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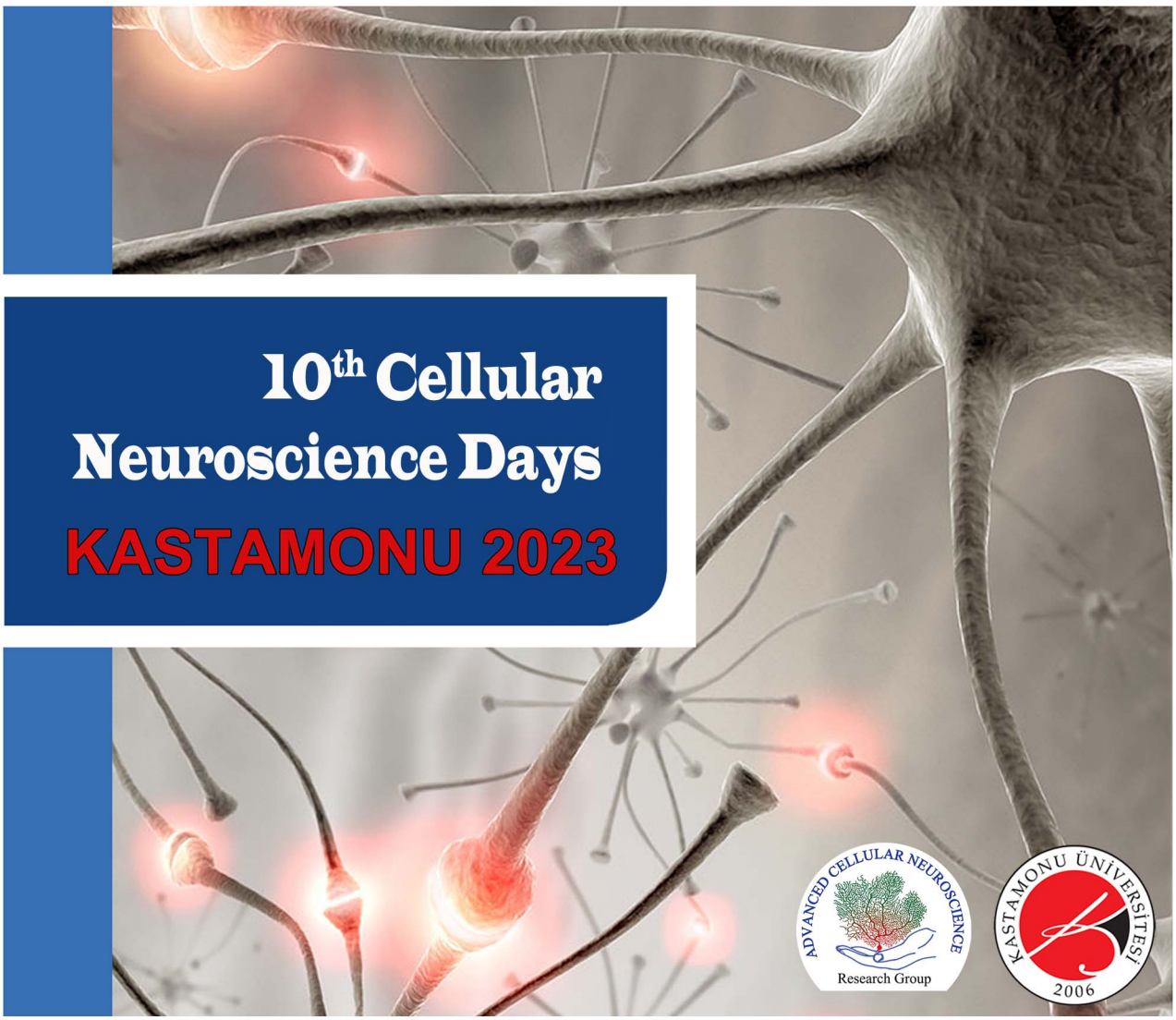

10th Cellular Neuroscience Days

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate cellular function, and the control or alteration of these processes by the action of receptors, neurotransmitters, second messengers, cation, anions, drugs or disease.

Areas of particular interest are four topics. They are;

A- Ion Channels (Na^+- K^+ Channels, Cl^- channels, Ca^{2+} channels, ADP-Ribose and metabolism of NAD^+, Patch-Clamp applications)

B- Oxidative Stress (Antioxidant vitamins, antioxidant enzymes, metabolism of nitric oxide, oxidative stress, biophysics, biochemistry and physiology of free oxygen radicals)

C- Interaction Between Oxidative Stress and Ion Channels in Neuroscience
(Effects of the oxidative stress on the activation of the voltage sensitive cation channels, effect of ADP-Ribose and NAD^+ on activation of the cation channels which are sensitive to voltage, effect of the oxidative stress on activation of the TRP channels in neurodegenerative diseases such Parkinson's and Alzheimer's diseases)

D- Gene and Oxidative Stress
(Gene abnormalities. Interaction between gene and free radicals. Gene anomalies and iron. Role of radiation and cancer on gene polymorphism)

READERSHIP

Biophysics	Biochemistry
Biology	Biomedical Engineering
Pharmacology	PhysiologyGenetics
Cardiology	Neurology
Oncology	Psychiatry
Neuroscience	Neuropharmacology

Keywords
Ion channels, cell biochemistry, biophysics, calcium signaling, cellular function, cellular physiology, metabolism, apoptosis, lipid peroxidation, nitric oxide, ageing, antioxidants, neuropathy, traumatic brain injury, pain, spinal cord injury, Alzheimer's Disease, Parkinson's Disease.

Abstract Book

of

10th Cellular

Neuroscience Days

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with collaboration of

Kastamonu University

10th Cellular Neuroscience Days

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Bayram Ufuk ŞAKUL
Department of Anatomy, School of Medicine
Medipol University, Istanbul, Türkiye

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Mechanical responses of neurons to axon injury

Gürkan ÖZTÜRK
Department of Physiology, School of Medicine
Medipol University, Istanbul, Türkiye

No abstract.

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Speak No. 3

Nanotechnology in neurology

Güven AKÇAY
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Current treatment options for central nervous system diseases only relieve symptoms. However, these treatments cannot significantly reduce or stop the progression of the underlying pathology of the disease. Therefore, there is an urgent need to develop more effective treatments. However, it is predicted that this need can be achieved with an in-depth understanding of the mechanisms and agents that play a role in the development of each disease. In these cases, the method of treatment with therapeutic compounds in diseases of the nervous system comes to the fore. However, the blood-brain barrier plays an important role in this treatment method. Since ischemic stroke is recognized as one of the most serious public health problems, there is an urgent need to design and develop smart drug carriers with physicochemical and biological properties that will provide enhanced drug delivery with specific targeting capability to the ischemic region. Recently, targeted nanoparticles (NPs) have been developed that can easily pass the BBB, thanks to nanobiotechnological developments. Among them, Selenium (Se) nanoparticles show promise in the treatment of CNS diseases. Se nanoparticles show high antioxidant activity and, accordingly, selenoproteins strengthen the endogenous antioxidant system. Iron oxide nanoparticles (Fe_3O_4 NP) are widely used in biomedical fields due to their excellent magnetic properties, biocompatibility and biodegradability. The surface of Fe_3O_4 NPs can be easily functionalized with small molecules, polymers and other inorganic materials. Given this flexibility, Fe_3O_4 has the potential to be used in many biomedicine applications, including MR imaging, photothermal therapy, chemotherapy and magnetothermal therapy. As a result, the fact that the cargo molecule carrying the active substance in nanoparticle-based drug delivery systems has antioxidant activity, is biocompatible and has a dual function to provide both treatment and imaging are promising studies that can increase the quality of diagnosis and treatment in neurological diseases.

SPEAKERS

Speak No. 4

Using the laser confocal microscope in the neuronal cells for the investigation of the TRP channels

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The calcium ion (Ca^{2+}) plays a key role in a number of physiological processes, including the release of neurotransmitters and the contraction of muscles. Several calcium channels, including voltage- and chemical-gated calcium channels, are activated to allow Ca^{2+} to travel through the cell membrane. The TRP superfamily was also found within the last few decades. The voltage gated and chemically gated calcium channels' activation and inhibition processes are very dissimilar from those of TRP channels. For instance, capsaicin activates TRPV1, whereas ADP-ribose activates TRPM2. Oxidative stress triggers the activation of both channels.

The cytosolic free Ca^{2+} concentration is analyzed in the laser scan confocal microscope (LSCM) by using the fluorescent dyes such as Fluo-3-AM and Fluo-8-AM. The excessive Ca^{2+} influx via the TRP activations induces indices increase of mitochondrial membrane depolarization, and the increase of mitochondrial membrane depolarization is analyzed by using the JC-1 and DHR123 stains (Yazğan and Nazıroğlu 2021). In turn, the increase of mitochondrial membrane depolarization causes the increase of cytosolic reactive oxygen species (cROS) and mitochondrial ROS (mROS). DCFH-DA and MitoSOX were used in the LSCM for the analyses of cROS and mROS, respectively (Yıldızhan and Nazıroğlu 2023). In the oral presentation, I will review the stains of LSCM for the investigation of TRP channels

Keywords: Fluo-3-AM; Laser scan confocal microscope; Oxidative stress; TRP channel.

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SPEAKERS

Speak No. 5

Vaccine development using expression systems against COVID-19

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No abstract.

Keywords: Microscopy, Imaging, Histology Education

SPEAKERS

Speak No. 6

Paramicroscopy

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Microscopes are main devices of histology. Microscopic images (photo-micrographies) are most important materials of histology education. In order to enable students to better understand of subjects, lecturers of histology often try to find best images which more efficient education. Sometimes, some similes/analogies from daily life are used to keep them in mind. For example, one of the frequently given examples is walnut and brain similarity.

In this presentation, some examples from our personal experiences will be listed. We set out from the similarities in the images of some plants, fruits, vegetables and their sections and microscopic photographs. For example, we liken the placement of corn kernels on the cob to the architecture of small intestinal epithelial cells. Another interesting similarity example is the cross sectional wives' of the renal corpuscule with the tomato.

For that reason we named this approach "paramicroscopy" for the first time in the literature. In Greek etymology: "para" means that; beside (paracapsular), parallel (paraxial), between (inter, paracellular), around (peri), surround (paracortex), adjacent (juxta, paracentral), near (parepididimis), over (paracondylar), across (trans, paraesophageal), beyond (trans, hyper, ultra, paranormal), through (paracostal), throughout (paracerebral), avoiding (parenteral), related (paraclinical, paramedical). We use "paramicroscopy" term to mean; associate, involve, relevant, almost, indirect, near (similarity) and beyond with microscopy.

SPEAKERS

Speak No. 7

Quantitative evaluation of cell organelles or synapse structures at the electron microscopy level

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It is important that the methods to be used in obtaining the numerical data required for the interpretations of the geometrical properties of biological structures are up-to-date and reliable, and that the biological interpretations reflect the truth. The series of methods that cover the evaluation of their three-dimensional properties from two-dimensional images of biological structures are defined as stereological techniques. Evaluation of the structure morphology at the electron microscopy level is necessary to understand the structure-function relationship. Physical disector counting technique, one of the stereological methods, is widely used in neuroscience researches. This technique is widely preferred in obtaining numerical data about the number of synapses, the number of mitochondria and similar structures at the electron microscopy level. Disector counting technique is a method that provides an unbiased estimation of the numerical density of the particles in the sections by comparing two consecutive sections with each other (Sterio 1984). One of the consecutive sections is called the *reference* and the other is the *look up* section. A volume is created with two consecutive sections, and the ends of the particles in one direction are counted in the volume created. A counting frame (unbiased counting frame) with special features is used in the application of this technique. With this frame, each particle is counted only once. Particle counts with the disector technique are accepted as the gold standard method by researchers because of their reliability and reproducibility.

Keywords: Stereology; Disector; Electron microscopy; Quantitation; Synapse number

References

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SPEAKERS

Speak No. 8

Neuroimmune mechanisms mediating neuronal survival and neural repair after ischemic brain injury

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Acute ischemic stroke; is defined by sudden cessation of focal cerebral blood flow due to thrombosis or embolism. Neurons that demand high glucose and oxygen are vulnerable to ischemia. Rapid neuronal death begins due to mitochondrial dysfunction. Disruption of the blood-brain barrier causes molecules and cells in the blood, including immune cells, to leak into the ischemic brain area. Some molecules increase microvascular permeability, causing swelling (edema) of brain tissue. Acute cerebral inflammation often worsens the functional prognosis of patients. Complex interactions between immunity and brain cells initially trigger stroke pathologies.

Inflammation and swelling of brain tissue resolve about one week after the stroke. During this healing phase, resident or infiltrating immune cells switch from a pro-inflammatory role to a reparative role. These cells produce neurotrophic factors and synapse-modulating molecules around the injured brain tissue for stroke recovery. This helps to restore brain tissue in the peri-infarction area, accelerate functional recovery, and reorganize neural tissues. This reflects the close relationship between reparative immune cells and neurons near and far from the infarct area. Although recovery persists for months or even years after stroke onset, these brain-endogenous repair mechanisms usually continue to disappear slowly. The lack of therapeutic drugs to accelerate neural repair later perpetuates neurological disorders. Although post-stroke recovery continues for months or even several years after stroke onset, these brain-endogenous repair mechanisms usually continue to disappear slowly, and the remaining neurological deficits then become permanent after-effects.

Functional recovery after brain injury; requires reorganization of the neural circuit, remodeling of nervous tissue, restoration of blood circulation, remyelination, axonal growth, synaptic modulation, and neurogenesis. Therefore, there is a need for new-generation therapeutics that will accelerate the resolution of acute harmful inflammation, promote beneficial neuro-immune interaction, and provide better functional recovery with restorative dynamics.

Keywords: Ischemic brain injury; neuroinflammation; neuro-immune interaction; repair

SPEAKERS

Speak No. 9

Mineral balance in neurodegenerative diseases

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[2]Istanbul Medipol University, Health Science and Technologies Research Institute - Regenerative and Restorative Medicine Research Center

Neurodegenerative diseases, as a group, are relatively common and cause significant medical and social problems. Neurodegenerative disorders include a group of pathologies characterized by progressive and irreversible loss of neurons in specific regions of the brain. The leading ones of these pathologies with many types are; Parkinson's disease (PH), Huntington's disease (HH), Alzheimer's disease (AD) and Amyotrophic lateral sclerosis (ALS) are mainly seen in neurologically normal individuals later in life.

In Parkinson's and Huntington's diseases, abnormalities in the control of movements occur with neuronal loss in the basal ganglia. Huntington's disease is a genetically inherited, autosomal dominant, less common pathology. In Alzheimer's disease, memory loss, thinking and communication disorders, behavioral changes, decreased speech and judgment abilities, orientation and coordination disorders develop due to the loss of hippocampal and cortical neurons. In ALS, muscle weakness is observed due to degeneration of spinal, bulbar and cortical motor neurons.

Today, the treatment of these pathologies is limited to symptomatic treatment, there is no treatment approach for the underlying disease. In addition to the cost of pharmacological treatment, the care of patients is a problem for both families and economic costs. Although the new treatment methods applied remain symptomatic and reduce the incidence of the disease relatively, a treatment method with clinical efficacy has not been developed yet. Since NDHs show multifactorial pathology, single-targeted treatment approaches that can give positive results are also insufficient. In order to treat cognitive dysfunctions, approaches such as lifestyle changes and nutrition education that consider the balance of macro and microelements are emphasized, as well as medications. For this reason, planning clinical studies to increase therapeutic power in order to use multifaceted treatment approaches together may open new horizons in this field.

Keywords: Neurodegeneration, Alzheimer's Disease, Huntington's Disease, Parkinson's Disease, ALS, mineral, nutraceuticals

SPEAKERS

Speak No. 10

Novel diagnostic, prognostic and treatment-response biomarkers in multiple sclerosis

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Multiple sclerosis (MS) is an autoimmune, inflammatory, and neurodegenerative central nervous system (CNS) disease that primarily impacts young people. MS is caused by an initial inflammatory damage to the myelin sheaths and subsequent degeneration of the neurons in CNS. The social and economic burden of this disease is high because it leads to severe disability in the advanced stages. Unfortunately, the etiopathogenesis of MS is not fully understood to date; therefore, early diagnosis of MS and identifying susceptible individuals before they develop MS is challenging subjects. This also limits the ability of scientists to develop curative therapies for MS. Currently, MS therapy includes corticosteroids administered during an attack and disease-modifying therapies which are used to prevent new attacks. However, the effectiveness of these agents varies from patient to patient. It is difficult to determine the effectiveness of a particular treatment due to a lack of objective parameters that define the efficacy. An effective early-onset therapy is important for prolonging the time until the next MS attack, reducing the number of new lesions seen on MRI scans and slowing the progression of disability and cognitive deficits. For this reason, new biomarkers need to be identified for both early diagnosis of MS, but also for following the disease activity and therapeutic response in MS patients using MS therapies. In this presentation, information will be given about diagnostic, prognostic, and therapy-response biomarkers in MS (Comabella & Montalban, 2014). Moreover, research studies conducted on developing new MS biomarkers in the speaker's research group will also be mentioned (Can Demirdöğen et al., 2022; Can Demirdöğen et al., 2023).

Keywords: Diagnosis, Neurodegenerative disease, Susceptibility

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Oral Presentations

Oral Presentation 1

Systemic immune inflammatory index value in the attack period in patients with multiple sclerosis

İdris KOCATURK
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Multiple sclerosis (MS) is an inflammatory, demyelinating disease of the central nervous system. It is known that the determination of biomarkers that help evaluate the disease process and treatment effectiveness provides a more effective follow-up to the patients. Especially the neutrophil-lymphocyte ratio (NLR) is a well-known marker associated with MS pathogenesis and prognosis. Systemic immune-inflammation index (SII) is a new inflammatory marker calculated with the formula of platelet count x neutrophil count/lymphocyte count. SII has been found to be associated with many inflammatory diseases and it has been determined that it can predict the prognosis with these diseases. However, as far as we know, there are only a few studies investigating the relationship between SII and MS (Gokce SF et al. 2023, Saçmacı H et al. 2021). In this study, we investigated the relationship between SII in the attack period in MS patients.

In this retrospective study, 40 patients who were hospitalized in the Neurology clinic of a tertiary hospital with the diagnosis of MS attack and 42 healthy volunteers who were similar in age and gender as the control group were included. Sociodemographic data and laboratory findings of patients were obtained from the hospital information management system. SII values were compared between the patient and healthy control groups with the Mann-Whitney U test. SII values were found to be significantly higher in MS patients during the attack period. (p=0.022)

In conclusion, SII is a promising biomarker that can be used in the monitoring of MS patients. Larger and prospective studies are needed on this subject.

Keywords: Multiple sclerosis, inflammation, Systemic Immune Inflammatory Index (SII)

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Oral Presentations

Oral Presentation 2

Evaluation of the relationship of multiple sclerosis with neutrophil lymphocyte ratio (NLR), an inflammatory marker

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Multiple sclerosis (MS) is an inflammatory and degenerative disease of the central nervous system and is characterized by demyelination and axonal damage (Hemmer et al. 2015 and Kaskow et al. 2018). The aim of this study was to evaluate the relationship between the neutrophil lymphocyte ratio (NLR), an inflammatory marker, and patients with MS.

42 MS patients and 47 healthy controls who applied to Kastamonu Teaching and Research Hospital were enrolled in this study between January 1, 2023, and March 31, 2023. There was no significant difference in terms of age and gender in the healthy group ($p > 0.05$). Complete blood counts were evaluated retrospectively and the NLRs were calculated for all participants in the study. The results were nonparametrically distributed and compared with the Mann-Whitney U test. The NLR was significantly higher ($p < 0.01$) in MS patients compared to the control group.

In conclusion, the NLR may be a useful marker for MS diagnosis and prognosis. It has been shown in many studies that the NLR can predict prognosis in neurodegenerative diseases (Sayed et al. 2020 and Wei et al. 2022). Hence, NLR can be a simple, rapid and inexpensive biomarker to predict the prognosis of MS patients. Therefore, further clinical studies are needed on its association with MS disease activity or disability.

Keywords: Multiple sclerosis; Neutrophil lymphocyte ratio (NLR); Inflammatory marker.

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Oral Presentations

Oral Presentation 3

Neuroprotective effects of remicade on hydrogen peroxide-induced oxidative stress in SH-SY5Y cells

Ayşegül ÖZTÜRK[1], Ahmet Şevki TAŞKIRAN[2], Fatih YULAK[2]

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Remicade (Infliximab) is an antagonist for TNF-α, a proinflammatory cytokine. Recent studies have shown that they can be used as important therapeutic agents to inhibit oxidative stress and apoptosis mechanisms that may occur in neurodegenerative diseases. However, its effect on hydrogen peroxide (H_2O_2)-induced oxidative stress in neuroblastoma cells is still unclear. This study was designed to examine the effect of remicade on neuronal damage after hydrogen peroxide-induced oxidative damage in SH-SY5Y cell line.

SH-SY5Y neuronal cell line was used in the study. The cells were divided into four groups to evaluate the effect of remicade on H_2O_2-induced oxidative damage. Control group; no treatment, H2O2 group; incubated with 250 µM concentration for 24 hours, remicade group; incubated with different concentrations (200, 100, 50, 25 and 12.5 nM) of remicade for 24 hours, remicade+ H2O2 group; pretreated with various concentrations (200, 100, 50, 25 and 12.5 nM) of remicade for 1 hour and then cells were exposed to 250 µM H2O2 for 24 hours. XTT assay was used to evaluate cell viability. Total antioxidant (TAS) and total oxidant (TOS) levels were calculated using commercial kits. Reactive oxygen species (ROS) levels were evaluated by ELISA kit. Apoptosis was determined by flow cytometry method. Remicade pretreatment at 50 nm concentration significantly increased cell viability in SH-SY5Y cells after H_2O_2 toxicity ($p < 0.001$). Furthermore, remicade significantly decreased TOS levels ($p < 0.001$), increased TAS levels ($p < 0.001$), and decreased ROS levels ($p < 0.001$). In addition, remicade decreased the number of apoptotic cells, flow cytometry revealed that it increased the percentage of viable cells ($p < 0.001$).

Remicade reduces neuronal cell death due to hydrogen peroxide-induced oxidative damage in SH-SY5Y cells by activating the antioxidant system.

Keywords: Remicade, Oxidative Stress, Apoptosis, SH-SYY cell line

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Oral Presentations

Oral Presentation 4

Glutamate of wheat germ oil in C6 glioma cell line effect on excitotoxicity

Rumeysa KILIÇER[1], Ahmet Şevki TAŞKIRAN[1]
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The excitatory in the central nervous system Glutamate , which acts as a neurotransmitter , interacts with the receptors on the cell membrane and plays a role as a regulator of many neurological events such as learning, memory, movement, synaptic and sensory connections (Yıldırım, 2009). The excessive increase of glutamate leads to the deterioration of calcium homeostasis . Glutamate concentration is impaired by factors such as genetic predisposition, increase in oxidant substances, and an external chemical stimulus . The increase in the release of glutamate increases neuronal damage and causes a toxic process called excitotoxicity . In the pathogenesis of neurological diseases such as Alzheimer's, Parkinson's and epilepsy glutamate excitotoxicity plays an important role. wheat germ _ germ) is a completely natural extract obtained from the living embryo of wheat . Wheat germ is a concentrated source of many essential nutrients, including vitamin E, folic acid, phosphorus, thiamine , zinc and magnesium, as well as essential fatty acids and fatty alcohols . For these reasons, my wheat germ It has antioxidant properties in fatty tissues (Leenhardt et al., 2008). Wheat germ has been shown to provide anti- inflammatory and lipid-lowering effects, such as other lipid factors, such as polycosanols , carotenoids , phytosterols , and essential fatty acids. Besides its role as a fat-soluble antioxidant, it has been shown to affect gene expression in many tissues. In the studies carried out , wheat germ oil glutamate Its efficacy against excitotoxicity is still unclear. Our aim in this study is to produce wheat germ oil, on glutamate-induced excitotoxicity in glioma cells .

C6 glioma cell line was used in this study. Wheat germ of the oil To evaluate its effect on glial cell death after glutamate- induced excitotoxicity , cells were divided into four groups. The control group was not treated, the glutamate group was 10 mM . Glutamate was applied, Wheat germ oil group was treated with different concentrations (2000, 1000, 500 and 250µM) Wheat germ oil , Wheat germ oil + glutamate group was treated with different concentrations (2000, 1000, 500 and 250µM) Wheat germ oil 1 hour after the cells glutamate was administered. Cell viability in C6 cells of the groups was examined using the XTT test. The cell viability of the obtained values was compared with the control group. Total antioxidant status (TAS) and total oxidant status (TOS) in cells were measured with commercial kits. 500 µM in concentration Wheat germ oil significantly increased cell viability in C6 cells after glutamate-induced cytotoxicity ($p < 0.05$). Wheat germ oil (500 µM) + glutamate significantly increased TOS levels in C6 cells, but did not change TAS ($p > 0.05$), compared to untreated control cells ($p < 0.05$). H/PI staining was performed to determine apoptosis /necrosis. Wheat germ oil (500 µM) + glutamate significantly reduced apoptotic cells compared to glutamate group cells ($p < 0.05$) . In conclusion; Wheat germ oil reduces glial cell death after glutamate- induced cytotoxicity in C6 cells . While wheat germ oil has a protective effect in the acute process, its long-term use can increase oxidative damage and cause cell death.

Keywords: Glutamate, Excitotoxicity, C6 cell line, Wheat germ oil

Oral Presentations

Oral Presentation 5

Effect of bromelain against glutamate-induced excitotoxicity in C6 cell line

Meryem YAPRAK[1], Ahmet Şevki TAŞKIRAN[1]
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Glutamate is one of the main excitatory neurotransmitters of the central nervous system (CNS) and contributes to normal nerve conduction, development, differentiation and plasticity. In pathological conditions, glutamate release is excessive, and excessive activation of glutamate receptors results in increased intracellular Ca2+ flux. Increased intracellular Ca2+ concentration disrupts calcium homeostasis and initiates a series of signaling pathways that lead to mitochondrial dysfunction, oxidative stress resulting from increased ROS production, ER stress, and release of lysosomal enzymes. Excitotoxicity, which is induced by excessive glutamate activation and causes excessive calcium ion entry into the cell, is the main mechanism in the development of various neurodegenerative disorders such as Alzheimer's, Parkinson's, and Epilepsy. Calcium entry into the cell causes mitochondrial dysfunction, causing the cell to enter the apoptosis process. Therefore, reducing oxidative stress in glial cells is important for the treatment of neurodegenerative diseases. Bromelain is a sulfur-containing protease enzyme found in pineapple (Ananas comosus), a tropical plant widely cultivated around the world. Bromelain has antioxidant properties due to its potential to scavenge free radicals and increase the level of enzymatic and non-enzymatic antioxidants. In this study, it was aimed to investigate the effect of bromelain, which has antioxidant properties, against glutamate excitoxicity.

C6 glioma cell line was used in this study. To evaluate the effect of bromelain on glial cell death after glutamate-induced excitotoxicity, cells were divided into four groups. The control group did not receive treatment, the glutamate group received 10 mM glutamate, the bromelain group received different concentrations (2000, 1000, 500 and 250μM) of bromelain, the bromelain + glutamate group treated cells with different concentrations ((2000, 1000, 500 and 250μM) bromelain. Glutamate was applied after one hour Cell viability was examined in the C6 cells of the groups using the XTT test.The cell viability of the obtained values was compared with the control group. Total antioxidant status (TAS) and total oxidant status (TOS) in cells were measured with commercial kits. Bromelain at 250 μM concentration significantly increased cell viability in C6 cells after glutamate-induced cytotoxicity ($p < 0.05$). Bromelain (250 μM) + glutamate significantly decreased TOS levels in C6 cells, increased TAS ($p < 0.05$), compared to glutamate group cells ($p < 0.05$). H/PI staining was performed to determine apoptosis/necrosis. Bromelain (250 μM) + glutamate significantly reduced apoptotic cells compared to glutamate group cells ($p < 0.05$).

In conclusion; Bromelain reduces glial cell death after glutamate-induced cytotoxicity in C6 cells. While bromelain has a protective effect in the acute process, its long-term use can increase oxidative damage and cause cell death.

Keywords: Glutamate, Excitotoxicity, C6 cell line, Bromelain

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Oral Presentations

Oral Presentation 6

Effect of mirtazapine on hydrogen peroxide toxicity in glioblastoma cells

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Recent studies have shown that mirtazapine has positive effects on nervous system. However, its effect on hydrogen peroxide-induced oxidative damage in glial cells is still unclear. This study was prepared for the control of mirtazapine on glial damage after peroxide-induced oxidative damage in C6 glial cells.

In this example, the C6 rat glioma cell line was used. Four cell groups were prepared to evaluate the effect of mirtazapine on glial cell death after hydrogen peroxide-induced oxidative damage. The control group was without any treatment. Cells in the H2O2 group were treated with 0.5 mM H2O2 for 24 hours. The structures of mirtazapine + H2O2 components are pretreated with mirtazapine of different protections (12.5, 25 50 and 100 µM/mL) for 1 hour and then exposed to 0.5 mM H2O2 for 24 hours. After live induction of oxidative stress, cellularity was evaluated by XTT analysis. Apoptosis and necrosis images were obtained by staining with Heust and PI dyes. Total antioxidant status (TAS) and total oxidant status (TOS) levels in cells were measured with commercial kits.

Mirtazapine at the concentrations of 50 and 100 µM/mL significantly increased the cell viability in C6 cells after hydrogen peroxide-induced oxidative damage ($p < 0.001$). In addition, the instrument of mirtazapine increased the decreased TAS level ($p < 0.01$) while overshadowed by increased TOS after peroxide-induced oxidative damage ($p < 0.001$).

By activating the antioxidant system, mirtazapine reduces glial cell protection due to peroxide-induced oxidative damage in C6 cells.

Keywords: Mirtazapine, Oxidative Stress, Cell Death, C6 Glioma cells

Oral Presentations

Oral Presentation 7

The role of RDW-SD, a hemogram parameter, in predicting survival of community acquired pneumonia patients

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Community acquired pneumonia is one of the major mortality reason in developing countries. RDW-SD (red cell distribution with standart deviation) is a hemogram parameter which indicates heterojenity of RBC (red blood cell) volume (Wang C et al. 2020) and has prognostic and diagnostic value in infectious disease (Hu ZD et al. 2020 and Wu J et al. 2019). The aim of this study was to investigate the role of RDW-SD predicting survival of community acquired pneumonia patients in Kastamonu Training and Research Hospital.

One hundred sixty-five community acquired pneumonia patients were included in this study. One hundred thirteen patients were hospitalized and survived. Patients were Hemogram samples of patients were analyzed with Sysmex-XN 1000 series (Kobe, Japan). Hemogram data were obtained from LIS records. RDW-SD values of survived and dead patients were compared with Mann-Whitney U test. ROC (receiver-operating characteristic) curve was used for predicting survival and area under curve (AUC) was calculated . The results were analyzed using Statistical Package for Social Sciences 18.0 (SPSS Inc., Chicago, IL, USA).

RDW-SD values were statistically different between dead and survived patients ($p<0.001$). Median values of dead and survived patients were 49.05 and 43.90, respectively. AUC value of RDW-SD was 0.735. 95% confidence interval (CI) of AUC was [0.654–0.819]. When cut-off value of RDW-SD was 45.2, sensitivity and specificity values were %75.9 and %61.9, respectively.

In conclusion, RDW-SD was found to be one of the predictor of survival in community acquired pneumonia.

Keywords: Red cell distribution width (RDW), pneumonia, receiver operating characteristic (ROC)

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Oral Presentations

Oral Presentation 8

Comparison of the effects of 6 weeks of high-intensity resistance training with low-intensity resistance training and blood flow restriction on bone markers in young women

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BFR (Blood Flow Restriction) is a training method that partially restricts arterial flow and completely restricts venous outflow in the working muscular system during exercise (Scott et al. 2015).

The aim of this study was to examine and compare the effects of different resistance training protocols HIRT (High-Intensity Resistance Training) with LIRT (Low-Intensity Resistance Training) and BFR on bone marker concentrations in young women.

Twenty-three physically active females aged 19 to 26 years old was randomly assigned to one of two groups: LIRT-BFR (n = 12) or HIRT (n = 11). Both groups underwent an experimental design consisting of six weeks and performed a 3-day-a-week routine of strength training at varying intensities, followed by measurements taken pre-training. The comparison of longitudinal training responses between LIRT-BFR and HIRT was measured on bone turnover markers human bone-specific alkaline phosphatase (B-ALP) and carboxy-terminal cross-linked telopeptide of type I collagen (β-CTX) serum concentration levels.

Time effect was detected in both the LIRT-BFR group and the HIRT group significantly improved their 1 RM leg press performance, mean heart rate (meanHR), rate of perceived exertion (RPE), β-CTX, and B-ALP β-CTX ratio after the follow-up six-week training program ($p<0.005$). No significant changes were observed in time effect on exercise maxHR and B-ALP ($p>0.05$). Furthermore, time effect observations on bone biomarker β-CTX were significantly changed -40.17% and -43.23% in the LIRT-BFR group and HIRT group respectively ($p<0.05$).

As a result, after 6 weeks of training, HIRT and LIRT-BFR gave similar results on bone formation-resorption markers.

Keywords: Low Intensity Resistance Training, Blood Flow Restriction, High Intensity Resistance Training, human bone-specific alkaline phosphatase, carboxy-terminal cross-linked telopeptide of type I collagen

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Oral Presentations

Oral Presentation 9

Therapeutic and neuroinflammatory effects of transcranial direct current stimulation on the GABAergic pathway in an experimental cerebral ischemia model

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Cerebral ischemia occurs as a result of obstruction of some or all of the arteries feeding the brain, resulting in significant impairments in both motor and cognitive functions. In our study, we aimed to investigate the effects of Transcranial Direct Current Stimulation (tDCS) treatment on GABAergic pathway and on inflammatory pathways mechanisms of rats with focal cerebral ischemia.

30 male Wistar rats, each of which weighs 290-310 g, were divided into three groups as Sham, Ischemia/Reperfusion (IR) and IR+tDCS groups, with 10 rats in each group. The IR model was created by MCA's 90-minute occlusion. tDCS treatment was applied 0.5 mA 30 minutes a day for 2 days after IR. GABA, $GABA_A$, TNFα, IL-1β, Bax and Bcl-2 levels in hippocampus tissues were evaluated. Statistical analyzes were performed by One-Way ANOVA test.

Increase was observed in IR an group compared to Sham group while significant decrease was observed in IR+tDCS group compared to IR in the neuroinflammatory data ($p<0.05$). While GABA and $GABA_A$ levels were significantly lower in IR group compared to Sham group ($p<0.05$), it was found to be significantly higher in IR+ tDCS group compared to IR group ($p<0.05$).

The results of our study showed that tDCS had a neuromodulative effect on GABA, $GABA_A$ receptor and TNFα, IL-1β, Bax and Bcl-2 neuroinflammatory levels, which play a vital role in cell death after ischemia reperfusion.

Keywords: GABA, ischemia reperfusion, neuroinflammation, tDCS

Oral Presentations

Oral Presentation 10

Roles of TRPC channels in epilepsy

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Epilepsy is one of the most common neurological disorders. According to the WHO, approximately 50 million people have epilepsy. Epilepsy is characterized by sudden, repetitive epileptic seizures to the electrical discharge of cortical neurons, including the symptoms of confusion, stiff muscles, and jerking movements (Stafstrom & Carmant, 2015). Several factors contribute to the etiology of epilepsy. One of the main reasons is increased excitation and decreased inhibition in the brain. Increased excitation is believed to be seen by either increased activity of the glutamatergic system or increased Ca^{2+} influx. Several medications are available for the treatment; however, current medications have limited effects on some types of epilepsy (Devinsky et al., 2018).

TRPC channels are a subfamily of the TRP superfamily, including seven receptors. They are cation channels permeable to Ca^{2+} ions, which several stimulants can activate (Wang et al., 2020). TRPC channels are believed to have a role in the slow depolarization of the membrane due to the Ca^{2+} influx, which contributes to epileptiform bursting (Yu et al., 2022). Studies have shown that activation of mGluRs elicit epileptiform bursting, which is also dependent on TRPC1/4 channels. Additionally, it has been shown that TRPC5 channels are highly expressed in CA1 pyramidal neurons and have a role in the epileptiform firing. Finally, TRPC3/6/7 channels are also highly expressed in the brain and are believed to contribute to epileptiform bursting due to the activation via an unknown mechanism (Zheng, 2017).

In conclusion, TRPC channels are believed to have a crucial role in epilepsy. Therefore, targeting these channels could be a new treatment strategy for treating epilepsy.

Keywords: Calcium signaling; Cortical neurons; Epilepsy; TRPC channels.

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Oral Presentations

Oral Presentation 11

An evaluation with blood parameters for early diagnosis of multiple sclerosis: A new ratio suggestion

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Multiple sclerosis (MS) is a non-traumatic neurological disease that can cause disability (Lublin et al. 2022). Since there is no curative treatment it is important to diagnose early and slow the course of the disease with current treatments. We aimed to evaluate the blood parameters and their ratios to each other, which may provide an advantage in early diagnosis.

Ethical approval was obtained from the Non-Interventional Clinical Ethics Committee (2022/07-05). Age and sex matched healthy controls (95) and MS patients (95) were included in the study. The MS group was evaluated as new diagnosis, relapse, and remission period. Mann Whitney U and Wilcoxon test were used.

The neutrophil-lymphocyte ratio increased in MS patients during the attack period ($p<0,05$). The erythrocyte-lymphocyte ratio was significantly higher than the control group in all 3 MS periods ($p<0,05$).

Neutrophil-lymphocyte ratio is accepted as a neuroinflammation marker (Bhikram and Sandor 2022). The high levels of neutrophil-lymphocyte ratio that we detected during the attack period in MS patients can be considered as an indicator of systemic inflammation. The high erythrocyte-lymphocyte ratio in all MS groups is thought to be due to dysfunction of erythrocytes and decreased number of lymphocytes. The erythrocyte-lymphocyte ratio may be a potential biomarker in the early diagnosis of MS.

Keywords: Multiple sclerosis, blood parameters, neutrophil-lymphocyte ratio, erythrocyte-lymphocyte ratio

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Oral Presentations

Oral Presentation 12

Retrospective analysis of clinical features and neurological findings in children diagnosed with COVID-19

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The first asymptomatic pediatric case in the COVID-19 pandemic was reported in January 2020. It is known that COVID-19 is generally mild in children, however, serious cases have also been reported. Significant differences in laboratory findings, clinical courses and treatments in children can be seen. Therefore, it is crucial to clarify the role of children in the transmission of variants in children.

The current study determined the rate of children under the age of 14 diagnosed with COVID-19, and the rate of hospitalized patients within the same age range in Medipol Mega University Hospital from March 2020 to January 2022. Additionally, their clinical symptoms, laboratory findings, and treatments were evaluated, and the prevalence of COVID-19 disease in children was compared with the face-to-face education period and before.

As of March 2020, a total of 20544 patients have been diagnosed with COVID-19 in our hospital. 1294 were under the age of 14, and 80 of them were hospitalized. Various abnormal laboratory findings were detected in these patients. Commonly used medications were paracetamol and antibiotics. The most common symptoms were high fever and cough; essential and common neurological symptoms are fatigue, myalgia, headache, taste and smell disturbances. The risk of ICU admission was detected to be higher in newborns, and hospitalization was common under the age of 1 or children aged 7 years and older. Additionally, since the beginning of face-to-face education, we determined that the number of pediatric cases has increased, and vaccination against COVID-19 was absent in the hospitalized patients.

Keywords: Children, COVID-19, COVID-19 treatment, COVID-19 vaccine, SARS-CoV-2

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Oral Presentations

Oral Presentation 13

The effect of gallic acid against glutamate-induced cytotoxicity in C6 glioma cells

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Gallic acid (GA) is present as a phenolic component of various foods and plants. GA is a molecule with broad biological properties such as antioxidant, anti-microbial, and anti-inflammatory activities (Kahkeshani et. al. 2019). As the main excitatory neurotransmitter in the mammalian central nervous system excessive extracellular glutamate (GLU) can activate the GLU receptors and neuronal/intracellular calcium (Ca^{2+}) overload, producing neurotoxicity, a common pathway for neuronal injury or death and is associated with neurodegenerative diseases (Meldrum et. al. 2000; Hacimuftuoglu et. al. 2016). The present study aimed to investigate the effect of gallic acid on GLU -induced cytotoxicity in C6 glioma cells. For the study, groups were formed from C6 cells as control, GA (100 μM) (Chandrasekhar et. al. 2018), GLU (10 mM, 24 h) (Doğan et. al. 2021), and GLU +GA. In the study, Total oxidant (TOS), total antioxidant (TAS), MDA, and caspase-3 levels in the cells were determined by ELISA kit. The results showed that GLU administration increased TOS, MDA and caspase-3 levels by causing cytotoxicity in C6 cells ($p<0.05$). However, in C6 cells treated with GA before GLU incubation, TOS, MDA and caspase-3 levels were decreased, and TAS level increased compared to the GLU group ($p<0.05$). As a result, it was determined that GA treatment showed a protective effect in the GLU-induced cytotoxicity model in C6 cells.

Keywords: Gallic acid, Glutamate, Oxidative stress, caspase-3, C6 glioma cells

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Oral Presentations

Oral Presentation 14

The relationship between *Toxoplasma gondii* infections of the central nervous system and neurological diseases

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Toxoplasma gondii is an obligate intracellular parasite known to cause various lesions in humans. This review investigated the role of *T. gondii*, which infects approximately one-third of the human population in developing and developed countries (Flegr, 2015), in the etiology of neurological diseases.

T. gondii spreads throughout the host body by cells such as dendritic cells and monocytes. The Trojan horse strategy can enter immune-privileged organs, eyes, testicles, and especially the brain. It can be located in the cerebral cortex, amygdala, hippocampus and basal ganglia regions of the brain and has been reported in studies to infect many cell types, such as Purkinje cells, neurons and microglial cells (Flegr, 2015; Ayaz et al., 2016). The immune system, which is activated by the ingestion of *T. gondii*, can affect the cell's electrical activity by changes in the release of neurotransmitter substances and the intracellular Ca^{+2} balance. As a result of the deterioration of electrical activity, both discharges in the cells and changes in the cognitive and psychological state of the host may occur (Ayaz et al., 2016). In addition, chronic infection of *T. gondii* has been reported to increase brain dopamine levels by up to 15% in mice. Therefore, neurobehavioral and neurological symptoms associated with *T. gondii* infection may be related to potential dopamine modulation in the host brain. Studies in mice have collected evidence that chronic *T. gondii* infection is associated with deficits in spatial learning and memory, impaired motor performance, reduced anxiety, longer reaction times, sensory attention deficits, and altered novelty-seeking behaviour (Virüs et al., 2021). In addition to in vivo studies, the relationship of *T. gondii* infection with neurobehavioral and neurological symptoms in humans was also investigated. It has been reported that the rate of *T. gondii* in patients with various neuropsychiatric disorders such as schizophrenia, cryptogenic epilepsy, bipolar disorder, unipolar depression, obsessive-compulsive disorder, homicides, generalized anxiety and panic disorders, drug abuse disorder, suicides, personality disorders and mood disturbances, is higher than in healthy controls (Flegr, 2015; Ayaz et al., 2016; Ekici et al., 2021). Current research consistently supports the relationship between *T. gondii* and schizophrenia. (Virüs et al., 2021).

In conclusion, although there is evidence that *T. gondii* infection may cause neurological disorders, more detailed studies should be conducted on this subject.

Keywords: *Toxoplasma gondii*, schizophrenia, dopamine, neurological diseases

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Oral Presentations

Oral Presentation 15

Diabetic neuropathic pain mechanism: Role of oxidative stress and TRP channels

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Diabetes mellitus (DM) is a chronic disease characterized by hyperglycemia caused by the body's inability to produce or use insulin effectively. Diabetic neuropathy is one of the early complications seen in approximately 66% of patients with diabetes (Paul *et al.* 2020). Oxidative stress plays a vital role in developing both microvascular and cardiovascular complications of diabetes. Increasing evidence indicates that reactive oxygen species (ROS) and reactive nitrogen species (RNS) have a crucial role in the pathophysiology of diabetic neuropathy (Matough *et al.* 2012). One target of ROS/RNS signaling is Ca^{2+} channels that mediate both long-term oxidative stress and acute cellular responses. The transient receptor potential (TRP) protein superfamily is voltage-independent calcium-permeable cation channels expressed in mammalian cells. TRP channels have physiological roles in mechanisms that control many physiological responses such as temperature and mechanical sensations, response to painful stimuli, taste and pheromones (Naziroglu *et al.* 2020). The TRP channel family includes six TRPs expressed in pain-sensing neurons and primary afferent nociceptors. TRPC and TRPM, in particular, have members activated by oxidative stress. The dysregulation and overproduction of oxidative stress products in diabetic neuropathic pain have focused attention on TRP channels. With the detection of genetic variations in cation channels such as TRP channels, the effectiveness of Ca^{2+} channels has become an increasingly appropriate approach for therapeutic interventions against painful and degenerative pain (Pariente *et al.* 2018). In conclusion, a comprehensive review study on the role of TRP channels in the pain mechanism of diabetic neuropathy was presented by searching the available literature data.

Keywords: Diabetes, neuropathic pain, oxidative stress, TRP channels

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Oral Presentations

Oral Presentation 16

Comparison of the effects of thymoquinone and K252a on DRG neurons in axotomy damage generated by laser microdisection technique

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Peripheral nerve injury (PNI) caused by trauma and other factors can lead to partial or complete loss of sensory, motor and autonomic functions as well as neuropathic pain. Thymoquinone (TQ) is the bioactive molecule of black cumin seeds. K252a is an effective serine/threonine protein kinase inhibitor, inhibiting the tyrosine phosphorylation of Trk A induced by NGF. K252a has been reported as a potential therapeutic agent for psoriasis in preclinical studies. In this study; By exposing traumatic damaged DRG neurons to TQ and K252a *in vitro*, regeneration was aimed to occur in injured neurons, and it was also aimed to elucidate the regeneration mechanism. In the study, dorsal root ganglion (DRG) were obtained from adult mice, primary neuron culture was prepared from the ganglion. Cultures were incubated for 48 hours to ensure adequate axon development for axotomy. For the design of the groups; four groups were formed from the cultures: the Axotomy (0.3% DMSO) group, the TQ (2.5 µM) group, the K252a (200 nM) group, and the TQ (2.5µM) + K252a (200 nM) group. Two hours after treatment administration, axons were cut with a microlaser (axotomy) 50 micrometers from the soma. In the Live Cell Microscopic Imaging System, propidium iodide was used to detect deceased neurons from injured neurons, and calcein fluorescent dyes were used to detect survivors. Imaging was performed before axotomy, at 24 and 48 hours after axotomy, using phase-contrast and fluorescent imaging techniques. Vitality rates; It was calculated as 50% in the axotomy group, 75% in the TQ group, 90% in the K252a group, and 91% in the TQ+K252a group. It was determined that the regeneration ability and survival rate of TQ and K252a were statistically significantly higher than the control. It was evaluated that both TQ and K252a have regenerative properties, and that K252a triggers regeneration through a different pathway instead of inhibiting the regenerative effect of TQ through Trk A or Trk B.

Keywords: Thymoquinone, K252a, DRG, neuron, axotomy, regeneration

Oral Presentations

Oral Presentation 17

Evaluation of neutrophil gene expressions of apoptosis markers BAX, BCL2, CASP3 during relapsing/remitting multiple sclerosis attack treatment

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Multiple sclerosis (MS) is a chronic inflammatory, neurodegenerative, autoimmune central nervous system disease. The most common type is relapsing/remitting MS (RRMS), in which periods of recovery and relapses (attack) follow each other. Corticosteroids are given to patients during an MS attack. Endoplasmic reticulum (ER) stress occurs when misfolded proteins accumulate in the ER. If ER stress cannot be alleviated, unfolded protein response (UPR) triggers apoptosis. Compared to healthy controls, neutrophil activity biomarkers increase in RRMS patients during an attack. So far, there is no study in the literature examining the ER-stress and apoptosis in neutrophils in MS. The aim of this study was to investigate the effects of corticosteroids on *BAX*, *BCL2* and *CASP3* relative expression levels (RELs), which are markers of apoptosis, in neutrophils during and after a relapse.

For this purpose, whole blood samples were taken from healthy controls (n=53) and RRMS patients during an MS attack before treatment (BT) (n=10), at the end of treatment (AT) (n=10) and 1 month after treatment (1M) (n=10) in Ankara City Hospital, Neurology Clinic. Neutrophils used to obtain RNA were isolated from blood and gene expressions were determined using qRT-PCR.

As a result of this study, *CASP3* REL in 1M group was higher than controls (P=0.038), while no significant difference was found between the controls and BT or AT groups. *BAX* and *BCL2* RELs didn't differ between groups. No significant difference was observed in the comparison of BT, AT and 1M groups.

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Keywords: Multiple Sclerosis; Treatment Response; Apoptosis; Neutrophil; Attack

Oral Presentations

Oral Presentation 18

Longitudinal study of neutrophil gene expressions of ER stress markers ATF6, IRE1, PERK and GRP78 during dimethyl fumarate therapy in patients with multiple sclerosis

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Multiple sclerosis (MS) is a demyelinating auto-immune central nervous system disease. Relapsing-remitting MS (RRMS) presents with periods of relapses and without progression between attacks. Immune-modulating treatments such as dimethyl fumarate (DMF) aim to slow disease progression. Neutrophils are associated with MS pathogenesis, and it is also known endoplasmic reticulum (ER) stress is related to neurodegenerative diseases. An increase in the number of unfolded proteins creates stress in the ER and an unfolded protein response (UPR) develops. UPR is initiated by removing GRP78 bound to IRE1, PERK, and ATF6. The relationship between neutrophil ER stress and MS has not been investigated before. This study examines the changes in the relative expression levels (REL) of *GRP78*, *IRE1*, *PERK*, and *ATF6* in neutrophils before and after DMF treatment.

For this aim, blood samples were obtained from controls (n=52) and naive RRMS patients (0M; n=30), 3 months (3M; n=20) and 6 months (6M; n=14) after they started using DMF at Ankara City Hospital Neurology Clinic. RNA to be used in qRT-PCR was isolated from neutrophils separated from the blood.

According REL analysis, *ATF6* was higher in 6M (P=0.003) and *GRP78* was lower in 0M (P=0.046) compared to the controls. No significant difference was observed in the comparison of the 0M, 3M, and 6M groups.

In conclusion, differences in *GRP78* and *ATF-6* REL may be related to the ER stress in MS pathogenesis and DMF therapeutic mechanism. These are the preliminary results of an ongoing study.

Acknowledgment: This work is supported by TUBITAK (218S578).

Keywords: Multiple Sclerosis, Neutrophils, ER Stress, Dimethyl Fumarate

Oral Presentations

Oral Presentation 19

Investigation of the relationship between *MIR155* rs767649 T>A genetic polymorphism and relapsing-remitting multiple sclerosis

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Multiple Sclerosis (MS) is a chronic inflammatory, autoimmune, neurodegenerative central nervous system disease. Relapsing-remitting MS (RRMS) continues with recovery after attacks. MicroRNAs (miRNA) are a class of non-coding RNAs involved in post-transcriptional regulation. There are many single nucleotide polymorphisms (SNPs) in miRNA genes. miR-155-5p has a role in the regulation of immune responses. The rs767649 T>A polymorphism is in the promoter region of the *MIR155* gene and can alter its expression. The association of this SNP with MS has not been studied in the Turkish population. In this study, association of the rs767649 T>A SNP with MS was investigated.

Blood samples were collected from 230 MS patients and 230 controls by Ankara City Hospital Neurology Clinic. DNA isolation was performed, and rs767649 T>A genotypes were determined using the TaqMan genotyping method.

The groups are similar in terms of age (*P*=0.169) and gender (*P*=0.074). The frequency of the polymorphic A allele was calculated as 0.130 in RRMS patients and 0.147 in the control group (OR=0.945, *P*=0.771). The frequency of the polymorphic AA genotype was calculated as 1.3% in the RRMS patients and 3.0% in the controls. When the frequency of AA genotype was compared with that of TA+TT genotypes, the OR value was 0.421 (CI=0.108-1.649, *P*=0.338) (recessive model); when AA+TA genotype was compared with TT genotype (dominant model), the OR value was 1.024 (CI=0.670-1.565, *P*=0.914).

According to these results, there was no relationship between rs767649 T>A SNP and MS disease. These are the first findings of an ongoing, large-scale study.

Keywords: Multiple Sclerosis, microRNA, Single Nucleotide Polymorphism, TaqMan

Poster Presentations

Poster No, 1

Possible role of TRPM7 channels in neuronal hypoxia: A bioinformatics approach

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Neuronal tissues need to high oxygen consumption and vulnerable to hypoxic conditions that is among the essential physical factors to affect neuronal cell viability. In hypoxic conditions, several transcription factors involve in the nuclear gene expression processes including activating transcription factor 4 (ATF4) and hypoxia-inducible factor 1 alpha (HIF1-α). TRPM7 cation channels are expressed in neurological cells. In this study, it is aimed to investigate that possible role TRPM7 channels in cellular molecular mechanisms with the bioinformatics approaches.

Top ten genes related with hypoxia were searched from the current literature by using Enrichr Tool (Xie et al. 2021) (https://maayanlab.cloud/Enrichr/). Interaction network analysis among the genes as well as TRPM7 were analyzed with String Database (https://string-db.org/). Expression patterns of ATF4, HIF1-α and TRPM7 proteins in human brain tissues were evaluated by using The Human Protein Atlas Database (https://www.proteinatlas.org/). The ATF4 and HIF1-α transcription factors binding points on TRPM7 cation channel gene sequence was defined with The Eukaryotic Promoter Database (https://epd.epfl.ch/) and JASPAR Database (https://jaspar.genereg.net/).

Although it is known that TRPM7 channels are related with hypoxia (Sun, 2017), the channel gene is not listed in the top ten genes related with hypoxia. Multiple protein analyzes result also showed that there is no possible interaction between top genes related with hypoxia and TRPM7 channel. However, when hypoxic transcription factors and TRPM7 channel expression pattern in human brain tissues checked, it is observed that three of proteins are expressed in neuronal tissues. When specific binding points of ATF4 and HIF1-α on TRPM7 gene sequence was evaluated, it was also shown that there are several binding points for these two transcription factors on TRPM7 gene promoter sequence.

In conclusion, TRPM7 cation channels may mediate the secondary effects of hypoxic conditions as they contain binding sites of transcription factors that affect nuclear gene expressions. These finding may be useful against to TRPM7 mediated harmful effects of hypoxia in neuronal cells.

Keywords: Hypoxia; TRPM7; ATF4; HIF1- α; Bioinformatics databases

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Poster Presentations

Poster No, 2

Preparation of SH-SY5Y Neuronal Cell Line for the investigation of the TRP cation channels

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The calcium ion (Ca^{2+}) has an essential role in the induction of several physiological processes such as muscle contraction and mitochondrial functions. The well-known calcium channels include voltage gated Ca^{2+} channels and chemical gated calcium channels. Within the last decades, new channels namely transient receptor potential (TRP) channels were discovered, (Müller et al. 2022). The superfamily contains several members such as TRPM2, TRPM7, and TRPV4. TRPM2 is activated by several stimuli including the ADP-ribose, although naltriben and GSK1016790A are well-known agonists of TRPM7 and TRPV4, respectively (Müller et al. 2022; Nazıroğlu 2022). The three cannels are also activated by oxidative stress.

The managements of animals and human are very difficult for the investigations, and they also need ethical approve applications. The cell culture models are very popular for TRP investigations. The neuroblastoma cell line SH-SY5Y was the source of the triple-subcloned cell line SH-SY5Y. Since the cells may be transformed into various types of functioning neurons by the addition of particular substances, it acts as a model for neurodegenerative diseases. There are several channels that lack naturally occurring TRPM2, TRPM7, and TRPV4 channels, such as Chinese hamster ovary cells (CHO) and human embryonic kidney 293 (HEK293) cells. For the channels, they therefore require a DNA transfection technique. However, TRPM2, TRPM7, and TRPV4 channels are naturally present in SH-SY5Y cells. Hence, they have been using in the channel investigations for a long time (An et al. 2019).

SH-SY5Y cells are cultured in the DMEM (50%) and Ham's F2 medium (50%) mixture. In addition, FBS (10%) and antibiotics (1%) should be added to the medium mixture. The cells should be kept in the cell culture conditions (37 ℃, 5% CO_2, and humidity).

In the presentation. I will summarize how the SH-SY5Y neuronal cell line was prepared for use in the analysis of the TRPM2, TRPM7, and TRPV4 channels.

Keywords: Cell culture; DNA Transfection; SH-SY5Y neuronal cells; TRP channel.

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Poster Presentations

Poster No, 3

Evaluation of plasma hsa-miR-24-3p relative expression level as a biomarker in relapsing-remitting multiple sclerosis

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Multiple sclerosis (MS) is a chronic, autoimmune, and demyelinating disease that affects the central nervous system. The most common type is relapsing-remitting MS (RRMS) with acute attacks. Immunomodulatory treatments such as glatiramer acetate (GA) slow down the prognosis of the disease. MicroRNAs (miRNAs) play important roles in the regulation of gene expression and disease activity. This study aimed to evaluate the biomarker potential of hsa-miR-24-3p, involved in the regulation of inflammatory responses.

Blood samples from 25 naïve 25 RRMS patients treated with GA, and 25 healthy individuals were collected in Ankara City Hospital Neurology Clinic. Relative expression levels (RELs) of hsa-miR-24-3p in plasma were determined by the qRT-PCR method.

The mean plasma hsa-miR-24-3p REL was 3.92 ± 2.19 in the GA group, 3.10 ± 1.87 in the naïve group, and 1.57 ± 1.57 in the controls (P<.001). The mean plasma miR-24-3p REL of the naïve and GA groups patients was significantly higher than the controls (GA vs. control: P<.001, naïve vs. control: P=.01). There was no significant difference between the GA and naïve groups. The diagnostic performance of the mean REL of plasma miR-24-3p between the GA and control groups was "good" (Cut-off point: 1.77, AUC=0.822, P<.001); between the naïve and control groups was "moderate" (Cut-off point: 1.57, AUC=0.751, P=.01) according to ROC analysis.

The plasma miR-24-3p REL has the potential to be developed as a blood biomarker for RRMS; new diagnostic tests aiming to measure this parameter can be developed.

Acknowledgment: This study was supported by TUBITAK (121S345).

Keywords: Multiple sclerosis, RRMS, microRNA, miR-24-3p, biomarker

Poster Presentations

Poster No, 4

Investigation of the relationship between the plasma miR-195-5p relative expression level and RRMS

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Multiple Sclerosis (MS) is a chronic demyelinating, autoimmune, and neurodegenerative disease of the central nervous system, and its main clinical form is relapsing-remitting MS (RRMS). MicroRNAs are important regulators of many physiological processes, and they are studied as candidates for diagnostic, therapeutic and prognostic biomarkers in MS. MiR-195-5p is the regulator of genes involved in cell cycle and apoptosis. Although there are studies showing its association with Alzheimer's and Parkinson's diseases, the role of miR-195-5p in MS is still unknown.

The participants recruited in Ankara City Hospital, Neurology Clinic consist of 30 treatment-naïve RRMS patients, 30 injectable (interferon-β or glatiramer acetate) disease modifying therapy (DMT)-receiving RRMS patients, and 30 healthy controls. Peripheral blood samples collected into sodium-EDTA tubes were centrifuged to obtain the plasma phase. Total RNAs were extracted from plasma samples and, miR-195-5p relative expression levels were determined using qRT-PCR method.

Plasma miR-195-5p relative expression levels determined in naïve RRMS (0.78 ± 1.939), DMT-receiving RRMS (0.15 ± 0.11), and control groups (1.94 ± 2.37) were significantly different between groups (P<.001). The miR-195-5p level was highest in the control group (naïve RRMS vs. control: P=.001, DMT-receiving RRMS vs. control: P<.001), and it was significantly higher in naïve RRMS compared to the DMT-receiving RRMS group (P=.03). Moreover, miR-195-5p was correlated positively with Multiple Sclerosis Severity Score (MSSS) in naïve RRMS (σ=.394, P=.04), but negatively in DMT-receiving RRMS group (σ=-.426, P=.02).

These results indicate that plasma miR-195-5p relative expression might be a potential candidate as a diagnostic, prognostic, therapeutic biomarker in MS.

Keywords: MS, RRMS, microRNA, miRNA, miR-195-5p, biomarker