

HÜCRESEL 11. SİNİRBİLİM GÜNLERİ

5-6-7 Temmuz 2024

İstanbul Medipol Üniversitesi

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ÖZET KİTABI

BİLİM KURULU

Prof. Dr. Ahmet Hacımüftoğlu

Atatürk Ünl. Tıp Fakültesi, Farmakoloji Ana Bilim Dalı, Erzurum.

Prof. Dr. Aydın Hım

Bolu Abant İzzet Baysal Ünl. Tıp Fakültesi, Fizyoloji Ana Bilim Dalı, Bolu.

Prof. Dr. Behar Güntekin

İstanbul Medipol Üniversitesi SABİTA, İstanbul

Prof. Dr. Ender Erdoğan

Selçuk Üniversitesi Tıp Fakültesi, Histoloji-Embriyoloji Ana Bilim Dalı, Konya.

Prof. Dr. Gökkan Öztürk

Bolu Abant İzzet Baysal Üniversitesi Tıp Fakültesi, Bolu.

Prof. Dr. Lutfi Hançğlu

İstanbul Medipol Üniversitesi SABİTA, İstanbul

Prof. Dr. Mustafa Hazroğlu

Süleyman Demirel Üniversitesi, Biyofizik Ana Bilim Dalı, Isparta

Prof. Dr. Mureddin Cangiz

Bandırma Onyedli Eylül Üniversitesi Histoloji ve Embriyoloji Ana Bilim Dalı, Balıkesir

Prof. Dr. Ramazan Bal

Gaziantep Üniversitesi Fizyoloji Ana Bilim Dalı, Gaziantep

Prof. Dr. Servet Kavak

Bakırçay Üniversitesi Biyofizik Ana Bilim Dalı, İzmir

Doç. Dr. Bilal Çığ

Ahi Evran Üniversitesi Fizyoloji Ana Bilim Dalı, Kırşehir

Doç. Dr. İshak Suat Övey

Alanya Alaaddin Keykubat Üniversitesi Fizyoloji Ana Bilim Dalı, Antalya

Prof. Dr. Ömer Çelik

Süleyman Demirel Üniversitesi Biyofizik Ana Bilim Dalı, Isparta

Doç. Dr. Ramazan Üstün

Yüzüncü Yıl Üniversitesi Fizyoloji Ana Bilim Dalı, Van

11. HÜCRESEL SİNİRBİLİM GÜNLERİ

İSTANBUL MEDİPOL ÜNİVERSİTESİ

PROGRAM

5 Temmuz 2024 Cuma

İstanbul Medipol Üniversitesi Kavacak Kuzey Kampüs
SABİTA (Sağlık Bilimi ve Teknolojileri Araştırma Enstitüsü)

10.00 - 13.00 ■ SABİTA Gezisi

14.00 - 18.00 ■ Sinirbilimide İleri Görüntüleme Kursu

Kurs I Dr. Öğr. Üye. Esra Nur Yiğit (İstanbul Medipol Üniversitesi, SABİTA)
Konfokal Mikroskopu ile Nöronlarda Kalsiyum Aktivitesinin
Canlı Görüntülenmesi ve Aksotomi Modeli

Kurs II Arife Ahsan Kaplan (İstanbul Medipol Üniversitesi, SABİTA)
Sinir Sistemi Hücre Kültüründe Korrelatif Konfokal ve
Elektron Mikroskopisi ile İncelenmesi

Kurs III Doç. Dr. Mehmet Şerif Aydın (İstanbul Medipol Üniversitesi, SABİTA)
Lightsheet Mikroskopu ile Sinir Sisteminin Organ Düzeyinde
3 Boyutlu Görüntülenmesi

6 Temmuz 2024 Cumartesi

İstanbul Medipol Üniversitesi Kavacak Güney Kampüs
Konferans Salonu Kat 2

09.00 - 10.00 ■ Kayıt

10.00 - 10.30 ■ Açılış Oturumu

Prof. Dr. **Bahar Güntekin** (İstanbul Medipol Üniversitesi)
Prof. Dr. **Bayram Yılmaz** (Yeditepe Üniversitesi)

10.30 - 11.00

Kahve Arası ve Poster Sunumlar

P01 - Arif Kamil Salıhoğlu

Neuroendocrine Gene Modulations in Cortical Parvalbumin Neurons Between Two Different Transgenic Mice Species

P02 - Melisa Eryaşar, Kıvanç Kök. (School of Medicine, Istanbul Medipol University, Istanbul, Türkiye).

Text Mining-Based Analysis of the Huntington Disease Literature: Identification of Key Concepts and Delineation of Potential Topics in Published Abstracts

P03 - Muhammed Emre Yüzer, Buşra Zencirci, Zümrüt Doğan. (Department of Anatomy, Faculty of Medicine, Adıyaman University, Adıyaman, Turkey)

Different Morphological Imaging Techniques Likely to be Used in Neuroscience

11.00 - 12.30

Nöral Devreler Oturumu

Moderatör: Prof. Dr. Ramazan Bal (Gaziantep Üniversitesi, Tıp Fakültesi, Temel Tıp Bilimleri, Fizyoloji ABD)

Konferans I Dr. Öğr. Üye. Caner Çağlar (Basmilem Üniversitesi BİLSAB)

Beslenme Davranışlarının Hipotalamus Tarafından Düzenlenmesi

Konferans II Prof. Dr. Stefan H. Fuss (Boğaziçi Üniversitesi)

Regulation of Stem Cell Activity in the Zebrafish Nervous System

Sözlü Sunum I Doç. Dr. Bilal Çiğ (Ahi Evran Üniversitesi, Tıp Fakültesi, Fizyoloji ABD)

Neuronal-Glial Interaction in Multiple Sclerosis: Focus on TRP Channels

12.30 - 14.00

Öğle Arası

14.00 - 15.30

Yeni Teknikler Oturumu

Moderatör: Prof. Dr. Nureddin Cengiz (Bandırma Özyedi Eyyül Üniversitesi, Temel Tıp Bilimleri)

Konferans I Doç. Dr. Mehmet Şerif Aydın (İstanbul Medipol Üniversitesi, SABİTA)

Sinir Sisteminin Lightsheet Mikroskop ile Görüntülenmesi

Konferans II Dr. Öğr. Üye. Nur Mustafaoglu (Sabancı Üniversitesi,

Mühendislik ve Doğa Bilimleri Fakültesi

In Vitro Human Blood-Brain Barriers

Sözlü Sunum I Büşra Zencirci, Zümrüt Doğan, Ahmet Türk (Adıyaman Üniversitesi, Tıp Fakültesi)

A New Perspective for Morphometric Analysis in Neuroscience: QUINT Workflow

Sözlü Sunum II Dr. Öğr. Üye. Kıvanç Kök, Melisa Eryeşar, (Dept. of

Biostatistics and Medical Informatics, International School of Medicine, Istanbul Medipol University)

Bioinformatics Analysis of The Huntingtin Protein-Protein Interactome: In Silico Discovery of Hub Proteins and Prediction of Putative Functional Modules

15.30 - 16.00

Kahve Arası ve Poster Sunumlar

P04 - Hatice Doğan Göçman, Okan Tutuk, Duygu Seren Özyılmaz,

Selda Kabadere, Cemil Tümer.

Cognition and Quality of Life in Parkinson's Disease

P05 - Esra Öztürk, Özgur Gedikli, Mehmet Yıldırım (Dep. of Physiology,

Graduate School of Medical Science, Karadeniz Technical University)

Effect of Melatonin on Cognitive and Motor Behaviors in WAG/Rij Rats with Absence Epilepsy

P06 - Seray Yıldız (Fatih Sultan Mehmet Vakıf Üniversitesi)
Tıp Barışında Büyük Kırılma: Galerlin Ruh Merkezi Teorisi

16.00 - 17.30

Nörolojik Bozuklukların Temelleri Oturumu

Moderatör: Dr. Haluk Yaşilkaya (Bakırköy Ruh ve Sinir Hastalıkları Hastanesi)

**Konferans I Prof. Dr. Erdem Tüzün (İstanbul Üniversitesi, Aziz Sancar
Deneyisel Tıp Araştırma Enstitüsü, Sinir Bilimi ABD)**
Nörolojik Hastalıklarda Kompleman Aracılı Mekanizmalar

**Konferans II Prof. Dr. Müge Yemişçi Özkan (Hacettepe Üniversitesi,
Nörolojik Bilimler ve Psikiyatri Enstitüsü, Nörolojik ve Psikolojik Temel Bilimler ABD)**
Serebrovasküler Hastalıkların Moleküler Mekanizmaları ve
Nöroproteksiyon Yaklaşımları

**Sözlü Sunum I Özlem Cemile Demirköken (Department of Medical
Biochemistry Gazi University, Faculty of Medicine)**
Understanding the Relationship Between Depression and
Mitochondrial Dysfunction

**Sözlü Sunum II Ceyda Nur Altınışık, Ramona Gerhards, Mina Mamipour,
Tuğba Özüm, Pınar Acar Özgen, Burcu Yüksel, Zeynep Önderir Acar, Mesude
Tufuncu, Gökçen Gönübatık Çelik, Mesure Koseoğlu, Rana Karabudak,
Gülşen Akman Demir, Meryem Aşık Yüncü, Aysun Soysal, Edgar Meril, Altay Yural**
Detection of Autoantibodies to Oligodendrocyte Myelin Glycoprotein
Antibody (OMGp) in Patients with Atypical Demyelination

7 Temmuz 2024 Pazar

📍 İstanbul Medipol Üniversitesi Kavacık Güney Kampüs
Konferans Salonu Kat 2

10.00 - 11.30

In Vitro Çalışmalar Oturumu Moderatör: Doç. Dr. Ramazan Üstün
(Van Yüzüncü Yıl Üniversitesi-Tıp Fakültesi, Temel Tıp Bilimleri, Fizyoloji ABD)

Konferans I Prof. Dr. Esra Çağrı
Nörokardiyak Etkileşimlerin Mikroakışkan Sistemlerde İncelenmesi ve
iPSC-Temelli ALS In Vitro Modelleri

Konferans II Dr. Ekin Sönmez (Gebze Teknik Üniversitesi, Biyoteknoloji Enstitüsü)

Hastalıkta Elk-1: In Vitro Nörodejenerasyon & Omik

Sözlü Sunum I Dr. Öğr. Üye. Öznur Gedikli, Esra Öztürk, Mehmet Yıldırım (Karadeniz Teknik Üniversitesi, Tıp Fakültesi, Temel Tıp, Fiziyojji)

Melatonin Reduces Spike-Wave Discharges in The WAG/Rij Rat Model of Absence Epilepsy

Sözlü Sunum II Elif Sade Karabay, Gülseren Çelik, Betül Yazgan, Yener Yağın (Kastamonu Üniversitesi)

TRP Kanallarnın Alzheimer ve Parkinson Hastalıklarındaki Rolü

11.30 - 12.00

Kahve Arası ve Poster Sunumlar

P07 - Pelin Başak Zor, İlke Naz Canolur, Canan Akın, Buse Aydın

Alta Ayhan Yılmaz, Birsan Can Demirdöğen (TOBB University of Economics and Technology, Department of Biomedical Engineering, Ankara, Turkey)

Investigation of the Effect of Pulsed Electromagnetic Field Application on PADI2 Gene Expression in PC12 Cells

P08 - Öznur Kalaba(İstanbul Aydın Üniversitesi), **Özlem Merve**

Cansever Güzeleöğlü (Yeditepe Üniversitesi/İstanbul Aydın Üniversitesi)

Cognition and Quality of Life in Parkinson's Disease

P09 - Akil Hakan Kurt, Cansu Kara Öztabağ, Uygur Zarif Sevinç, İrem Kallı

(Department of Medicinal Pharmacology, Faculty of Medicine, Bolu Abant İzzet Baysal University, Bolu, Turkey)

Exploring the Protective and Therapeutic Effects of Spinorfin in an In Vitro Parkinson's Model

P10 - Gizem Yörüköğlü, Sevilay Şahoğlu Göktaş, Yusuf Enes Karo, Behnaz

Karadoğan, Mehmet Şerif Aydın, Gürkan Öztürk, Esra Çağavi

Whole Organ Mapping of Afferent Innervations of the Mouse Heart

12.00 - 13.30

İn-Vivo Çalışmalar Oturumu Moderatör: Prof. Dr. Ender Erdoğan
(Selpuk Üniversitesi Tıp Fakültesi Histoloji ABD)

Konferans I Dr. Öğr. Üye. Yasemin Önder (Kadir Has Üniversitesi)
Ventromedial Hipotalamusta Adaptif GABAerjik Mekanizmaların
Metabolik Etkisinin Analizi

Konferans II Prof. Dr. Mustafa Nazıroğlu (Süleyman Demirel Üniversitesi,
Tıp Fakültesi, Biyofizik ABD)

Alzheimer Hastalığında TRPM2 Katyon Kanal Uyarılması Apoptozis ve
Oksidatif Strese Neden Olur

Sözlü Sunum I Dr. Mustafa Bürgöl, Elgin Uluer Türköz, Tuğba Çavuşoğlu
(Department of Histology and Embriology, Manisa Celal Bayar University)
Effect Of Intermittent Fasting on Semaphorins in An In-Vivo
Experimental Alzheimer Model

Sözlü Sunum II Dr. Mohammadreza Yousefi, Ayşe Özkan, Yiğit Uysalç,
Kaan Kutay Özmen, Dila Atak, Şafak Evren Erdener, Alper Kiraz, Naoto Kawakami,
Yasemin Gürsoy Özdemir, Atay Vural. (Koç University Research Center for
Translational Medicine (KUTTAM))

Deneyssel Otölimün Ensefalomiyelitte Serebrovasküler Reaktivite ve
Kortikal Mikrodolaşımın Bozulması ve Nörodejenerasyon ile İlişkisi

13.30 - 14.30

Öğle Arası

14.30 - 16.00

Translasyonel Yaklaşımlar Oturumu

Moderatör: Op. Dr. Ömer Burak Eriçek (Medipol Üniversitesi, Çamlica
Suam, Beyin ve Sinir Cerrahisi)

Konferans I Doç. Dr. Ali Zırh (İstanbul Medipol Üniversitesi, Tıp Fakültesi,
Cerrahi Tıp Bilimleri Bölümü, Beyin Ve Sinir Cerrahisi ABD)
Hareket Bozukluklarının Tedavisinde Mikroelektrod Kayıt ve
Stimülasyon Tekniği

Konferans II Prof. Dr. İhsan Solanoğlu (Koç Üniversitesi, Tıp Fakültesi,
Nöroşirürji ABD)

Translasyonel Tıp Araştırmaları ve Nörobilim

Sözlü Sunum I Münteha Girgin, Sevim Işık, Shirin Tarbiat, Nigar Kantarcı
Çarşıbaşı (Department of Chemical Engineering, Uskudar University)

Exploring Competitive Inhibition and Synergistic Effects:
Novel Natural Compounds Targeting Acetylcholinesterase for
Alzheimer's Disease Treatment

Sözlü Sunum II Dr. Öğr. Üye. Selahattin Aydemir

The Role of Toxoplasma Gondii in Substance Abuse

16.00 - 16.30

Kahve Arası ve Poster Sunumlar

**P11 - Hasan Berke Bilkı, Halise Ertan, H. Bulut, B. Özhan Zaidi ,
A. Ayyaz , M. Kocaçırık (İstanbul Medipol University)**

Rats Perceive the Change in Motion Direction of a Cursor on PC Monitor

**P12 - Gizem Yörükoğlu, Özlem Mutlu Burnaz, Zahra Babaie, Bihiter Özhan
Zaidi, Fazlı Kemal Bayat, Mehmet Kocaçırık, Barbaros Çetin, Esra Çağcı**

Human iPSC-Derived Sensory Neuron - Cardiac Interactions in
Multielectrode-Integrated Microfluidic Chips

**P13 - Sevgan Mercan, Murhan Çon (Pathology Laboratory Techniques Program,
Health Services of Vocational School, Ondokuz Mayıs University, Samsun, Turkey)**
Alcohol use During Adolescence Activates the Fibrous Astrocytes in
the White Matter of Cerebellum of Male Rats

16.30 - 17.30

Sinirbilim Gündemi Oturumu

Prof. Dr. Gürkan Öztürk

Sinirbilimdeki Güncel Gelişmeler ve Gelecek Perspektifi



AÇILIŞ KONUŞMASI

PROF. DR. BAHAR GÜNTEKİN

PROF. DR. BAYRAM YILMAZ

6 TEMMUZ CUMARTESİ /10.00 - 10.30

"AÇILIŞ OTURUMU"

PROF. DR. BAHAR GÜNTEKİN

İstanbul Medipol Üniversitesi, SABİTA Sinirbilim Araştırmaları Merkezi Müdürü

Prof. Dr. Bahar Güntekin, 2006 yılında Dokuz Eylül Üniversitesi'nde biyofizik alanında doktoraını tamamladı. 2006-2016 yılları arasında İstanbul Kültür Üniversitesi Beyin Dinamiği Araştırma Merkezi'nde müdür yardımcısı olarak çalıştı. İstanbul Medipol Üniversitesi Tıp Fakültesi'ne 2016 yılında Biyofizik Profesörü olarak atandı. Araştırma alanı, sağlıklı kontrollere ve Alzheimer ve diğer demans hastalarında EEG-Event Related Brain Oscillations kullanarak bilişsel işlevleri analiz etmeye odaklanmaktadır. Alzheimer Demeyi'nin ISTAART Elektrofizyoloji Meziyetli İlgili Alan başkanı ve Uluslararası Psikofizyoloji Derneği'nin yönetim kurulu üyesidir. Türkiye Bilimsel ve Teknolojik Araştırma Kurumu (TUBİTAK) Sağlık Bilimleri Araştırma Grubu'nda 2019-2022 yılları arasında danışma kurulu üyesi olarak görev yapmıştır. Scientific Reports, International Journal of Psychophysiology, Aging Brain gibi bilimsel dergilerin Yönetim Kurulu üyesidir. Türkiye Bilimler Akademisi (TUBA) tarafından 2015 yılında, ÖZGÜ Parlatır Vakfı tarafından 2016 yılında ve Elektrofizyoloji ve Klinik Nöroloji Derneği tarafından 2023 yılında Roy John Ödülü'ne layık görülmüştür. Türkiye Bilimler Akademisi asosye üyesidir.

PROF. DR. BAYRAM YILMAZ

TUBAS Derneği Başkanı, Yeditepe Üniversitesi Fizyoloji ABD Başkanı

Prof. Dr. Bayram Yılmaz, 1971 yılında Rize'de doğdu. Lisans ve yüksek lisans eğitimini Fırat Üniversitesi Veteriner Fakültesi'nde tamamladı. Doktoraını 1999 yılında University of Glasgow'da, Tıp Fakültesi Fizyoloji Enstitüsü'nde yaptı. 2007'den beri Yeditepe Üniversitesi Tıp Fakültesi'nde Fizyoloji Anabilim Dalı Başkanı olarak görev yapmaktadır. Aynı zamanda State University of New York at Albany & WING Collaborating Center danışma kurulu üyesidir. Araştırma alanları arasında nöroendokrinoloji, nörobilim, öğrenme endokrinolojisi ve endokrin bozukluklar bulunmaktadır. Uluslararası dergilerde 150 makale, ulusal dergilerde 18 makale yayımlanmıştır ve çoğunlukla 5000'den fazla atıf almıştır. Yurt içinde ve yurt dışında birçok önemli bilimsel kongrede başkanlık yapmış ve sunumları gerçekleştirmiştir. Prof. Dr. Bayram Yılmaz, bilimsel yayınları ve akademik başarılarıyla tanınan bir akademisyendir.



NÖRÖL DEVRELER OTURUMU - KONFERANS II

PROF. DR. STEFAN H. FUSS

BOĞAZİÇİ ÜNİVERSİTESİ ÖĞR. ÜYE

6 TEMMUZ CUMARTESİ / 11.30 - 12.00

"REGULATION OF STEM CELL ACTIVITY IN THE ZEBRAFISH NERVOUS SYSTEM"

The nervous system generally has a low capacity for repair and regeneration. Especially in the mammalian brain, only few sites exist that generate new nerve cells under physiological conditions and in response to traumatic injury. We use the zebrafish olfactory system as an experimental model to identify and functionally test molecular signals that modulate stem/progenitor cell activity. The olfactory epithelium contains two distinct stem cell populations that selectively contribute to tissue maintenance and repair and which are sensitive to different signaling molecules.



NÖRAL DEVRELER OTURUMU - SÖZLÜ SUNUM I

DOÇ. DR. BİLAL ÇİĞ

AHI EVRAN ÜNİVERSİTESİ TIP FAKÜLTESİ FİZYOLOJİ ABD ÖĞR. ÜYE.

6 TEMMUZ CUMARTESİ / 12.00 - 12.15

"NEURONAL-GLIAL INTERACTION IN MULTIPLE SCLEROSIS: FOCUS ON TRP CHANNELS"

Neuronal-glial communication is vital for the proper functioning of the brain. The human brain requires high amounts of white matter for higher brain functions such as social and cognitive learning. Approximately 50% of the white matter in our brain is myelin, but the cortex, referred to as the gray matter in the human brain, is also extraordinarily myelinated compared to other species. Multiple sclerosis (MS) is a demyelinating disease of the central nervous system characterized by neuroinflammation, accompanied by gliosis, phagocytic activity and axonal damage, affecting more than two million people in the world. It is more common in young and middle-aged women than in men. It is more likely to be seen in the optic nerve, spinal cord and brainstem. White matter abnormalities have been found in white matter diseases such as MS and leukodystrophy, as well as psychiatric disorders. Increasing evidence indicates that neuronal-glial communication is important for proper neural network functioning. Ion channels are very important protagonists in this neuro-glial communication. Approximately 25% of the ion channels family consists of the TRP super family (Transient Receptor Potential). Although the physiological and pathophysiological roles of this channel family in myelination and demyelination are not yet fully understood, these channels have the potential to be therapeutic targets in the treatment of many neurological diseases. Hamilton and his team revealed the presence of TRPA1 in oligodendrocyte cells and their role in demyelination for the first time. Findings regarding the role of TRPV1 in MS are conflicting and further research is needed. TRPV2 may play a role in myelination. TRPM2, TRPM4, TRPM7, and TRPA1 all play an important role in MS. We do not have sufficient information about the roles of other TRP subfamilies in MS. In this presentation, I will refer to the physiological-pathophysiological roles of TRP channels in neuronal-glial interaction and their importance as therapeutic targets.

Bilal Çiğ 1

1- Kırşehir Ahi Evran University, Medicine Faculty, Physiology Department 40100, Kırşehir, Türkiye



YENİ TEKNİKLER OTURUMU - KONFERANS II

DR. ÖĞR. ÜYE. NUR MUSTAFAOĞLU

SABANCI ÜNİVERSİTESİ, MÜHENDİSLİK VE DOĞA BİLİMLERİ FAKÜLTESİ

6 TEMMUZ CUMARTESİ / 14.30 - 15.00

“DECIPHERING THE HUMAN BLOOD-BRAIN BARRIER WITH IN VITRO PHYSIOLOGICAL MODELS”

The brain, the most enigmatic organ of the human body, is effectively protected by the blood-brain barrier (BBB), which functions as its administrative center. This complex and dynamic interface of the central nervous system (CNS) separates the brain from the circulatory system and other organs, playing a crucial role in safeguarding brain health. However, the BBB also presents challenges by impeding drug transport to the CNS, complicating the treatment of brain diseases. Despite the BBB's significance, our understanding of the fundamental processes underlying its development, maintenance, and interaction with other bodily systems remains limited. Impaired BBB function has been associated with various neurodegenerative and neurodevelopmental disorders, as well as traumatic brain injury, stroke, and brain malignancies. One major obstacle to effectively treating neurological disorders is the inability of many drugs to penetrate the BBB. This challenge is compounded by the fact that many of these disorders are not yet curable, and our current knowledge about them is incomplete. At the Mustafaoglu Lab, we are addressing these challenges by developing innovative microfluidic systems that mimic the BBB under physiological conditions, both in healthy and diseased states such as epilepsy, brain cancer, and fetal familial insomnia. Our research involves creating new protocols for differentiating stem cells into neurons and brain endothelial cells. Additionally, we design and fabricate novel microfluidic devices capable of applying shear and tensile stresses to cells, simulating physiological vascular movements in the brain. By leveraging these state-of-the-art bioengineering platforms, we aim to deepen our understanding of the human BBB and its implications for health and disease. Ultimately, our goal is to develop unique nano-shuffle systems that can effectively deliver drugs to the brain, offering new avenues for treating and potentially curing various brain diseases.

Nur Mustafaoglu

1 - Sabanci University Nanotechnology Research and Application Center - Istanbul, TR

YENİ TEKNİKLER OTURUMU - SÖZLÜ SUNUM I

BÜŞRA ZENCİRCİ

ADYAMAN ÜNİVERSİTESİ TIP FAKÜLTESİ

6 TEMMUZ CUMARTESİ / 15.00 - 15.15

"A NEW PERSPECTIVE FOR MORPHOMETRIC ANALYSIS IN NEUROSCIENCE: QUINT WORKFLOW"

Neuroscience has recently become a very popular field that includes many studies investigating the diagnosis and treatment methods of neurocognitive diseases. In neuroscience studies conducted for years, rodents are frequently preferred in creating experimental models (Javanmard et al. 2024, Gardon et al. 2023). Determining the areas and volumes of brain morphological areas is of great importance in determining the preliminary diagnosis and treatment protocols of neurodegenerative diseases in terms of determining pathological cases. For this purpose, 3D reference atlases were prepared based on existing stereotaxy atlases. Considering these atlases, the QUINT workflow was created for quantitative and qualitative analysis from rodent brain image series (Kates et al. 2019, Whilden et al. 2021). Aim: In this study, the effectiveness of the QUINT workflow, a new morphometric measurement method, will be investigated using the basal ganglia of normal and demented rats. The QUINT workflow consists of a software package designed to support atlas-based measurement of labeled areas in serial histological images from mouse or rat brain. In this study, histological sections of the basal ganglia of Alzheimer's rats and control group rats were taken and morphometric analyses were performed in the QUINT workflow. As a result of our study, the basal ganglia volumes of rats were analyzed via QUINT. When the volumes of the amygdala, pallidum and striatum of the rats in the Alzheimer's group were examined, a significant difference was observed with the control group: No significant difference was found when the volumes of the nucleus subthalamicus, nucleus accumbens, and substantia nigra structures were compared. Neurodegeneration and Alzheimer's disease are among the most common health problems we encounter. Determining the anatomical areas that atrophy with Alzheimer's is the most fundamental research topic today. Thanks to the QUINT workflow, morphometric analyses can be evaluated easily and reliably. We believe that it will be very useful, especially for researchers dealing with neuroscience.

1. Javanmard, A., Asmadi-Isfahani, M., Tahaei, C., Esmailzadeh, A., Shariqar-Khor, M., Haghayeghi, S. M. H. (2024). In-silico structural analysis of heterocyclization of glycer- amyloid beta: an anti-Alzheimer's peptide. *Mol Biol Res Commun*, 13(1), 29-42. doi:10.22093/mbr.2023.48223.1862
2. Gardon, B., Vain, S. E., Evans, E., Greenbaum, N. E., Haddad, N., Tolpeltchikova, W., Ouellette, A., Ouellette, T., O'Connell, R., Singh, S., Maréchal, T., Morand, E., Borko, J., Kline, H., Schlegel, U., Langford, T. B., Puchades, M. A., Bjalis, J., Gantakornwatt, C. C. (2023). Detecting the effect of genetic diversity on brain composition in an Alzheimer's disease mouse model. *bioRxiv* doi:10.1101/2023.02.27.530298
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SÖZLÜ SUNUM II

DR. ÖĞR. ÜYE. KIVANÇ KÖK

İSTANBUL MEDİPOL ÜNİVERSİTESİ, ÜTF, BİYOSTATİSTİK A.B.D.

6 TEMMUZ CUMARTESİ / 15.15 - 15.30

"BIOINFORMATICS ANALYSIS OF THE HUNTINGTIN PROTEIN-PROTEIN INTERACTOME: IN SILICO DISCOVERY OF HUB PROTEINS AND PREDICTION OF PUTATIVE FUNCTIONAL MODULES"

Huntington's disease (HD) is a rare, devastating, neurodegenerative disease. A mutation in the huntingtin (Htt) protein causes this fatal disease. Despite the growing body of knowledge on this genetic disorder, many aspects of the underlying molecular mechanism remain elusive. Application of systems biology and network bioinformatics in many subfields of neogenetics has a great potential as a complementary methodology to fill in the research gaps. In this work, we implemented a network bioinformatics approach to gain novel insights into HD. To this end, the Htt protein-protein interaction (PPI) interaction data was retrieved from the STRING database. We focused on mining this Htt (PPI) data using Cytoscape software. The reconstructed Htt PPI interactome provided information about the interaction patterns of the Htt partners. More specifically, the topological analysis put forward the central proteins as hub proteins and the 4 delineated topological clusters as putative functional modules. Remarkably, the discovered hubs may be of particular interest as novel HD-relevant proteins and potential drug targets. The subsequent functional annotation analysis, based on multiple gene annotation databases, put the Htt interaction map into a biomedical context and facilitated functional interpretation. The enriched terms included mitochondrial respiratory chain complex 3 and postsynaptic neurotransmitter receptor internalization. Overall, this work contributes to an improved understanding of HD through integrative bioinformatics perspective and shows the applicability of such in silico approach in this field. In this work, only a publically available interactomics dataset was used, which is a limitation of the implemented approach. Integration of multi-omics and machine learning in future computational frameworks and system biology efforts can lead to a deeper understanding of HD.

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NÖROLOJİK BOZUKLUKLARIN TEMELLERİ - KONFERANS II

PROF. DR. MÜGE YEMİŞÇİ ÖZKAN

HACETTEPE ÜNİVERSİTESİ NÖROLOJİK BİLİMLER VE PSİKİYATRİ ENSTİTÜSÜ

6 TEMMUZ CUMARTESİ / 16.30 - 17.00

"SEREBROVASKÜLER HASTALIKLARIN MOLEKÜLER MEKANİZMALARI VE NÖROPROTEKSİYON YAKLAŞIMLARI"

İskemik inme, dünya çapında yüksek mortalite ve morbidite oranları ile ciddi bir sağlık sorunudur. Mevcut tedavi yöntemleri, tıkalı beyin damarlarını açmaya odaklanır; ancak rekanalizasyon tedavileri mikrovasküler seviyesinde her zaman yeterli kan akımı sağlamaz. Bu durum 'no-reflow' fenomeni olarak bilinir. Bu nedenle, rekanalizasyon tedavilerine ek olarak mikrovasküler odaklanılmalı ve iskemik kaskadın birçok yinısını hedef alan kombinö terapiler kullanılmalıdır. Laboratuvarımızda, beyin ve retina iskemisinde küçük damarları büyük sanılara yol açtığı gösterilmiştir.

Nörovasküler ünite (NVU), beyin parankimi ile kapiller düzeydeki damarlar arasında yer alır; beyin kan akımını düzenlenmesinde. Perisit hücrelerinin mikrovasküler düzenlemedeki rolü, beyin ve retina iskemisinde 'no-reflow' fenomeninde önemlidir. Alfa düz kas aktin proteini, perisitlerin kasılmasını sağlayan bir protein olarak tanımlanmıştır ve bu nedenle iskemide önemli bir rol oynar. Farmakolojik tedaviler veya alfa düz kas aktin hedefli siRNA kullanımı, mikrovasküler düzenlenmesinde önemli ve uygulanabilir bir hedef olabilir.

NVU'un disfonksiyonu kan-retina ve kan-beyin bariyerinin bütünlüğünü de etkiler. Laboratuvarımızda, indijeneleşmiş Falsat Topçu 1 (SLC19A1, RFC1) proteininin, kan-retina ve kan-beyin bariyerinde perisitlerde bulunduğu gösterilmiştir. RFC1 seviyelerinin genetik veya farmakolojik olarak modifiye edilmesi, bariyer bütünlüğünün korunmasını sağlayabilir. Beyin ve retina iskemisinde 'istirovaskülerproteksiyon' kritik öneme sahiptir. Hem mikrovasküler hem de tüm hücrelerin korunması, prognozu iyileştirebilir. Patofizyolojinin tam olarak anlaşılması, potansiyel tedavi hedeflerine ulaşmayı sağlayacaktır.

Müge Yemişçi-Özkan 1, 2, 3, 4

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- 2- Tıp Fakültesi Nöroloji Anabilim Dalı
- 3- Nörobilim ve Nöroteknoloji Mükemmeliyet Merkezi (NÖROM), Ankara
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NÖROLOJİK BOZUKLUKLARIN TEMELLERİ - SÖZLÜ SUNUM I

ÖZLEM CEMİLE DEMİRKEŞEN

GAZİ ÜNİVERSİTESİ TIP FAKÜLTESİ TİBBİ BİYOKİMYA ABD

6 TEMMUZ CUMARTESİ / 17.00 - 17.15

"UNDERSTANDING THE RELATIONSHIP BETWEEN DEPRESSION AND MITOCHONDRIAL DYSFUNCTION"

Depression is a complex process involving changes in neurotransmitters, neuromodulators, the hypothalamic-pituitary-adrenal (HPA) axis, cellular and anatomical changes in the brain, interaction of neural networks, inflammation mechanisms, and many unknowns. Recent research indicates that factors related to cellular energy metabolism play a significant role in the onset and progression of depression. Mitochondria, the main source of cellular energy, are also responsible for crucial processes such as oxidative stress, apoptosis, and Ca^{2+} homeostasis. Clinical and preclinical investigations indicate that mitochondrial dysfunction and heightened oxidative stress are significant contributors to the pathophysiology of depressive disorders. Numerous observations have highlighted changes in mitochondrial gene expression, degradation of mitochondrial membrane proteins and lipids, disruptions in the electron transport chain, oxidative stress, cellular apoptosis, and escalated neuroinflammation as key features associated with depression. On the other hand, mitochondrial dysfunction is considered a triggering factor in the development of depressive states. Changes in mood, cognitive function, psychosis, and anxiety have been reported in individuals with mitochondrial diseases and mitochondrial DNA mutations. A study investigating the prevalence of psychiatric disorders in individuals with mitochondrial cytopathies found depressive symptoms in 54% of patients. In addition to mitochondrial dysfunction associated with depression pathogenesis, it is suggested that pre-existing mitochondrial dysfunction due to other causes may also contribute to the development and exacerbation of depression pathogenesis. Improving mitochondrial function may help prevent or alleviate depression-like symptoms. This article will summarize recent research on the relationship between mitochondrial dysfunction and depression pathogenesis. The importance of increasing research on energy metabolism to enhance the effectiveness of current depression treatments will be emphasized.

Özlem Cemile Demirkeşen 1

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NÖROLOJİK BOZUKLUKLARIN TEMELLERİ - SÖZLÜ SUNUM II

CEYDA NUR ALTINIŞIK

KOCÜNİVERSİTESİ, TRANSGLASYONEL TIP ARAŞTIRMA MERKEZİ (KUTİTAM)

6 TEMMUZ CUMARTESİ /17.15 - 17.30

"DETECTION OF AUTOANTIBODIES TO OLIGODENDROCYTE MYELIN GLYCOPROTEIN ANTIBODY (OMGP) IN PATIENTS WITH ATYPICAL DEMYELINATION"

Autoimmune target antigen remains unidentified in many atypical demyelinating disease (ADD) cases. The presence of antibodies against oligodendrocyte myelin glycoprotein (anti-OMGp antibodies) was demonstrated in the sera of a few patients diagnosed with Multiple Sclerosis (MS) and a child with ADEM. Furthermore, animal studies showed that autoimmune reactions targeting OMGp are pathogenic (Gerhardt et al., 2020). However, the clinical implications of anti-OMGp autoantibodies are still unclear. Our study aims to define the clinical and radiological features of anti-OMGp antibody-associated disease in a sizeable demyelinating disease cohort, including MS, Neuromyelitis Optica Spectrum Disorders, Myelin Oligodendrocyte Glycoprotein (MOG) Antibody Disease, and ADD. For this, sera of 542 patients, aged between 18-90, with a female-to-male ratio of 1.62, and cerebrospinal fluid (CSF) samples of 88 patients aged between 18-82, with a female-to-male ratio of 1.60 were screened for anti-OMGp antibodies via flow cytometry-based live cell assay that we optimized. The cut-off value for the test was specified using 30 healthy controls. Nine patients had a precise positivity in serum (diluted 1:50), whereas all CSF samples were negative. Eight patients with antibodies to OMGp were diagnosed with ADD, whereas one with autoimmune encephalitis; all showed ADD features, and none was positive for anti-MOG and anti-AQP4 antibodies. Atypical demyelinating characteristics comprised tumefactive lesions (n=4), ADEM-like lesions (n=3), longitudinally extensive transverse myelitis (n=2), and cortical lesions (n=1). In summary, antibodies targeting OMGp were tested and detected in the sera of cohorts rich in ADD for the first time. Our findings suggest that anti-OMGp antibodies may cause a distinct disease entity and should be tested in individuals with ADD features.

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İN VİTRO ÇALIŞMALAR OTURUMU - KONFERANS II

DR. EKİN SÖNMEZ

GEZİTE TEKNİK ÜNİVERSİTESİ, BİYOTEKNOLOJİ ENSTİTÜSÜ

7 TEMMUZ PAZAR / 10.30 - 11.00

"HASTALIKTA ELK-1:

İN VİTRO NÖRODEJENERASYON & OMİKS"

Transcription factors play a critical role in regulating gene expression (activating, stopping, speeding up, slowing down) and are essential proteins that allow cells to adapt to different conditions, differentiate, and perform specific functions. Elk-1, a member of the ETS transcription factor family, is phosphorylated by MAPK and Mitotic Kinases. Its activity is regulated by various post-translational modifications such as SUMOylation, phosphorylation, and ubiquitination at different sites. Elk-1 is involved in numerous biological processes, including cell survival, growth, differentiation, and cancer development. It has also been shown to play a role in neurodegenerative diseases, with its neuroprotective or neurotoxic effects in neurons depending on the phosphorylation sites. Microarray studies conducted with the overexpression of Elk-1 in SH-SY5Y cells have observed that significantly altered genes are associated with Amyotrophic Lateral Sclerosis (ALS), Huntington's disease (HD), and Parkinson's disease (PD). The analysis of transcriptome data obtained from in vitro or in vivo disease models and correlation-based regulation networks of Elk-1 with ALS, HD, and PD-related genes have demonstrated the potential target genes of Elk-1 and their relationships in disease states. When Elk-1 overexpression occurs before the formation of in vitro disease models, cell death and aggregate analyses have shown neuroprotective effects in both PD and HD disease models. Notably, Elk-1 mutated at S383 or S324 has been observed to significantly reduce HTT aggregate formation. The common point of the three disease models, which have different mechanisms, affected cell types, and pathologies, is the formation of protein aggregates. It has been shown that Elk-1 activation changes depending on its phosphorylation status and that the phosphorylation sites have neuroprotective and neurotoxic effects on the disease. We believe that the effect of Elk-1 on aggregate formation or inhibition in disease states is due to post-translational modifications. We aim to identify the Elk-1 phospho-forms and mutants that are effective in diseases through multi-omics and interactome approaches such as transcriptomics, proteomics, and metabolomics, and then observe their effects on aggregate formation or inhibition through peptide synthesis and application, with the goal of their therapeutic use.

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İN VİTRO ÇALIŞMALAR OTURUMU - SÖZLÜ SUNUM I

DR. ÖĞR. ÜYE. ÖZNUR GEDİKLİ

KARADENİZ TEKNİK ÜNİVERSİTESİ, TIP FAKÜLTESİ, TEMEL TIP, FİZYOLOJİ

7 TEMMUZ PAZAR / 11.00 - 11.15

“MELATONIN REDUCES SPIKE-WAVE DISCHARGES IN THE WAG/RIJ RAT MODEL OF ABSENCE EPILEPSY”

Melatonin has been shown to exhibit anti- or pro-convulsant effects in clinical and experimental studies. In the presented study, it was aimed to investigate the effect of acutely administered melatonin on epileptic seizures *in vivo* electrophysiologically in WAG/Rij rats, a genetic animal model of absence epilepsy. 24 male WAG/Rij rats, 6-month-old, were used in the experiments. Physiological (group I: 2 ml/kg 0.9% NaCl, n=8), melatonin (group II: 80 mg/kg, n=8) and ethosuximide (group III: 100 mg/kg, n=8) were administered as a single dose intraperitoneally to the experimental groups. Electroencephalographic (EEG) recordings were performed during the period covering 30 minutes before and 180 minutes after drug injection (between 10:00 and 14:00). The number and duration of spike-wave discharge (SWD) were analyzed statistically using the Wilcoxon test. When the experimental groups were compared with their baseline values, it was determined that, unlike the control group, 80 mg/kg melatonin and 100 mg/kg ethosuximide significantly reduced the total SWD number and duration ($p<0.05$). According to the findings, acutely administered melatonin showed an anti-absence effect by reducing spike-wave discharges in WAG/Rij rats. Melatonin may be used as a potential treatment agent for absence epilepsy and many other complications associated with epilepsy, such as depression and sleep disorders.

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İN VİTRO ÇALIŞMALAR - SÖZLÜ SUNUM II

ELİF SUDE KARABAY & GÜLSEREN ÇELİK

KASTAMONU ÜNİVERSİTESİ TIP FAKÜLTESİ

7 TEMMUZ PAZAR / 11.15 - 11.30

“TRP KANALLARININ AZHEIMER VE PARKINSON HASTALIKLARINDAKİ ROLÜ”

Nörodegeneratif hastalıklar, ilerleyici nöronal hücre kaybı ile karakterize hastalıklardır. Bu hastalıkların farklı moleküler düzeyde etyolojileri rol oynamaktadır. Bu hastalıkların başında Alzheimer ve Parkinson hastalıkları gelmektedir. Alzheimer hastalığı beyinde prefrontal korteks ve hipokampusta progresif hücre kaybına bağlı olarak yakın geçmişte hatırlanmadaki zorluklarla karakterizedir. Parkinson hastalığında Substantia nigra pars compactadaki (SNpc) orta beyin dopamin nöronlarının seçici ölümü ve striatum gibi hedefteki kontrol etmek için önemli olan hedef beyin bölgelerindeki dopamin seviyelerinin önemli ölçüde azlığı bilinmektedir. Nörodegeneratif hastalıklarda Ca homeostazının bozulmasında TRP kanal aktivasyonu aracılık etmektedir. TRP kanalları katyon geçirgen integral bir protein ailesidir. Altı alt aileye alt 28 TRP üyesi vardır. TRP kanalları beyin olmak üzere her memeli hücrelerinde yaygın ekspresye edilir. İncelenmiş olduğu birçok derinlemesine incelemede elde ettiğimiz sonuçlara göre: Alzheimer hastalığında TRPA1, TRPC3, TRPC6, TRPC1/4/5, TRPM2, TRPV1, TRPV4 önem kazanmaktadır. TRPA1 hücre içersine Ca akışı sağlayarak AD ilerleyişine katkıda bulunmaktadır. TRPC3'ün BDNF ile TRPC6'nın hyperforin ile aktiflenmesi sonucunda hücre içersine Ca akışında bir artış ile çeşitli yaklaşımlar aracılığıyla hipokampal nöronal işlevleri sağlamıştır. TRPC1/4/5 ise heteromultimerler halinde bulunmakta beraber beyin zihinsel, fonksiyonel işlevlerinde önemli rol oynamaktadır. TRPM2 Ca aracı hücre ölümüne neden olmaktadır. TRPV1 kanal aktivasyonu farklı fare modellerinde hastalık için olumlu ve olumsuz sonuçlara neden olabilmektedir. Son olarak TRPV4 ise kolesterolle bağımlı olarak AD patogenezinde önemli bir rol oynamaktadır. Parkinson hastalığında TRPC1, TRPM2, TRPA1, TRPV1 önem kazanmaktadır. TRPC1 aynı itadesinin mitokondriyal membran potansiyelini koruyarak ve anti-apoptotik genler Bcl-2 ve Bcl-xl'in itadesini düzenleyerek MPP+ ile inhibe edilen PC12 hücrelerinin hayatta kalmasını artırdığı da bulunmuştur. TRPM2'nin bozulmaması nöronun H2O2 aracı hasardan korunmuştur. TRPM2'nin bozulmamasının nöroprotektif olduğu gösterilmiştir. TRPV1'in endojen CNF üretimine aracılık ettiğini ve bunun da in vivo olarak dopamin nöronlarını MPP+ toksitesini önlediği gösterilmiştir.

Belül Yargan 1, Yener Yargan 2, Elif Suve Karabay 3, Gülseren Çelik 3

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İN VİVO ÇALIŞMALAR OTURUMU - KONFERANS II

PROF. DR. MUSTAFA NAZIROĞLU

SÜLEYMAN DEMİREL ÜNİVERSİTESİ, TIP FAKÜLTESİ, BİYOFİZİK A.B.D.

7 TEMMUZ PAZAR / 12.30 - 13.00

"ALZHEİMER HASTALIĞINDA TRPM2 KATYON KANAL UYARILMASI APOPTOZİS VE OKSİDATİF STRESE NEDEN OLUR"

Alzheimer hastalığı (AH) kronik bir nörodejeneratif hastalık olup, beyinde yapısal bozulma ve moleküler hasarlar sonucu unutkanlık ile karakterizedir. Günümüzde en az 50 milyon Alzheimer hastası bulunmaktadır ve bu sayı 2050'lere kadar 155 milyona ulaşacağı tahmin edilmektedir. Hücre içi mikrotübül agregatları, nörofibril bozulmaları ve erimeyen beta amiloid (A β) parçacıklarının oluşması AH oluşumuna neden olmaktadır. AH oluşum nedenleri tamamen keşfedilememiştir ve bu nedenle tam kesin tedavisi de yoktur. Üzerinde dünden en önemli mekanizma olarak A β parçacıklarının hücre içi açığı Ca²⁺ artışıyla neden olduğu oksidatif stres önlenmiştir ve human sonucunda glutatyon (GSH) ve selenyum (Se) gibi antioksidanların yeterliliğidir.

Çingili kalsiyum kanalsız ve nörotransmitter maddelerin salınmasını da içersine alan birçok fizyolojik olaya hücre içi serbest Ca²⁺ artışı ile düzenlenmektedir. Ca²⁺ ancak katıyoni kanalları vasıtası ile hücre içersine girebilmektedir. Bu kanallara uzun zamanda bilinenleri voltaja dayalı Ca²⁺ kanalları ve nörotransmitter maddeleri ile aktive olan kimyasal kapılarıdır. Bu kanallara ilave olarak son yıllarda TRP süper ailesi keşfedilmiştir. TRP süper ailesi üyelerinin aktivasyon ve inhibisyon mekanizmaları bu iyi bilinen kapılardan çok farklıdır. Örneğin, TRPM2 kanalı DNA hasarı sonucu oluşan ADP-riboz ile aktive olurlarken, TRPV1 kimyasal acı biberin içersinde bulunan kapsaisin ile aktive olmaktadır. Ayrıca her iki kanal oksidatif stres ile de aktive olmaktadır.

Yakın zamanda yapılan sınavı sayıdaki çalışma AH oluşumu üzerinde TRPM2 kanalının rolüne dikkat çekmiştir. Ayrıca, GSH ve Se gibi TRPM2 kanal antagonisti etki yapan antioksidanların oksidatif stres, açığı Ca²⁺ akışı, apoptozis ve hipokampus ölümünü önleyerek AH üzerinde koruyucu rolünün olduğu bildirilmiştir. Bu konseptimizde, AH oluşumunda TRPM2 katyon kanalının rolü anlatılacaktır ve yakın zamanda yapılan çalışma sonuçları gösterilecektir.

Mustafa Naziroğlu 1,2

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İN VİVO ÇALIŞMALAR OTURUMU - SÖZLÜ SUNUM I

DR. MUSTAFA BİRGÜL

MANİSA ÇELİK BAYIR ÜNİVERSİTESİ, TIP FAKÜLTESİ/ HİSTOLOJİ VE EMBRİYOLOJİ

7 TEMMUZ PAZAR / 13.00 - 13.15

"EFFECT OF INTERMITTENT FASTING ON SEMAPHORINS IN AN IN-VIVO EXPERIMENTAL ALZHEIMER MODEL"

In Alzheimer's disease (AD), in addition to other treatment methods, the beneficial effects of intermittent fasting (IF) are well known (Nicolas & Nolan, 2022). Mechanisms such as neuronal stability, neuroplasticity, and neurogenesis play important roles in this effect of IF (Elias et al., 2022). The aim of our study is to investigate the effect of semaphorins, which are the key components of perineuronal interactions (Quintreuil et al., 2019), on IF-induced changes in AD. The rats were divided into 4 groups: Control, IF, Model AD and Model AD+IF. To create the AD model (ADM), animals were administered Aβ1-42 (50 mg/kg-p.o) for 20 weeks and D-galactose (50 mg/kg/day-i.p) for 10 weeks. Morris Water Maze, Open field test and ELISA (serum I-Tau) were then performed to validate the model. For IF, feeding of animals was limited to 8 hours for 3 months after the 20th week. In addition to behavioral and biochemical tests, Sema-3A, Sema-4C, Sema-7A, Nrp1, Nrp2, AchE and p-Tau levels were determined immunohistochemically at the end of the experiment. In our study, behavioral and biochemical parameters in ADM were in favor of AD, and AD-related molecules such as p-Tau, AchE, Sema-3A, Nrp1 were detected higher compared to the control. These effects were mostly reversed with IF treatment. Sema-4C and Nrp2 levels decreased in ADM, but the effect of IF on them was low. The most notable result of the study is the low detection of Sema-7A (an immunity related semaphorin) levels in the IF applied groups, revealing the importance of neuroinflammatory/neuroimmune pathways in the treatment of AD with IF application.

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İN VİVO ÇALIŞMALAR OTURUMU - SÖZLÜ SUNUM II

DR. MOHAMMADREZA YOUSEFI

KOÇ UNIVERSITY RESEARCH CENTER FOR TRANSLATIONAL MEDICINE (KUTIAM)

7 TEMMUZ PAZAR / 13.15 - 13.30

“DENEYSEL OTOİMMÜN ENSEFALOMİYELİTTE SEREBROVASKÜLER REAKTİVİTE VE KORTİKAL MİKRODOLAŞIMIN BOZULMASI VE NÖRODEJENERASYON İLE İLİŞKİSİ”

The mechanisms underlying neurodegeneration in multiple sclerosis (MS) remain incompletely understood. In this study, we aimed to investigate the role of vascular dysfunction in cortical neurodegeneration using a chronic cranial window model of experimental autoimmune encephalomyelitis (EAE) in mice. Following the establishment of chronic cranial window and induction of EAE with MOG peptides in C57BL/6J mice, we assessed cerebrovascular reactivity (CVR) using both laser speckle contrast imaging (LSCI) and optical signal imaging (IOSI) in awake animals, with stimuli including air puff to the whisker pad and 5% hypercapnia. Histological analysis was performed using immunofluorescence and confocal imaging. We observed a significant reduction in cortical CVR during peak inflammation in the EAE group, as detected by LSCI after 5% hypercapnia ($p=0.008$) and IOSI after whisker pad stimulation. Additionally, there was a significant increase in CD11b⁺ pericyte coverage ($p=0.001$), focal IgG deposition within the microvascular lumen ($p=0.04$), and microglial activation in the cortex of EAE mice ($p=0.04$). Foci exhibiting loss of NeuN reactivity were also detected in subpial and intracortical regions in EAE, but not in control mice ($p=0.03$). Axonal loss, revealed by SM-32 staining, was also evident ($p=0.007$). Furthermore, we observed an increased amount of intravascular CD45⁺ leukocytes stalled in microvessels ($p=0.03$) and infiltrated CD45⁺ leukocytes to the parenchyma in the EAE. Microglial activation was particularly evident around affected microvessels, even in regions without detectable leukocytic infiltration.

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TRANSLASYONEL YAKLAŞIMLAR OTURUMU - SÖZLÜ SUNUM I

MÜNTEHA GİRGİN

ÜSKÜDAR ÜNİVERSİTESİ MOLEKÜLER BİYOLOJİ VE GENETİK / KİMYA MÜHENDİSLİĞİ

7 TEMMUZ PAZAR / 15.30 - 15.45

"EXPLORING COMPETITIVE INHIBITION AND SYNERGISTIC EFFECTS: NOVEL NATURAL COMPOUNDS TARGETING ACETYLCHOLINESTERASE FOR ALZHEIMER'S DISEASE TREATMENT"

Alzheimer's disease is widely recognized as a significant global public health issue. Despite the availability of three FDA-approved drugs (Donepezil, Galantamine, and Rivastigmine), they exhibit low bioavailability and significant side effects. Recent research has focused on identifying new drug candidates with improved bioavailability and reduced hepatotoxicity to achieve effective therapeutic outcomes in Alzheimer's disease (AD). In this study, *in silico* screenings were conducted to identify potent natural molecules capable of inhibiting acetylcholinesterase (AChE) and thus potentially serving as treatments for AD. Our screenings identified Quercetin, Etoposide, and Thiamine as molecules with promising binding affinities and interactions with the AChE protein. Molecular dynamic simulations confirmed the formation of stable complexes between these molecules and AChE. Subsequently, effective half-maximal inhibitory concentrations (IC50) were calculated for each molecule using the FITCA and MIT methods on the SH-SY5Y human neuroblastoma cell line. Additionally, the effects of drug combinations on cells were evaluated using the RTCA method, and their impact on AChE inhibition was assessed using the Ellman technique. Neither individual molecules nor their combinations induced toxicity in the cell line. The results showed inhibition percentages of 70%, 61%, 46%, and 51% for Donepezil, Etoposide, Quercetin, and Thiamine, respectively, in terms of acetylcholinesterase activity. The analysis of drug combinations revealed competitive inhibition in Quercetin-Donepezil and Quercetin-Thiamine combinations, with enzyme inhibition percentages decreasing to 47% and 27%, respectively. Conversely, a significant synergistic effect with 74% inhibition was observed in the Quercetin-Etoposide combination.

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TRANSLASYONEL YAKLAŞIMLAR OTURUMU - SÖZLÜ SUNUM II

DR. SELAHATTİN AYDEMİR

VAN YÜZÜNCÜ YIL ÜNİVERSİTESİ TIP FAKÜLTESİ, PARAZİTOLOJİ BÖLÜMÜ

7 TEMMUZ PAZAR / 15.45 - 16.00

"THE ROLE OF TOXOPLASMA GONDII IN SUBSTANCE ABUSE"

Toxoplasma gondii is an obligate intracellular protozoan of the phylum Apicomplexa. It is a major public health problem as it infects a wide range of vertebrates, including humans. It is estimated that approximately one third of the world's population is infected with toxoplasmosis (Kato, 2018; Rahdar et al., 2023). In the acute phase of toxoplasmosis, the immune system controls the spread of the parasite in the body and alleviates the symptoms of the disease. In the chronic phase of the disease, the parasite transforms into latent tissue cysts in muscle and neurone cells and persists in the infected host for life. The course of the disease varies according to the immune status (Jiel Grande et al., 2017; Malta et al., 2021). *T. gondii* tissue cysts have been reported to be a risk factor for encephalopathy, epileptic seizures, schizophrenia, anxiety and depression depending on their location in the brain. In addition, it has been reported that tissue cysts may affect cellular mechanisms in the cells in which they are located and cause changes in the amounts of neurotransmitters such as Gamma-Amino Butyric Acid (GABA), glutamate, serotonin, dopamine and ions such as calcium, potassium, chloride, magnesium in the cerebrospinal fluid (Ayar et al., 2016). The balance of neurotransmitters such as dopamine, serotonin, GABA and glutamate in the brain is directly related to addiction and substance use. As dopamine is associated with reward and pleasure mechanisms, an increase in the level of this neurotransmitter may contribute to the triggering of addictive behaviours (Karakuş et al., 2021). GABA and its interaction network, the GABAergic system, play an important role in the development of physiological dependence, especially from the use of alcohol and some hypnotics and sedatives. In this context, changes in the GABAergic system may affect the tolerance to substance use and withdrawal symptoms (Karakuş et al., 2021; Kaya et al., 2019). This study was conducted to investigate the relationship between *T. gondii* and substance abuse. The study included 90 substance abusers who applied to the AMATEM service of SBU Van Training and Research Hospital. As a control group, 40 individuals who applied to any outpatient clinic of SBU Van Training and Research Hospital and who were not substance abusers were included. The presence of anti-*Toxoplasma gondii* IgG antibodies in serum was determined using ELISA IgG kit (DRG, Germany). In this study, *T. gondii* IgG antibodies were found in 57.8% of substance abusers and 27.5% of controls ($P=0.001$). In conclusion, the high rate of *T. gondii* seroprevalence in substance abusers suggests that this factor plays a role in substance abuse.

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POSTER SUNUMLAR

Neuroendocrine Gene Modulations in Cortical Parvalbumin Neurons Among Two Different transgenic Mice Species

Arif Kamil Salıhoğlu - (Karadeniz Technical University, Faculty of Medicine, Department of Physiology)

Text mining-based analysis of the Huntington disease literature: Identification of key concepts and delineation of potential topics in PubMed abstracts

Melisa Eryazar, Kınan Kılıç - (School of Medicine, Istanbul Medipol University, Istanbul, Türkiye, Regenerative and Restorative Medicine Research Center (REMERC), Research Institute for Health Sciences and Technologies (SABITA), Istanbul Medipol University, Istanbul, Türkiye)

Different morphological imaging techniques likely to be used in neuroscience

Mahammed Emre Yüzer, Büğra Zencirci, Zümrüt Doğan
(Department of Anatomy, Faculty of Medicine, Adıyaman University, Adıyaman, Turkey)

Role of Semaphorin 4D in Pentylenetetrazole Induced Kindling Model of Epilepsy

Hatice Doğan Güçmen, İlkan Tatuk, Duygu Seren Özyılmaz, Selda Kabadere, Cemil Tümer -
(Department of Physiology, Faculty of Medicine, Hatay Mustafa Kemal University, Hatay, Turkey)

Effect of Melatonin on Cognitive and Motor Behaviors in WAG/Rij Rats with Absence Epilepsy

Esra Öztürk, Ömer Gedikli, Mehmet Yıldırım
(Department of Physiology, Graduate School of Medical Science, Karadeniz Technical University, Trabzon, Türkiye)

POSTER SUNUMLAR

A Major Breakthrough in Medicine History: Galen's Theory of the Soul's Center

Seray Yıldız - (Department of History of Science PhD Student, Fatih Sultan Mehmet Foundation University, İstanbul)

The comparison of clinical parameters of patients with RRMS by gender

Birsen Can Demirdöğen, Melisela Kalmar- (TOBB University of Economics and Technology, Department of Biomedical Engineering, Ankara, Turkey)

Investigation of the effect of pulsed electromagnetic field application on P402 gene expression in PC12 cells

Pelin Başak Zor, **İlke Naz Çavdar**, Canan Akpin, Buse Aydın, Ata Ayhan Yılmaz, Birsen Can Demirdöğen- (TOBB University of Economics and Technology, Department of Biomedical Engineering, Ankara, Turkey)

Cognition and Quality of Life in Parkinson's Disease

Özmar Kalaba1, **Özlem Merve Cansever Güzeloglu 2-**(1- Department of Psychology, Faculty of Science and Literature, Istanbul Aydın University/ Remzi Akman Psychiatry Clinic), (2- Department of Neuroscience, Graduate School of Health Sciences, Yeditepe University / 1 Istanbul Aydın University, Faculty of Arts and Sciences, Psychology)

Alcohol use during adolescence activates the fibrous astrocytes in the white matter of cerebellum of male rats

Nurhan Çan and **Sevcan Mercan** - (Pathology Laboratory Techniques Program, Health Services of Vocational School, Ondokuz Mayıs University, Samsun, Turkey)

POSTER SUNUMLAR

Whole Organ Mapping of Afferent Innervations of the Mouse Heart

Sevilay Şahinçiftçi Göktaş, Yusuf Enes Kızı, Belma Karadoğan, Mehmet Şerif Aydın, Gülcan Öztürk, Esra Çağcı - (Güzem Yürükoğlu Research Institute for Health Sciences and Technologies (SABITA), Istanbul Medipol University, Istanbul, Turkey and Medical Biology and Genetics Program, Institute of Health Sciences, Istanbul Medipol University, Istanbul, Turkey)

Rats perceive the change in motion direction of a cursor on PC monitor

H. Bulut, B. Öztan Zaidi, A. Aygöz, M. Kocatürk- (Hasan Berke Bilgi ve Halise Erten İstanbul Medipol Üniversitesi)

Human iPSC-Derived Sensory Neuron - Cardiac Interactions in Multielectrode - Integrated Microfluidic Chips

Güzem Yürükoğlu, Özlem Mutlu Burnaz, Zehra Bataie, Bilal Öztan Zaidi, Faruk Kemal Bayat, Mehmet Kocatürk, Barbaros Çetin, Esra Çağcı - (Research Institute for Health Sciences and Technologies (SABITA), Istanbul Medipol University, Istanbul, Turkey and Medical Biology and Genetics Program, Institute of Health Sciences, Istanbul Medipol University, Istanbul, Turkey)