Glioblastoma multiform(GBM) is the most prevalent primary brain tumor. Exosomes are extracellular vehicles for exchanging information between various cell types including cancer and normal cells. Exosomes are indicative of pathophysiological conditions of brain tumors that could be used in diagnosis and prognosis of GBM. In tumors, exosomes could carry various molecules like several miRNAs and proteins from host cells to recipient cells leading to development of tumor. Exosomes can be isolated from blood serum in different manners. One way is using antibodies against exosomal markers. What makes the exosomes “ideal” biomarkers for clinical diagnosis and prognosis is that exosomal miRNAs and proteins are protected from RNases and proteases respectively, thus can be stably detected in circulating serum. For instance, it has been shown that the upregulated miRNA-326 and miRNA-130a, and downregulated miRNA-323 and miRNA-329 could be associated with long overall survival in GBM patients. Also it is found that circulating miRNA-128 and miRNA-342-3p were positively correlated with histopathological grades of GBM. Moreover, it is indicated that miRNA-24 could be an oncogene and be used as diagnostic biomarker. On the other hand, Serum exosomes from patients with brain tumors possess EGFR, EGFRvIII, TGF-beta, and Tetraspanins which are potentially useful in diagnosis of GBM. There are few studies in term of using exosomes as tumor biomarkers but we claim that the most important advantage of exosomes is their potential to be used as biomarkers for clinical diagnosis and prognosis. Further studies are needed to prove this concept and make it operative in clinics.

P155

The Role of Vegan Diet in Epilepsy

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Food habits have a serious role in the emergence of the diseases and inflammation that causes many kinds of diseases. Some case-control studies concluded that high animal fat and cholesterol in diet is associated with some neuroinflammatory diseases such as Parkinson and Alzheimer. High level of cholesterol and lipopolysaccharides(LPS) include the peripheral inflammation in the body. There is evidence that activation of immune and inflammatory processes occurs in a variety of epilepsies. Also, there are animal trials that show peripheral inflammation can cause the NeuroInflammation and oxidative stress and in this condition, activation of microglial cells and producing inflammatory cytokines have an important

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role in epileptic disorders. LPS-induced Peripheral inflammation evokes NeuroInflammation in the hippocampus and increases the seizure susceptibility. On the other hand, studies show the vegetarianism can obviously reduce the level of inflammation. level of inflammation by the anti-inflammatory effect of vegan diet and some eastern diets which decrease peripheral inflammation and consequently mitigate the neuro inflammation and neuropathy, the main causes of epileptic disorders. It seems vegan diet or diets with a low level of LPS or animal fat can be a reducing factor in epilepsy seizers.

P156

Effect of Air Pollution on Alzheimer's Disease

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Alzheimer, a chronic neurodegenerative disease, usually starts slowly and worsens gradually. It causes 60% to 70% of dementia cases. Difficulty in remembering recent events (short-term memory loss) is the most prominent early symptom. As the disease progresses, patients may have problems such as impaired language, disorientation (including easily getting lost), mood swings, loss of motivation, not managing self-care, and behavioral issues. The potential damaging effect of air pollution on the central nervous system is also investigated and there's mounting evidence of a link between air pollution exposure and neurodegenerative pathologies, especially Alzheimer's disease. Air pollution is thought to increase the risk of neurological diseases by developing neuro-inflammation, oxidative stress, glial activation and cerebrovascular damage. In animal models, contaminated particles can activate microglial cells and increase the secretion of IL-6, IL-1b, and TNF-a, leading to neuro-inflammation. Also, exposure to complex mixtures of air pollutants produces inflammation in the upper and lower respiratory tract and causes systemic inflammation; As a result, neuro-inflammation and neuropathology then lead to Alzheimer. In polluted regions, we expect a higher level of systemic inflammation, prefrontal white matter hyper-intensities, and hallmarks of Alzheimer.

P157

Periostin Recruits Tumor Associated Macrophages in Glioblastoma Multiform

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Glioblastoma multiform (GBM) is the most common and lethal type of primary brain tumors with high rates of morbidity and mortality. Treatment options are limited and ineffective in most of the cases. Epidemiological s tudies have shown a link between inflammation and glioma genesis. In addition, at the molecular level, pro-inflammatory cytokines released from activated microglia can increase proliferation of Glioma stem cells (GSCs) and migration of these cells to the inflamed area. GSCs increase tumor progression and decrease survival with several different mechanisms. One of the genes expressed in GSCs is POSTN. The product of this gene is a matricellular protein named periostin which has a critical role in carcinoma metastasis. This protein is up regulated in glioblastoma. A common feature of GBMs is abundant macrophage infiltration. Tumor-associated macrophages (TAM) have been shown to promote cancer cell proliferation, neo-vascularization and interfere with the anti-tumor functions of other immune cells. TAM density correlates with POSTN levels in human GBMs. POSTN causes an increase in cancer cell proliferation, invasion, TAM recruitment, and angiogenesis. As a result, it can be said that POSTN gene expression promotes GBM progression and it is possible to improve GBM by targeting POSTN gene.

P158

Targeting of Microglial M1/M2 Polarization Through Stem Cells Therapy as A Promising Candidate in Traumatic Brain Injury (TBI)

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Traumatic brain injury is a serious global health problem with irreversible high morbidity and disability and Because of its unknown pathophysiological mechanisms, efficient therapeutic approaches to improve the poor outcome and long-term impairment of behavioral function are still remains lacking. The microglial cells are the resident macrophage cells of the brain and have M1/M2 phenotype, for expression of pro-inflammatory and anti-inflammation cytokines, respectively. The results have been shown that pharmacological inhibiting of M1 phenotype and activating M2 phenotype of microglial cells could relieve cerebral injuries and increase neurological function recovery after Traumatic

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brain injury. Mesenchymal Stem Cells (MSCs), a type of multipotent stem cells, are regarded as promising therapies in several CNS diseases clinical trials. In animal models, transplantation of stimulated MSCs could promote the activation of microglia via transforming the classic M1phenotype into alternative M2 phenotype to inhibits the release of pro-inflammatory cytokines and raise tissue repair after traumatic brain injury (TBI). In this review, we summarized the beneficial effects of MSCs on TBI damaged tissues and their function in regulating the immune system to maintain the CNS. Although, lab trials studies have also confirmed that MSCs are able to promote positive outcomes in TBI models, however, there are still some unanswered ques tions regarding MSCs-based therapy due to complex ethical and safety concerns.

P159

New Findings in The Diagnosis of Autoimmune Diseases of the Nervous System

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The antibody of the nervous system is called antibodies that the body makes to the nervous system cells. These antibodies are also very diverse based on the complexity and diversity of the nervous system, and therefore their detection is also associated with particular challenges. Generally, autoimmune diseases are on the rise. The diagnostic techniques for autoimmune diseases are also in progress, many of the neurological diseases previously classified as idiopathic are now in the autoimmune category. The laboratory has a special role to diagnosis of autoimmune nervous system. There is an important needs for the training of physicians, medical students, and authorities of the medical diagnostic laboratories to identify the pathways for the diagnosis of autoantibody in the nervous system. The antibody of the nervous sys tem is divided into two general categories. Intracellular antibodies that are often tumor-dependent and cause par neoplastic neuropsychological disorders(PNS). The other batch of extracellular antibodies is synaptic, less tumor dependent and often causes encephalitis. Simultaneous use of immunofluorescence, ELISA, immunoblotting, radioimmunoassay is performed on the basis of diagnostic protocols. In this paper, we will review the types of neuroautoanibodies, the physiological structure of their antigens, and the primary ways to detect them, and then confirmed them with applying new methods. Transaction cells with specific antigens also increase the sensitivity and specificity of the methods, which is described. The following table provides an overview of the overall categorization of autoimmune diseases in the nervous system and specific antibodies. Autoimmune encephalitis:Hu, CV2, Ma, Amphiphysin, GAD, NMDAR, AMPA, GABAR, LGI1, CASPR2, DPPX, mGluR5
Autoimmune neuropathies: GM1, GQ1b, MAG, Hu, CV2, Ma, AGNA, Amphiphysin, ANNA-3
Cerebellar syndromes: Hu, Yo, Ri, CV2, Ma, Tr, mGluR1, Zic4, Amphiphysin, ANNA-3, PCA-2, AGNA
Demyelinating diseases: AQP4, MBP, MOG
Myasthenia syndrome: AChR, MuSK, Titin, LRP4
Stiff-person syndrome: Amphiphysin, GAD, GlyR

P160

Effects of Cardiovascular Diseases on Cognitive Impairment in Elderlies

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As increasing in elderly population cognitive impairment such as dementia is increasing dramatically. Alzheimer and vascular dementia are two types of dementia that can be result of cardiovascular disorder. Dementia affects quality of life and life expectancy, thus caring and paying attention to mental and somatic complementation after chronic disease is necessary and may be useful in delay the onset of dementia. The aim of this study is to review the link between dementia and cardiovascular disease. In this review study, we searched PubMed by English keywords such as dementia, eged or elderly, cardiovascular disease, atherosclerosis in the title and elderly in abstract and title, during 2000-2018. We found 55 articles, after reading the abstracts, articles were selected by inclusion and exclusion criteria (2 review, 20 originals and 33 unrelated studies). Results of this study reveals that cardiovascular disease, especially atherosclerosis could lead to dementia in the elderly individuals. Thus preventive and conservative implementation were suggested in this risky group.

P161

A Review of the Effect of Self-Care on the Quality of Life of Patients with Multiple Sclerosis

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Multiple Sclerosis (MS) is a chronic non- traumatic neurotic inflammatory disease that affects the quality of life and has physical, psychological, social, and social impairment. MS affects all economic, social and emotional aspects of the individuals, families and society, thus only medication therapy and control of the course of these patients are not enough. Self- care is taken to increase awareness and assistance to patients to achieve individual autonomy and facilitate their adaptation to the problem of improving quality of life. The present study aimed to determine the impact of self- care on the quality of life of patients with MS. Symptoms of MS include sensory, hearing, visual, speech, psychological, sexual function disorders, and abnormal, and impairment of quality of life. The data from the current study, including the MAGIRAN, SID, and Google Scholar datacenters, were collected from the database, and the related queues were subtracted from the study. The findings show that between self-care education such as improving physical activity, increasing self-efficacy, rehabilitation, exercise, psychotherapy, and interactions Behavioral relationships have a reciprocal relationship with the increase in quality of life. The lowest level of quality of life has been related to the emotional well being. Individual differences, differences in education, the type of education provided, and follow-up of patients have contributed to the increase in quality of life. With regard to the outbreak of MS, the strengthening of educational and support associations, specialist self-care education techniques, and encouraging patients to undertake self-care activities in order to promote health and reduce the cost of treatment can be a step towards the quality of life of patients with MS.

P162

Emerging Perspectives on Mtor-Associated Inflammation in Neurodegenerative Diseases

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Inflammatory processes have been shown to be involved in development and progression of neurodegenerative diseases. Mammalian target of rapamycin (mTOR) involves in various cellular processes including autophagy, apoptosis and energy metabolism. Recently, studies have been shown an association between mTOR pathway and inflammation, supporting the role of the pathway in the pathogenesis of inflammatory disorders including neurodegenerative diseases. There are several studies have been shown that rapamycin, an antagonist of mTOR pathway, or PF-4708671, a mTOR substrate inhibitor, exhibits high neuroprotective effects through reducing inflammation. For example, rapamycin attenuates proinflammatory responses by increasing anti-inflammatory activity of regulatory T cells to restrain post-stroke neuro-inflammation. Moreover, pharmacological inhibition of mTOR decreases neuronal inflammation in cerebral palsy mice model subjected to hypoxia-ischemia and lipopolysaccharide-induced inflammation. Similarly, Liu et al indicated that inhibition of mTOR inhibits amyloid-β or LPS-induced neuro-inflammation in mice models. Consistent with the anti-inflammatory effects of mTOR inhibitors, Ding et al, reported that melatonin negatively regulates the release of proinflammatory cytokines by inhibition of the mTOR in traumatic brain injury in animal models. Taken together, these results clearly suggest that mTOR inhibitors can be considered as a promising therapeutic target to suppress neuronal inflammation in neurodegenerative diseases. Understanding of the exact molecular mechanism of mTOR signaling could be helpful to design a novel mTOR inhibitor to regulate the inflammatory responses in neurodegenerative diseases.

P163

The Anti-Inflammatory Effects of Human Amniotic Membrane Epithelial Cells-Derived Condition Media

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The human amniotic membrane known as the innermos t single epithelial-covered layer provides many applications such as applicable anti-inflammatory and anti-cancer effects. These immunomodulatory effects belongs to the epithelial cells, a type of epiblast-derived fetal stem cells which currently used for regenerative medicine and transplantation. These cells are collected by author-prepared facilities and expanded in 75 cm2 cell culture flask (Biofil) in the DMEM, 12% FBS and penicillin-streptomycin antibiotic incubated in 80% humidity, 5% CO2 for 72 hours. These cell released the special macromolecules modulate the inflammatory pathways so the 2×105 cells were expanded in the 25 cm2 flask and incubated in the standard incubation condition. After 72 hours, the media changed and after 5 days, the cellular supernatant were collected as the conditioned media. The U937 cell line were treated with 50% condition media and standard medium (RPMI 1640, 5% GlutaMax and 10% FBS) for one week. The level of mRNA expression of IL1α and β and IL 8 were evaluated in the U937 cells after 1 week treatment

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with conditioned media. The obtained results illus trated the significant reduction in the IL1α and β and IL 8 cellular expression in the treated cells ($p<0.001$). The conditioned medium obtained from expanded human amniotic membrane epithelial cells has the anti-inflammatory effects based on obtained results on U937 cell line. This properties may provide the promising way in regenerative medicine.

P164

Adeno-Associated Viral Vectors in Duchenne Muscular Dystrophy

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Duchenne muscular dystrophy (BMD) is an inherited X-link disease. The incidence of this muscle-wasting disease is 1:5000 male live births. Mutation in the gene coding for dystrophin is the main cause of BMD. Most cases of this disease succumb to respiratory and cardiac failure in 3rd to 4th decades. The slow progression of BMD and recent achievement of gene therapies make it as an appropriate candidate for this strategy to res tore dystrophin production in most affected tissues. This review has focused on elucidating the role of Adeno-associated viral vectors in duchenne muscle dystrophy. Some strategies in gene therapy of BMD exon skipping, protein upregulation, stem cell transplants and mutation suppression in order to restore dystrophin production. Serious adverse events have been limited them. One of the novel and functional strategy to replace dystrophin is using shuttle vectors derived from adeno-associated virus (AAV). This method has been tested in numerous human clinical trials without life threatening adverse effects. Major limitations of AAV vectors include limited cloning capacity and activation of immune response. Therefore, using miniaturized dystrophin and effective methods in order to attenuate immune system can promote this strategy.

P165

Therapeutic Potentials of Stem-Cell-Based Therapy for Parkinson's Disease; Current Status of Human Endometrium-Derived Mesenchymal Stem Cells

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Parkinson's disease (PD) is a progressive neurodegenerative disease characterized by motor and non-motor symptoms. It is expected to impose an increasing economic and social burden on human populations. The motor symptoms of PD are well known, including age-dependent uncontrollable res ting tremor, bradykinesia, rigidity, posture instability. In the non-motor symptoms, cognitive changes, dementia, behavioral or neuropsychiatric changes, pain and fatigue, autonomic dysfunction, psychosis and hallucinations, sleep disorder, depression, mood disturbances and anxiety occur. Currently, available therapeutic approaches are mainly aiming to relief PD motor symptoms including L-DOPA replacement therapy, administration of DA agonist, and deep brain stimulation, in subthalamic nucleus and globus pallidus via surgically implanted electrodes. All of these therapeutic approaches are palliative and they are incapable for contrary to progression of PD. In recent years, neurons and glia have been generated successfully from stem cells. By the progress of stem cell therapy, expand of using stem cell promise the revolution of medical therapy for neurological disorders like PD. The recent upcoming research for PD treatment using human endometrium-derived stem cells (HEDSCs) has unveiled in bringing stem cell technology in the expected future in the form of disease modeling and s tem cell therapy. HEDSC represent a new cell source for neurological disorders, which is abundant and can be easily isolated by a simple, safe, and painless procedure such as Pap smears. HEDSCs have ability for use as an autologous or allogenic stem cell source, so resolve concerns regarding rejection in human beings. HEDSCs are a highly inducible source of allogenic stem cells that can rescue dopamine concentrations in PD animal model. Stem cells have become attractive candidates for cell therapy in neurological disorders including PD. Stem cell therapy especially with HEDSCs shows a promising technology for PD treatment in which more advanced research should be done in creating ways to tackle the disease.

P166

The Role of Interlukin-6 of Immune Cells in Neuronal Dysfunctions in the Autism Disease

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About thirty years ago, the interlukin-6 (IL-6) which is the proinflammatory cytokine, was detected as the differentiation factor of B cell. IL-6 is able to induce maturation in B cells and as a result, B cells achieve the ability to produce antibodies. In addition to immune responses, the role of IL-6 has been known in neurogenesis (neurons and glial cells). The studies have showed that the abnormal immune responses are associated to the autism. In the autism disease, the levels of cytokines increase in blood, brain and cerebrospinal fluid (CSF). Conclusion: Increased levels of IL-6 in the brain of mouse are related to the autism properties such as abnormal cognitive abilities, loss of learning, anxiety, abnormal habits and reduction of social behaviors. Furthermore, Increased levels of IL-6 are associated to preventing of the transmission of inhibiting/inducing synaptic secretions. Also, IL-6 results in abnormal changing in the shape, length and distribution pattern of dendritic cells and can be neurotoxic. Findings show that the increase of IL-6 in the brain can relatively mediate the autism-like behaviors which are created by unbalancing the neuron circuits and dysfunctions of synaptic formations. High expression of IL-6 in several major brain diseases and animal models shows that it can play an important role in neuropathology and that is why IL-6 is the target for strategic treatment.

P167

Key Role of Inflammation in Central Nervous System Damage and Disease; TNFα, IL-1

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Inflammation is portion of the body's immune response and it is basically a host protective response to tissue ischemia, injury, autoimmune responses or infectious agents. Although the information presented so far points to a detrimental role for inflammation in central nervous system (CNS) disease, it may also be useful. CNS demonstrates characteristic of inflammation, and in response to damage, disease or infection, resident CNS cells generate inflammatory mediators, including prostaglandins (PGs), pro-inflammatory cytokines, free radicals and complement, which in turn induce chemokines and adhesion molecules, recruit immune cells, and activate glial cells. In response to a brain injury, astrocytes become activated, increasing expression of glial fibrillary acidic protein, and producing cytokines. Cytokines including both tumor necrosis factor-α (TNF-α) and IL-1 are strongly implicated in neuronal loss during acute and chronic neurodegenerative disease, but also participate in repair and recovery. Although TNF-α is found associated with active MS lesions, induces death of oligodendrocytes. TNF-α appears not to be needed for mast cell–dependent pelvic pain. TNF-α, is released from Schwann cells immediately after nerve damage. IL-1 can also attach nerve terminals and influence substance P release and migration of polymorphonuclear White blood cells (WBCs). IL-1β is also selectively upregulated in astrocytes in the spinal trigeminal nucleus, spinal cord and rostral ventromedial medulla in models of inflammation, cancer pain and nerve damage. IL-1β is an important messenger between neurons and glia.

P168

Delayed Imatinib Treatment for Spinal Cord Damage; Role of Serum Biomarkers and Recovery

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With no routine accessible medicine intervention for spinal cord damage, there is a demand for more remedial candidates. However, for Imatinib to have translational price, it requires to have encouraged obliging effects with delayed start of treatment, as well. Serum levels of 3 chemokines/ cytokines, MIP-3α, MCP-1, and GRO/ KC (IL-8), to raise over time with Imatinib treatment and to be obviously higher in damaged Imatinib treated animals than in manages pending the early treatment period. Lymphoid organs, first the spleen, were tested to supply information on systemic effects of Imatinib with consider to inflam- matory responses and lymphoid organs as the source of monocyte/ macrophage infiltration into the damage site of the spinal cord. Serum samples at one, three and seven days after damage were tested for INF-1β, MIP-3α, MCP-1, IL-2, IL-4, IL-5, IL-6, IL-10, IL-13, GRO/ KC and TNF. Three cytokines/ chemokines, MIP-3a, MCP-1 and GRO/ KC demons trated definite potential as biomarkers in serum. Serum levels of MIP-3α and MCP-1 were raised one day after damage or sham surgery. GRO/KC concentrations were instead higher in the sham group compared to the contusion-damaged groups without or with Imatinib treatment, making this chemokine a potential serum

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biomarker for CNS damage. At one day after surgery, there was however no strong effect of Imatinib treatment of animals with spinal cord damage among the tested chemokines. Serum concentrations of MCP-1 and MIP-3a remained elevated in the damaged and sham damaged group throughout the seven days in comparison to concentrations in uninjured managements.

P169

The Role of Lymphocytes in Spinal Cord Injury and Pain; T Helper Cells (TH1 and TH2 Cells)

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Lymphocyte is one of the subtypes of white blood cell (WBC) in immune system. Lymphocytes contain T cells, natural killer cells , and B cells. They are the head type of cell found in lymph, which for this reason the name “lymphocyte”. Lymphocytes can be recognized by their large nucleus. Infiltration of immune cells in the central nervous system (CNS) helps the start of chronic pain. CD4+ T cells infiltrate into the spinal cord, whereas B lymphocytes and NK cells are not locate in the spinal cord after L5 spinal nerve cross section. T cells infiltrate the sciatic nerve and dorsal root ganglion after nerve damage. Hyperalgesia and allodynia influenced by nerve damage are typicaly attenuated or abrogated in rodents missing T cells and the immunosuppressant rapamycin attenuates neuropathic pain in rats, partially due to an effect on T cells. Type 1 and 2 T helper cells (TH1 and TH2 cells) are subsets of T cells and have been demonstrated to have variety roles in neuropathic pain. TH1 cells help neuropathic pain behavior by secrete proinflammatory cytokines (interferon-γ (IFNγ) and IL-2), whereas TH2 cells block it by secreting anti-inflammatory cytokines (IL-13, IL-10 and IL-4). It is noteworthy that the condensation of IL-17 in the spinal cord of rats is rised after nerve damage.

P170

The Role of Th1 Lymphocytes in The Pathogenesis of Multiple Sclerosis (MS)

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Th1 lymphocytes produce cytokines such as IL-2, IFN-γ, and TNF-α, TNF-β and GM-CSF. IFN-γ is the most important Th1 cell cytokine that induces the production of IgG, activation of macrophages, enhancing phagocytosis, and also increasing MHC class I and class II molecules. Increasing serum level of Th1 cytokines have also been observed in MS patients. It has also been proven that in humans, exacerbation of MS disease is often accompanied by the increase of myelin-specific Th1 cells in the CSF and according to pathological observations, in thrombolytic plaques, the accumulation of Th1 cells and the production of IFN-γ is directly linked to the demyelination process, which also proves the pathogenicity of Th1 cells. Moreover, the treatment of multiple sclerosis with IFN-γ increases the severity of the disease; while treatment with an anti-IFN-γ antibody improves the disease. Th1 cells cytokines activate macrophages, and activated macrophages cause damage to myelin and subsequently oligodendrocytes and can also produce other inflammatory cytokines that can exacerbate tissue damage. Conclusion: According to studies, Th1 lymphocytes seem to play an important role in immuno-pathological reactions in MS. Preventing the entry of Th1 cells into the CNS, differentiation of native T-cell into Th1 cellsand also activation of Th1 cells, and in the other hand targeting cytokines secreted from Th1 cells or their receptors can significantly reduce the process of demyelination in MS. This review study aimed to investigate the role of Th1 Lymphocytes in the Pathogenesis of Multiple Sclerosis.

P171

Microglia Cell, Major Player in the Central Nervous System Inflammation

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Inflammation, a self-defensive reaction against various pathogenic stimuli, may become harmful self-damaging process. Increasing evidence has linked chronic inflammation to a number of neurodegenerative disorders including alzheimer’s disease (AD), parkinson’s disease (PD), and multiple sclerosis (MS). In the central nervous system, microglia, the resident innate immune cells play major role in the inflammatory process. Although they form the first line of defense for the neural parenchyma, uncontrolled activation of microglia may directly

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toxic to neurons by releasing various substances such as inflammatory cytokines (HMGB1, IL-1β, TNF-α, IL-6), NO, PGE and superoxide. Our recent study demonstrated that activated microglia, example BV2 cell, phagocytose not only damaged cell debris but also neighboring intact cells. These cells originated from yolk sac and fetal live in embryonic stage and after birth from bone marrow. Microglia bears some kinds of pattern recognition receptors (PRR) including TLR4 that can recognize pathogen associated molecular pattern(PAMP) and damage associated molecular pattern (DAMP). One of the most important items for PAMP is LPS which included in cell wall of bacteria especially gram negative bacteria. Moreover, LPS can be used by researchers in order to induction of inflammatory situation. It further supports their active participation in self-perpetuating neuronal damaging cycles. Besides, these interesting cells bear mannose and scavenger receptor for phagocytosis.

P172

Study the Effect of Carbamazepine During the Epileptogenesis by Dorsal Hippocampal Kindling on Balance and Locomotor Activity in Adult Male Rats

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Epilepsy is a chronic cerebral disorder associated with recurrently occurring seizures resulting from over activity of brain neurons. Since more than one percent of the world's population is suffering from epilepsy, this disease is recognized as one of the most important neurological disorders in modern medicine. Studies indicate that impairment in balance and motor activity are known as one of the side effects of epilepsy. Therefore, the use of an antiepileptic drug such as carbamazepine can help to improve these disorders. So, in this research, the effect of carbamazepine during epileptogenesis in dorsal hippocampal kindling on balance and motor activity in adult male rats was investigated. In this study, 60 adult male rats were randomly divided into 6 groups: surgical control, methylcellulose (MC), Kindled, carbamazepine (CBZ), kindled-carbamazepine (KCBZ) and the methylcellulose-Kindled (MCK). Animals in the Kindled group stimulate were rapidly kindled by daily stimulation of dorsal hippocampus (12 stimulation per day, 1 ms pulse duration at 50Hz for 3 seconds) in the dorsal hippocampus region(CA1). While animals in the control groups did not receive any stimulation. Animals in the CBZ group received 8 mg of carbamazepine intraperitoneally on the first day after the recovery. The CBZK-group, in addition to receiving 8 mg of carbamazepine on the first day after recovery, received kindling stimulations for 6 days, as the same method with Kindled group. The MCK-group was similar to the CBZK-group with this difference that they received MC instead of CBZ. At the end of kindling stimulation, open field and rotarod tests were respectively used to examine the effect of CBZ on motor activity and balance. The open field test results showed a significant decrease in the motions and rearing frequency in the CBZK-group compared to the MCK-group ($p < 0.01$). Frequency of rearing and motions in the Kindled group also showed a significant increase compared to the control group ($p < 0.01$). Grooming in the CBZK-group compared to the MCK-group show significant decrease ($p < 0.05$) and in the Kindled group showed a significant increase compared to the control group ($p < 0.05$). In the Rotarod test, the balance in the Kindled group was significantly lower than the control group ($p < 0.05$) and in the CBZK-group compared to the MCK-group show significant increase ($p < 0.01$). It seems that Carbamazepine injection during the epileptogenesis by dorsal hippocampal kindling in male rats reduces motor activity but improves balance.

P173

LPS Preconditioning Declines Oncogenic Factors and Inflammatory Responses in PC12 Cells

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The release of molecules from injured tissue leads to produce inflammatory response that can result in apoptosis and cell death. Preconditioning (PC) can decrease the inflammatory response, increase neuroprotective mechanism on different levels. So, we investigated the role of PC as a suitable preventative approach in neurodegenerative disease in and inflammatory oncogenic factors in PC12 cells. So, we treated differentiated PC12 cells with ultra-low and high doses LPS 3μg/ml and 750μg/ml respectively. Our results showed that C-myc and IL-1β, were significantly increased in high dose LPS respect to the control. In addition, C-myc was enhanced despite the inhibition of apoptosis even if cells were treated by high dose LPS. But results have shown that C-myc level was markedly reduced in presence of PC induction respect to the high dose LPS group. C-myc in the PC group in compare with the control has shown no significant difference.

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Despite of the apoptosis inhibition in the PC group, C-myc level was not significantly increased. Further evidences have shown that IL-1β in the preconditioned cells were significantly decreased in compared with high dose LPS group and PhosphoSer46P53/P53 significantly decreased in PC group in presence of the apoptosis inhibitor compared with PC group. It has concluded that PC could be effectively reduced the level of inflammatory responses and oncogenic factors. Some PC agent like ultra-low dose LPS causes gene reprogramming which can induce neuroprotection and decrease proinflammatory responses.

P174

Expression of Cannabinoid Receptor 1 (CB1) in Animal Model of Multiple Sclerosis (EAE) Treated with Hemp Seed Oil

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Multiple sclerosis (MS), as an auto-immune disease, is confined to the central nervous system and is usually accompanied with debilitating condition in MS patient. The rate of disease in the females is more than males (2:1), and it is diagnosed between 20 to 40 years old. Phototherapy, as a traditional remedy, is used to treat different pathological conditions including MS. Hemp seed oil, as an herbal drug, is used in this study to alleviate the symptoms EAE as an animal model of MS through alteration of the gene expression of Cannabinoid receptor 1. In this study 24 female C57bl/6 mice randomly divided into three groups: healthy group (group 1), Control group (group 2) and experimental group or hemp seed oil group (group 3). Immunization of all mice after one week acclimation in laboratory environment has been induced with Hooke kit except group 1. One day before of induction, feeding with hemp seed oil initiated and continues for 4 week in standard condition. Clinical score recorded daily through the study and in 28 days after immunization, all mice sacrificed after ketamine/xylazine anesthesia and spinal cord tissue removed for molecular and histopathological evaluation. Data analyzed with SPSS and ML win and P value <0.5 determined as significant. We observed significant differences in clinical scores between the control and experiment groups (p values < 0.001). Also, the expression of CB1 showed a statistically significant increase in the experiment group. In the present study, Hemp seed oil, due to its immunological effects, caused a decrease in the levels of inflammatory factors during the progress of multiple sclerosis in EAE animal model. Moreover, the clinical findings confirm the above result, showing a decrease in debilitating conditions of the disease. Therefore, administration of hemp seed oil alleviates the symptoms of the disease and it is useful for control of the inflammatory and auto-immune diseases. Nevertheless, it seems that additional research is needed to confirm the findings by clinical trials.

P175

Neonatal Homocysteine Administration Induces Cerebellar Toxicity Via Oxidative Stress in Rats

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Several in vitro studies have showed the neurotoxic effect of homocysteine (Hcy) to various neuronal types, including cerebellar Purkinje neurons. In this study, we investigated whether the Hcy has neurotoxic effects on the rat pup's cerebellum in the postnatal period that it continues to development. Pups rats divided into control and Hcy group. Hcy administration (0.3–0.6 mmol/g body weight) was initiated on postnatal day (PD) 4 and continued until PD 25 by subcutaneous injection twice on a day with 8 hours interval. On PD 25, the animals were decapitated and the cerebellums were removed. The body and cerebellum weight were measured and the cerebellum to body weight ratio was calculated by dividing the cerebellum weight by the body weight of pups at sacrifice. Level of lipid peroxidation, the glutathione peroxidase (GPx) activity in the cerebellum were determined. Also, caspase3 protein level was assayed by Elisa kit in the cerebellum. Our results demonstrated that chronic administration Hcy significantly decreased GPx activity and increased lipid peroxidation in the cerebellum. The cerebellum weight and the cerebellum to body weight ratio significantly reduced in Hcy treated rat compared with the control group. Moreover, Hcy increased caspase 3 protein levels in Hcy treated group. Our results showed sys temic administration of Hcy induces cerebellar toxicity through oxidative stress.

P176

Neurological Diseases: Causes, Symptoms and Treatments

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The nervous system is an extremely complex communication system that can send and receive large amounts of information simultaneously. The nervous system has two distinct parts: the central nervous system (the brain and the spinal cord) and the peripheral nervous system (the nerves located outside the brain and spinal cord). The main unit of the nervous system is neural cells (neurons). The routine work of nerve cells is to increase or decrease the number of connections they have with other neurons. This process may somewhat explain how people learn, adapt, and shape their memories. But the brain and spinal cord rarely produce new neurons. Many people refer to neurologists when they have complicated or very uncommon complications of the brain and the nervous system. Neurologists have expertise in the treatment of all types of brain and nervous sys tem disorders. They, in collaboration with a team of physicians and other professionals, determine the bes t treatment option according to your needs. They treat the disease by examining the disease. Neurologists, in addition to the complications listed below, also examine people with signs of neurology or complications from other diseases.

P177

The Human Microbiome and PTSD, the Mechanisms of Interaction-A Narrative Review

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Present therapeutic methods for PTSD are not efficient enough to reduce or disappear all the symptoms. several peripheral factors can affect developing and treating PTSD, such as human microbiota. There is a growing volume of evidence showing the effect of gut microbiota on brain and behaviour.PTSD is associated with an inflammatory state in blood, brain and cerebrospinal fluid.also, there is evidence showing that the gut microbiota can affect the pathogenesis of mental diseases with the mechanism of inflammation. so, the inflammation can be considered as a mechanism of interaction between microbiota and PTSD. PTSD is along with sleep disorders and there are evidence showing that by treating the sleep disorders, we can have a general improvement in PTSD symptoms.also it is shown that intestinal dysbiosis can cause sleep disorders and by adding probiotics to the dysbiosed rats, their sleep disorder improved, so it can be considered as an evidence on the efficiency of altering the gut microbiota on treating PTSD. Also, it is shown that oxytocin can decrease the anxiety and depression, and its intranasal usage after exposing to the traumatic event can decrease the probability of affecting PTSD. on the other hand, there is some kind of probiotics that can increase the oxytocin in the blood.so by altering the gut microbiota with this probiotic, we may have an improvement in PTSD symptoms. gut microbiota may have a key role in both predisposing people to PTSD and also PTSD treatment, so we propose more researches on the therapeutic interventions with the approach of altering the gut microbiota.

P178

Can Human T-Lymphotropic Virus Proviral Load Predict the Severity of Clinical Features in HAM/TSP Patients?

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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. The indirect involvement of the nervous system by lymphocytes is more probable than the direct attack of the virus to the neurons. The proviral load (PVL) is defined as the percentage of HTLV-1–infected peripheral blood mononuclear cells (PBMCs). We reviewed the literature to understand if the PVL could predict the severity of clinical symptoms in HAM/TSP patients. Studies show that the virus proviral load in PBMCs can differentiated asymptomatic carriers from HAM/TSP patients. A significant association has been demonstrated between higher HTLV-1 proviral load and poor long-term prognosis. One study has presented a diagnostic model for the early detection of HAM/TSP using plasma SPARC, VCAM1, and HTLV-1 viral load. Another study has suggested that a high ratio of proviral DNA load in CSF to peripheral blood mononuclear cells (PBMCs) may distinguish HAM/TSP from HTLV-1-infected patients with MS. Also, HTLV-1 proviral loads measured in the CSF of HAM/TSP patients are typically

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greater than twice the proviral load in PBMCs, whereas the ratio of CSF to peripheral blood HTLV-1 proviral loads are typically lower in asymptomatic carriers. In general, the association between PVL in PBMCs and the severity of neurologic symptoms of HAM/TSP patients has not been reported in any available literature and it is necessary to further investigate this issue.

P179

The Effect of Sports Exercises for Eight Weeks on the Rate of Fatigue and the Quality of Life in Patients with Multiple Sclerosis

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MS illness is a self- safety and inflammatory illness in Central Nervous System in which Myelin Sheath and Axon of neuronal cells of the brain and the spinal cord is destroyed by the Safety System, and the level of its prevalence is two or three times more in women rather than men. The most important complications of this illness are fatigue, cramp, shake, the lack of balance, and walking disorder. These factors cause the sharp drop of the level of quality in these patients' life, and it results in reduction of their daily activities. This study aims at investigating the effect of exercise on the rate of fatigue and the quality of life in patients with Multiple Sclerosis. The performance of sports exercises proportional to the ability of patients with MS can decreases the cost of Physiology, and therefore it can improve the rate of fatigue and the level of quality in their life if the symptoms such as thermal sensitivity and fatigue isn't exacerbate. Thus, it is recommended to do these exercises as an effective and time-consuming way, because it has the ability for learning and it is easy to perform. Regarding these results, the respective experts can use these exercises as a complementary along with medical treatments for patients with MS, and they can prevent these patients' increasing and quick disability.

P180

The Effect of Complementary Medicine on Treatment and Prevention of MS Illness in Animal Model

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Multiple Sclerosis is a chronic neurological disease that is accompanied by inflammation and myelin deficiency, and this pathological myelin disorder is irreversible. The prevalence of this disease is in 20-40 ages, and the rate of its prevalence in women is 1.7 times more than men. Yet, no effective medicine has been offered for this illness, and the present treatment methods are costly. Regarding this issue, the researchers have been attracted towards the complementary medicine for treatment of this disease. The results show that getting infected by MS disease depends on genetic and environmental factors; for example, in some regions in which the consumption and absorption of D vitamin is high, the level of its prevalence is low.In addition to medical treatment of MS illness, complementary medicine (such as the effects of Aloe Vera Gel on the changes of Es trogen and Progesterone Hormones, the impact of bee venom on serum level of Intercolein6, edible Genistein extracted from Soya and D3 vitamin) has remarkably affected the treatment and prevention of MS disease in animal model. Of course, clinical use of this medicine in treatment is a complex principle, as its effects are relative, and they are not yet known.

P181

Effect of Aquatic Therapy on Improvement of Patients with Parkinson Disease: A Systematic Review

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Parkinson disease (PD) is a degenerative neurological disorder that affects 0.5- 1% of old people in wes tern countries. It primarily affects mobility function, and associated with increasing disability over time. People with PD express bradykinesia, rigidity, tremor, progressive postural instability and muscle weakness as symptoms of PD. Various exercise programs, including resistance or aerobic training, physical therapy and other complementary therapies have been suggested to address movement deficits in order to improve mobility function and quality of life for individuals with PD in the moderate or early stages. A special aquatic program is able to reduce joint rigidity and limb bradykinesia,

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decrease pain and improve quality of life. According to the results, aquatic exercise therapy for patients with PD has become a recent focus of attention. Aquatic exercise therapy is an enjoyable, feasible and safe method to improve quality of life in people with Parkinson disease. It seems that we can utilize aquatic exercise therapy for improvement of people with Parkinson disease. The impression mechanism of aquatic exercise therapy is unknown and needs to more research.

P182

Legal Issues Related to Nervous System Inflammation

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Every day, complaints are filed against the medical s taff due to Negligence with health and nervous system inflammation -related issues. Negligence in the legal sense, means that one does not perform assigned task. The task that the legislator has placed on that person. The legislator in the Islamic Penal Code, adopted in 1392, Defines negligence as carelessness or omission and also includes lack of skill and non-compliance with state regulations as part of this. Omission: Nonperformance of an act which scientifically and technically is expected to be carried out. eg: Non-administration of antibiotics for bacterial meningitis. Carelessness: Performance of an action which scientifically and technically should not be done. eg: Prescribing antibiotics for a patient with a viral nervous system inflammation. Lack of skill: Includes cases in which the physician does not have the scientific and technical expertise necessary for a certain work. eg: Incorrect lumbar puncture. Failure to comply with government regulations: Namely, failure to pay attention to regulations, departmental letters, regulations of administrative superiors, medical system, Ministry of Health eg: Not having an autoclave at the health center. It is hoped that compliants against medical staff will be prevented by complying with legal requirements.

P183

Key Function of Complement System in Interactions between Pain and Nociceptors, C5a, and C3a

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A part of the immune system that improves (complements) the ability of antibodies and phagocytic cells to clear microorganisms and injured cells from an organism, attacks the pathogen's cell membrane, and encourages inflammation called complement system. It is main part of immune system. Over thirty proteins and protein pieces compose the complement system, including cell membrane receptors, and serum proteins. The complement system activates by 3 biochemical pathways: the alternative complement pathway, the classical complement pathway, and the lectin pathway. The complement system is a main portion of the innate protection. Effectors of the complement cascade attack microorganisms, activate basophils and mas t cells, and promote chemotaxis of white blood cells (WBC). The complement system also has a function in inflammatory hyperalgesia and neuropathic pain. C5a, an anaphylatoxin, is a main effector of the complement cataract and upon banding to C5aR1 receptors on neutrophils it develops a potent neutrophil absorbent. Complement segments also have a direct effect on nociceptors. Injection of C3a and C5a into the hind-paw of mice or rats influences behavioral hyperalgesia. Using of C3a or C5a to peripheral nerves ex vivo sensitizes C fiber nociceptors. This effect might be mediated by a direct effect of banding C5a receptors. C5a activates spinal microglia in neuropathic pain and C5a block of the complement cascade in the spinal cord reverses neuropathic pain behavior and is also participated in neuropathic pain.

P184

Combination of Herbal Medicine and Nanomedicine: a Novel Therapeutic Target for Neurodegenerative Diseases

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Inflammation is a pathologic condition that includes a wide range of diseases namely neurodegenerative diseases. Several natural anti-inflammatory components have been identified in plant extracts used in traditional medicine for the relief of inflammation. Herbal medicine is showing difficulty in crossing the blood-brain barrier (BBB). So that the ability to pass the BBB is the main

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concern for using them at neurodegenerative diseases treatment. Nano-Liposomes and Noisomes provide a unique opportunity to deliver pharmaceuticals into the cells and interaction with the target site. So they have been considered as Nano-carriers for brain drug targeting and overcoming the BBB and transition anti-inflammation herbal medicine namely curcumin for neurodegenerative disease curing.The aim of this review is to explore the different approaches studied to transport and deliver herbal medicine to the brain by using liposome and noisome systems as a carrier. This review has analyzed the most recent approaches for herbal medicine delivery to the central nervous sys tem (CNS). The overall literature clearly shows that herbal medicine needs a modern delivery system for treatment of neurodegenerative disease. Several sys tems have been used to deliver drugs to the brain, such as using peptides, antibodies, and RNA aptamers for optimizing targeting ability liposomes and noisome for passing BBB. It can be concluded that the development of liposomes and noisome for brain delivery are still in their infancy, although these systems have the potential to revolutionize the ways in which medicine is ordered.

P185

Survey Effect of Histamine on Microglia in Neurodegenerative Diseases

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Neurodegenerative diseases contain Multiple Sclerosis (MS), Alzheimer's disease (AD) and Parkinson's disease (PD), are characterized by neuronal death and neuronal degeneration in specific regions of the central nervous system (CNS). Microglia are the basic immune brain cells and play a role in homeostasis after inflammation challenge. Microglia involves in Neurodegenerative diseases, neuroinflammation and microglial activity are the common features of the neuropathy. Histamine is a biogenic amine acting as a major in the modulation of innate immune responses. Source of histamine in brain includes neurons, mast cells, and microglia. His tamine regulates NO factor in SN microglial cells. Histamine modulates cytokine release and microglial migration. Histamine is viewed as the main player in the pathogenesis of neurodegenerative diseases and physiologic activities. Though all receptors (H1R, H2R, H3R, and H4R) are presented in the CNS, H3R is the treatment target for the psychiatric and neurologic disorder. H4R modulate the immune response to inflammation. No specific therapeutic agent is available to restore the damages as the disease is not understood. Effective drugs only reduce the severity of symptoms. They limit neuroinflammation in PD and MS patients. Chorionic neuroinflammation is very important in the onset and progression of the Neurodegenerative disease. Neuroinflammation is the supportive response in the brain, but too much inflammatory responses lead to neuronal regeneration inhibition. We aimed to explore the role of histamine in ROS production and modulate microglial function, phagocytosis action, increasing cell motility making to death of dopaminergic neural cells. Altogether, histamine as a target to make the new treatment for Neurodegenerative diseases.

P186

Efeect of Morphine State-Dependent Memory on Pentylenetetrazole in the Rat

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It has been shown that pre-test systemic administration of morphine was able to reverse memory impairment induced by pre- or post-training morphine in an inhibitory avoidance paradigm. Since the recall of the learned information is possible only if the subject is in the same state as during the encoding phase, this kind of learning is known as state-dependent learning. Several drugs have been demonstrated to replace the pre-tes ting effect of morphine on the restoration of memory. Pentylenetetrazole (PTZ) has been shown to affect many processes involved with central nervous system functions including memory. Considering the above variables, PTZ has been reported to impair or to enhance memory Several hypotheses have been proposed to explain the acute effects of PTZ on memory in the laboratory animals. Morphine (2.5, 5 and 7 mg/kg, i.p.) was adminis tered as pre-/post-training and 24 h later as pre-test drug, and the latencies (Sec.) were measured for rats. PTZ (60 mg/kg, i.p.) was administered before the pre-test morphine. The step-through inhibitory apparatus in two train and test sessions were used for memory assessment. One-way ANOVA was used for studying the difference between the groups in step-through experiments. Pos t-training i.p. injection of morphine (2.5–5 and 7 mg/ kg) dose dependently reduced the step-through latency, showing morphine-induced amnesia. Amnesia induced by post-training morphine was reversed by pre-test administration of morphine (2.5, 5 and 7 mg/kg, i.p.) and induced morphine-state-dependent learning. Pre-test injection of PTZ (60 mg/kg) by itself significantly impaired the memory retrieval ($P<0.001$). However, pre-test administration of PTZ potentiated morphine state-dependent learning ($P<0.001$). The results indicate that PTZ mechanism participate in the facilitation of morphine-induced recovery of memory, on the test day.

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Dose-Dependent Delay of Wallerian Degeneration Induced by Dexamethasone after Sciatic Nerve Transection in Rat

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Wallerian degeneration is an innate immune response which occurs after some kinds of nerve damages such as neurodegenerative disease and acute axonal injury. In clinic, dexamethasone as an anti-inflammatory drug has been used for many years to treat nerve injuries. In present study, we aimed to study the effects of various doses of dexamethasone on the Wallerian degeneration in rat. Twentyseven male Wistar rats were divided into three experimental groups; A, B and C (n=9). Right sciatic nerve was transected and animals in groups A and B were received intraperitoneally low dose (0.2 mg/kg/day) and high dose (2 mg/kg/day) of dexamethasone, respectively. The group C was considered as control (saline, 1ml/day). Five, 7 and 10 days after surgery, 3 rats in each group were sacrificed, the distal stump of sciatic nerves were collected and semi-thin sections were sampled for histological assessment. Morphometric analysis showed a significant increase in the number of myelinated nerve fibers and a significant decrease in the diameter of nerve in group B when compared with group C ($P < 0.05$ at days 5, 7 and 10), whereas there was no significant difference between groups A and C. Our findings suggest that dexamethasone treatment, in a dose dependent manner, reduce the severity of Wallerian degeneration and delay the clearance of myelin debris after peripheral nerve injury.

P188

The Role of Cryotherapy in Progression of Brain Stroke

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Stroke is a leading cause of mortality and morbidity in developed countries and has increased incidence due to progression of average population age. Pharmalogical and mechanical reperfusion therapy, as primary therapeutic approaches, are only applicable to less than 10% of patients with a 50-70% efficacy. but about 90%of patients are severe restricted to these treatments. Glutamate excitotoxicity is associated to the deleterious effects of hyperthermia during the acute phase of brain stroke; therefore management of body temperature is becoming one of the most promising neuroprotective strategies during the acute phase of s troke for patients with resistance to routine treatment. on this subject researches have shown a direct correlation between increase Glutamate concentration in blood that is reflected in an increase of extracellular Glutamate levels on the ischemic brain. Glutamate-Oxaloacetate transaminase (GOT) is a blood-borne enzyme. Glutamate and Oxaloacetate are competitive substrates for this enzyme. so it seems that reduction of temperature or competitive inhibition of GOT, can eliminate Glutamate related damage in brain stroke.

P189

The Role of Periodontitis in Alzheimer Pathogenesis

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Alzheimer disease (AD), the most common cause of dementia, is an irreversible progressive neurodegenerative condition. It is characterized by the salient inflammatory features, microglial activation and increased levels of pro-inflammatory cytokines which contribute to the inflammatory states of the CNS. Accumulating data suggest the key role of inflammation in AD pathogenesis. In a prospective longstanding study, increased level of serum Tumor Necrosis factor alpha (TNF-alpha) and C Reactive Protein (CRP) following both acute and chronic systemic inflammation is associated with cognitive decline in AD patients. Periodontitis is the most common oral infection which is initiated by gram-negative bacteria like spirochetes. It's associated with a raised serum pro-inflammatory s tate with increasing in (CRP), Tumor Necrosis Factor-alpha, interleukin1 and interleukin6 levels. Periodontitis can lead to progression of the AD through two probable mechanisms; Pro-inflammatory cytokines produced by periodontopathic microorganisms and host response to a systemic/peripheral inflammation. Inflammatory molecules can pass the blood-brain barrier to activate microglia cells which results in neural damage. The second mechanism is due to brain invasion by microorganisms present in dental plaque biofilm. They can enter brain either through the bloodstream or via

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peripheral nerves. Although lacking causal relationship between periodontitis and AD, periodontitis may be accounted as one of the possible risk factors for perpetuating the neurodegenerative process of the AD. Cohort studies profiling the oral clinical pre-sensation with different cognitive functions and during the progression of the AD is needed to clarify this casual association.

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