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An International Journal of Experimental and Clinical Anatomy

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Title page

In all manuscripts the title of the manuscript should be written at the top and the full names and surnames and titles of the authors beneath. These should be followed with the affiliation of the author. Manuscripts with long titles are better accompanied underneath by a short version (maximum 80 characters) to be published as running head. In the title page the correspondence address and telephone, fax and e-mail should be written. At the bottom of this page, if present, funding sources supporting the work should be written with full names of all funding organizations and grant numbers. It should also be indicated in a separate line if the study has already been presented in a congress or likewise scientific meeting. Other information such as name and affiliation are not to be indicated in pages other than the title page.

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- *Standard entire book:* Sengul G, Watson C, Tanaka I, Paxinos G. Atlas of the spinal cord of the rat, mouse, marmoset, rhesus and human. San Diego (CA): Academic Press Elsevier; 2013. 360 p.

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- *Citation to a book on the Internet:* Bergman RA, Afifi AK, Miyauchi R. Illustrated encyclopedia of human anatomic variation. Opus I: muscular system [Internet]. [Revised on March 24, 2015] Available from: http://www.anatomyatlases.org/AnatomicVariants/AnatomyHP.shtml

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- *Contributed section with editors:* Johnson D, Ellis H, Collins P, editors. Pectoral girdle and upper limb. In: Standring S, editor. Gray's anatomy: the anatomical basis of clinical practice. 29th ed. Edinburgh (Scotland): Elsevier Churchill Livingstone; 2005. p. 799–942.

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15th Turkish Neuroscience Congress

7–10 May 2017, Sakarya, Turkey

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Welcome Address of the Congress Presidents

Anatomy 2017;11(Suppl 1):Sv

Dear Neuroscientists,

It is our great pleasure to welcome you to the 15th National Neuroscience Congress that is being organised by the Neuroscience Society of Turkey (www.tubas.org.tr) and hosted at Sakarya University in Sakarya between 7–10 May 2017.

The congress will begin with six different workshops on various aspects of neuroscience on the first day. The keynote lecture will be delivered by Professor Gazi Yaşargil (the World Brain Surgeon of the 20th Century) and Professor Fahrettin Keleştimur (President, Turkish Institutes of Health, TÜSEB) later on the same day. Professors Paul Bolam (Oxford University), Banu Onaral (Drexel University), Gareth Leng (University of Edinburgh), Deniz Kırık (Lund University), Hagai Bergman (Hebrew University), Daniela Schulz (Yeditepe University), Dirk Hermann (University Hospital Essen), Feyza Arıcıoğlu (Marmara University), İlhan Raman (Middlesex University) and Tarık Tihan (University of California at San Francisco) will give lectures and meet with the congress participants.

There will be eight symposia, 11 conferences and two panels on all aspects of neuroscience. A workshop on publishing of scientific papers will be introduced by Professor Paul Bolam for young neuroscientists. A total of 60 oral presentations will be also be included in the scientific program. On each day of the congress, posters will be displayed at the exhibition hall at lunch time.

We would like to gratefully acknowledge that this congress is supported by 12 different sponsors, federations and institutions. Congress delegates will have the opportunity to visit and meet representatives of the sponsors in the exhibition area during the entire meeting.

There will also be an exciting social program; a welcome reception at the congress center on the evening of 7th May and the Gala Dinner on the evening of 9th May (Altın Güneş Garden Park) on the side of Sapanca lake. Congress delegates will have an opportunity to visit cultural and natural heritage of Sakarya during the meeting.

Turkish neuroscience meetings have a history of over 20 years and the first National Neuroscience Congress was held in 2001. Participants to the meetings have increased over the years and diversified from all pre-clinical and clinical disciplines. There will be about 400 neuroscientists from various departments at the congress.

We are looking forward to seeing you all in Sakarya and very much hope that you will contribute to and benefit from an excellent scientific program and also enjoy the social events.

Sincerely,

Congress Co-Presidents

Prof. Dr. Bayram Yılmaz *(Neuroscience Society of Turkey)*
Prof. Dr. Nurettin Cengiz *(Sakarya University)*

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15th Turkish Neuroscience Congress

7–10 May 2017, Sakarya, Turkey

Program Schedule

7 May 2017 Sunday		*8 May 2017 Monday*		*9 May 2017 Tuesday*		*10 May 2017 Wednesday*	
10.00–16.30	**Workshops**	08.45–12.30	**Scientific Program**	08.30–12.35	**Scientific Program**	08.30–13.00	**Scientific Program**
16.30–17.00	**Opening Ceremony**	12.20–14.00	**Lunch & Poster Presentations**	12.35–14.00	**Lunch & Poster Presentations**	13.00–14.00	**Lunch & Poster Presentations**
17.00–18.00	**Conference 1**						
18.00–19.00	**Conference 2**	14.00–18.30	**Scientific Program**	14.00–18.30	**Scientific Program**	14.00–16.40	**Scientific Program**
19.30–21.00	**Reception**			20.00–23.00	**Gala Dinner**	16.40–17.00	**Closing Ceremony**

7 May 2017 Sunday

10.00–17.00	**Registrations**
10.00–16.30	**Workshops**

Workshop 1

The Use of Compact Organ Electrophoresis System for Clearing of the Brain Tissue (CLARITY)

Prof. Dr. Esat Adıgüzel, Dr. Ayşegül Güngör Aydın *(Pamukkale University)*

Workshop 2

Electromyography and Human Reflexes

Prof. Dr. Kemal Türker, Dr. Gizem Yılmaz, Dr. Görkem Özyurt *(Koç University)*

Workshop 3

Tractography Methods Apply to Cerebral White Matter Using Diffusion Tensor Images and Demonstrate 3D

Prof. Dr. Niyazi Acer *(Erciyes University)*

Workshop 4

Applied Basic Biostatistics Course

Doç. Dr. Ünal Erkorkmaz *(Sakarya University)*, Doç. Dr. Zübeyir Bayraktaroğlu *(Istanbul Medipol University)*

Workshop 5

Principles of Functional Near Infrared Spectroscopy-fNIRS

Prof. Dr. Lütfü Hanoğlu *(İstanbul Medipol University)*, *Dr. Sinem Burcu Erdoğan (Acıbadem University)*

Hall 1		
	16.30–16.50	**Opening Ceremony**
	16.50–17.00	**Prof. Dr. Yücel Kanpolat Session** "To the Memory of Late Prof. Dr. Yücel Kanpolat" Prof. Dr. Turgay Dalkara
	17.00–18.00	**Conference 1** **Chairs:** Prof. Dr. Canan Aykut Bingöl, Prof. Dr. Bayram Yılmaz "Traumatic Brain Injury (TBI)-Induced Neuroendocrine Changes" Prof. Dr. H. Fahrettin Keleştemur
	18.00–19.00	**Conference 2** **Chairs:** Prof. Dr. Canan Aykut Bingöl, Prof. Dr. Bayram Yılmaz "The Significant Relationship Between Physiology, Neurophysiology and Neurosurgery" Prof. Dr. M. Gazi Yaşargil
	19.30–21.30	**Reception – Sakarya University Congress Center**

8 May 2017 Monday

Hall 1		
	08.45–10.15	**Oral Presentations (O-01–O-06)** **Chairs:** Prof. Dr. Elvan Özbek, Doç. Dr. Selma Düzenli
Hall 2		
	08.45–10.15	**Oral Presentations (O-07–O-12)** **Chairs:** Doç. Dr. Emine Eren Koçak, Doç. Dr. Ayşe Demirkazık
Hall 3		
	09.00–16.00	**Stroke Symposium**
	10.15–10.45	**Coffee Break**
Hall 1		
	10.45–11.30	**Conference 3** **Chair:** Prof. Dr. Emel Ulupınar "The Functional Organisation of the Basal Ganglia" Prof. Dr. Paul Bolam
	11.35–12.20	**Conference 4** **Chair:** Prof. Dr. Filiz Onat "Seizures and Epilepsy: From Lab to Clinics" Prof. Dr. Çiğdem Özkara
Hall 2		
	11.35–12.20	**Conference 5** **Chair:** Prof. Dr. Ümit Şehirli "The Role of Dopamine Functions in Animal Models of Depression" Yrd. Doç. Dr. Daniela Schulz
	12.20–14.00	**Poster Communications & Lunch**
		Poster Communications Moderators: Group 1: Prof. Dr. Gülgün Şengül, **Group 2:** Prof. Dr. Işıl Aksan Kurnaz, **Group 3:** Doç. Dr. Birsen Aydemir

Hall 1	
14.00–16.00	**Symposium 1: "Brain Banks, Neurodegeneration and Biomarkers"** **Chairs:** Prof. Dr. Turgay Çelik, Doç. Dr. Esra Yazıcı
	"Biomarkers of Two Main Types for Alzheimer's Disease" Prof. Dr. Engin Eker
	"Definitive Diagnosis of Demenia and Brain Banking" Prof. Dr. Ahmet Turan Işık
	"Can Neurodegeneration Be Mimicked?" Prof. Dr. Turgay Çelik
Hall 2	
14.00–16.00	**Symposium 2: "Basal Ganglia and Deep Brain Stimulation"** **Chairs:** Prof. Dr. Hagai Bergman, Prof. Dr. Atilla Erol
	"Computational Physiology of the Basal Ganglia and Deep Brain Stimulation: Closing the Loop Between Health, Disease and Treatment" Prof. Dr. Hagai Bergman
	"Deep Brain Stimulation and Target Selection in Movement Disorders" Yrd. Doç. Dr. Boran Urfalı
	"Functional Physio-Anatomical Network of Basal Nuclei: Past, Present and Future" Doç. Dr. İlkan Tatar
16.00–16.30	**Coffee Beak**
Hall 1	
16.30–17.30	**Oral Presentations (O-13–O-16)** **Chairs:** Prof. Dr. Şerif Demir, Prof. Dr. Fatma Töre
Hall 2	
16.30–17.30	**Oral Presentations (O-17–O-20)** **Chairs:** Prof. Dr. Neslihan Seral Şengör, Yrd. Doç. Dr. Mehmet Ozansoy
Hall 3	
16.30–17.30	**Oral Presentations (O-21–O-24)** **Chairs:** Prof. Dr. Esat Adıgüzel, Yrd. Doç. Dr. Bilgehan Acar
Hall 1	
17.35–18.30	**Conference 6** **Chair:** Prof. Dr. Gürkan Öztürk
	"Observing the Brain-on-Task Using Functional Optical Brain Imaging" Prof. Dr. Banu Onaral

9 May 2017 Tuesday

Hall 1	
08.30–10.30	**Symposium 3: "Growth Factors in Neurological and Psychiatric Disorders"** **Chairs:** Prof. Dr. Turgay Dalkara, Prof. Dr. Müge Yemişçi
	"Growth Factors in Neurological and Psychiatric Disorders" Prof. Dr. Turgay Dalkara
	"Growth Factors in Neuroprotection: Importance of Administration Route and Timing" Prof. Dr. Müge Yemişçi
	"The Role of FGF2 and FGF-As in Affective Disorders" Doç. Dr. Emine Eren Koçak
	"Neurotrophic Factors and Para-neuroinflammation" Doç. Dr. Hülya Karataş Kurşun

Hall 2		
	08.30–10.30	**Symposium 4: "*In Silico* Drug Design"** **Chairs:** Prof. Dr. Kemal Yelekçi, Doç. Dr. Demet Akten
		"In Silico Design of Novel and Selective Neuronal Nitric Oxide Synthase (nNOS) Inhibitors" Prof. Dr. Kemal Yelekçi
		"Investigation of Allosteric Coupling in Human β2-Adrenergic Receptor (β2-AR) in the Presence of Intracellular Loop 3" Doç. Dr. Demet Akten
		"Homology Modeling of Human Dopamine Transporter; Understanding the Difference Between Inhibitors and Substrates" Dr. Teodora Đikić
	10.30–11.00	**Coffee Break**
Hall 1		
	11.00–11.50	**Conference 7** **Chair:** Prof. Dr. Kemal Türker
		"Oxytocin: The Sweet Hormone?" Prof. Dr. Gareth Leng
	11.50–12.35	**Oral Presentations (O-25–O-27)** **Chairs:** Prof. Dr. Muzaffer Sindel, Doç. Dr. Meral Yüksel
Hall 2		
	11.50–12.35	**Oral Presentations (O-28–O-30)** **Chairs:** Prof. Dr. Fatma Sultan Kılıç, Yrd. Doç. Dr. Neslihan Alagöz
Hall 3		
	11.50–12.35	**Oral Presentations (O-31–O-33)** **Chairs:** Doç. Dr. Arzu Aral, Yrd. Doç. Dr. Birsen Elibol
	12.35–14.00	**Poster Communications & Lunch**
		Poster Communications Moderators: Group 4: Prof. Dr. Ertuğrul Kılıç, **Group 5:** Prof. Dr. Müge Yemişçi, **Group 6:** Prof. Dr. Fatma Töre, **Group 7:** Prof. Dr. Ramazan Bal
Hall 1		
	14.00–16.00	**Symposium 5: "Approach to ALS and Spinal Cord Injury Patients and Improving Their Quality of Life"** **Chairs:** Yrd. Doç. Dr. Tunç Laçin, Prof. Dr. Yeşim Parman
		"Diagnosis and New Treatment Options in ALS" Prof. Dr. Yeşim Parman
		"Acute Traumatic Spinal Injury Management" Prof. Dr. Erkan Kaptanoğlu
		"Principles of Rehabilitation in Amyotrophic Lateral Sclerosis and Spinal Cord Injury" Dr. Esra Giray
		"Nutrition Treatment in Amyotrophic Lateral Sclerosis Patients" Doç. Dr. F. Esra Güneş
		"Effect of Diaphragm Pacing on Quality of Life of ALS and Spinal Cord Injury Patients, Who to Pace and When to Pace?" Yrd. Doç. Dr. Tunç Laçin
		"Living with ALS" İsmail Gökçek *(President of ALS MNH Patients Society)*
Hall 2		
	14.00–16.00	**Symposium 6: "Cutting Edge Technological Methods in Neuroscience Research"** **Chairs:** Doç. Dr. Aydın Him, Yrd. Doç. Dr. Deniz Atasoy
		"*In Vivo* Micro Sensor Technology in the Diagnosis and Treatment of Brain Diseases" Prof. Dr. Ahmet Hacımüftüoğlu

		"Acute Impact of VMH Activity on Appetite" Yrd. Doç. Dr. Deniz Atasoy
		"Dissecting Myelination and Demyelination at High Resolution" Yrd. Doç. Dr. Bilal Kerman
	16.00–16.30	**Coffee Break**
Hall 1		
	16.30–17.45	**Oral Presentations (O-34–O-38)** **Chairs:** Prof. Dr. Hale Sayan Özaçmak, Doç. Dr. Hülya Karataş Kurşun
Hall 2		
	16.30–17.45	**Oral Presentations (O-39–O-43)** **Chairs:** Prof. Dr. Nurcan Dursun, Doç. Dr. İsmail Abidin
Hall 3		
	16.30–17.45	**Oral Presentations (O-44–O-48)** **Chairs:** Prof. Dr. Ramazan Bal, Doç. Dr. Gamze Tanrıöver
Hall 1		
	17.45–18.30	**Conference 8** **Chair:** Prof. Dr. Nurettin Cengiz
		"Theory and Practice of Reporting Diffuse Glial Tumors in Adults" Prof. Dr. Tarık Tihan
Hall 2		
	17.45–18.30	**Conference 9** **Chair:** Prof. Dr. Ahmet Hacımüftüoğlu
		"New Insight into the Pathogenesis and Treatment of Psychiatric Disorders: Inflammation" Prof. Dr. Feyza Arıcıoğlu
	20.00–23.00	**Gala Dinner (Altın Güneş Garden Park, Sapanca Lake)**

10 May 2017 Wednesday

Hall 1		
	08.30–10.30	**Symposium 7: "Understanding Neural Networks"** **Chairs:** Prof. Dr. Işıl Aksan Kurnaz, Prof. Dr. Ender Erdoğan
		"Pea3 Family of Transcription Factors in Neural Circuit Formation and Selectivity" Prof. Dr. Işıl Aksan Kurnaz
		"A Grave Dilemma: Get Connected, We Die" Prof. Dr. Gürkan Öztürk
		"Molecular Control of Motor Neuron Circuitry Development" Prof. Dr. Emel Ulupınar
		"A Biochemical View to Axonal Outgrowth" Doç. Dr. Meral Yüksel
Hall 2		
	08.30–10.30	**Symposium 8: "Multimodal, Personalized Treatment and Neuromodulation in Neurodegenerative Diseases"** **Chairs:** Prof. Dr. Lütfü Hanoğlu, Prof. Dr. A. Savaş Çilli
		"Neuromodulatory Treatment Approaches in Neurodegenerative Diseases" Prof. Dr. Lütfü Hanoğlu
		"The Role of Neuromodulation in Neurodegenerative Diseases: *In-vivo* and *In-vitro* Studies" Doç. Dr. Burak Yuluğ

		"The Human Microbiome in Alzheimer's Disease as Potential Source of Biomarkers and Therapy" Doç. Dr. Süleyman Yıldırım
		"EEG Brain Oscillation Abnormalities in Neurodegenerative Diseases" Prof. Dr. Bahar Güntekin
		"Role of Steroids and Neurosteroids in the Pathogenesis and Treatment of Neurodegerative Disorders" Prof. Dr. Mustafa Öztürk
	10.30–11.00	**Coffee Break**
Hall 1		
	11.00–11.50	**Conference 10** **Chair:** Prof. Dr. Gülgün Şengül
		"Understanding the Value of Alpha-Synuclein as a Biomarker in Parkinson's Disease Dementia: A Story from Animal Models to Human Biospecimens" Prof. Dr. Deniz Kırık
	11.55–13.00	**Oral Presentations (O-49–O-52)** **Chairs:** Prof. Dr. Fazilet Aksu, Doç. Dr. Ebru Bodur
Hall 2		
	11.55–13.00	**Oral Presentations (O-53–O-56)** **Chairs:** Prof. Dr. Tamer Demiralp, Prof. Dr. Metehan Çiçek
Hall 3		
	11.55–13.00	**Oral Presentations (O-57–O-60)** **Chairs:** Prof. Dr. İbrahim Tuğlu, Prof. Dr. Naciye Yaktubay Döndaş
	13.00–14.00	**Poster Communications & Lunch**
		Poster Communications Moderators: Group 8: Prof. Dr. Ahmet Hacımüftüoğlu, **Group 9:** Prof. Dr. Meltem Bahçelioğlu, **Group 10:** Prof. Dr. Emel Ulupınar
Hall 1		
	14.00–14.50	**Conference 11** **Chair:** Prof. Dr. Gönül Peker
		"Contemporary Research and Issues on Cognitive Processes Involved in Visual Word Recognition: The Case of Turkish Orthography" Doç. Dr. İlhan Raman
Hall 2		
	14.00–14.50	**Conference 12** **Chair:** Prof. Dr. Ertuğrul Kılıç
		"Promoting Brain Remodeling and Plasticity in Ischemic Stroke: Underlying Mechanisms, Potential Pitfalls and Clinical Translation Strategies" Prof. Dr. Dirk Hermann
	14.50–15.15	**Coffee Break**
Hall 1		
	15.15–16.40	**Panel 1: Chair:** Prof. Dr. Bayram Yılmaz "Neuroscience in Turkey and Future Perspectives, 2017" Prof. Dr. Emel Ulupınar, Prof. Dr. Gülgün Şengül, Prof. Dr. Metehan Çiçek, Prof. Dr. Tamer Demiralp, Prof. Dr. Cafer Marangoz, Prof. Dr. Gürkan Öztürk, Prof. Dr. Ertuğrul Kılıç ve Bayram Yılmaz
Hall 2		
	15.15–16.40	**Panel 2: Chair:** Prof. Dr. Paul Bolam "The Publishing of Scientific Papers" Prof. Dr. Paul Bolam
Hall 1		
	16.40–17.00	**Presentation of Awards & Closing Ceremony**

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Conferences

(C-01 — C-12)

C-01

Traumatic brain injury (TBI)-induced neuroendocrine changes

Fahrettin Keleştimur

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Traumatic brain injury which is a growing public health problem worldwide has recentlybeen recognized as one of the most common causes of hypopituitarism.The causes of TBI-induced pituitary dysfunction are car accidents, falls, violence and war accidents including blast-related brain injuries. Neuroendocrine abnormalities were also reported in athletes dealing with contact sports including boxing and kickboxing. Boxing and kickboxing are characterized by chronic repetitive head trauma and they are accepted as mild traumatic brain injury. The prevalence of hypopituitarism after TBI is about 30%. GH is the most common hormone lost. The mechanisms underlying the hypopituitarism are still unclear; however, recent studies have demonstrated that genetic predisposition, neuroinflammation and autoimmunity may be responsible for the development of pituitary dysfunctiom. The frequency of hypopituitarism is significantly lower in TBI victims with APO E3/E3 than in victims without APO E3/E3 genotype. The positivity of anti-pituitary and anti-hypothalamic antibodies is also a significant risk factor. Altered expression of miR-126-3p and miR-3610 may play an important role in the occurrence of hypopituitarism after TBI. Treatment of hypopituitarism with appropriate replacement therapies is beneficial in the improvement of manifestations caused by insufficient hormones.

C-02

The significant relationship between physiology, neurophysiology and neurosurgery

M. Gazi Yaşargil

Department of Neurosurgery, Faculty of Medicine, Yeditepe University, Istanbul, Turkey

Advances in mathematics, physics, chemistry, pharmacology and biology in the 19th century established a solid foundation that shaped and intensified the evolution in neuroanatomy, neuropathology and neurophysiology. In the 20th century, scientific technology created neurovisualisation, neuro-recording, neuromolecular biology, neurogenetics and neuroanesthesiology which resulted in the founding of neurodiagnostic and neurotherapeutic specialties. The many electronic media for immediate communication are an advantage and greatly appreciated. However, a closer and more assertive cooperation for exchange of knowledge and mutual support is necessary. In each field of neuroscience substantial data has been acquired and, at the same time, many questions have emerged, some examples of which will be presented and discussed.

C-03

Dopamine neurons, synapses and susceptibility in Parkinson's disease

Paul Bolam

Department of Pharmacology, MRC Brain Network Dynamics Unit, University of Oxford, Oxford, U.K

Genes, protein aggregates, environmental toxins and other factors associated with Parkinson's disease (PD) arewidely distributed in the nervous system and affect many classes of neurons. Theories that explain the loss of dopamine neurons in PD must account for the exceptional and selective vulnerability of dopamine neurons of the substantia nigra pars compacta (SNc) and their selective vulnerability to toxins. Some molecular differences between susceptible and non-susceptible dopamine neurons may contribute to differential susceptibility but there is very little difference in the electrical activity and afferent synapses of different populations of dopamine neurons (Brown et al., 2009; Henny et al., 2012). However, the axon and synaptic output of SNc dopamine neurons are remarkably different to other populations of dopamine neurons and to all other neurons in the brain. Individual dopamine neurons

give rise to hundreds of thousands of synapses in their target region in the striatum where their connections are not targeted, but provide a massive and dense network (Moss & Bolam 2008, 2010). This is an order of magnitude greater than other types of dopamine neurons and several orders of magnitude greater than other neuron types in the brain. Single-cell filling by Matsuda and colleagues (2009) is consistent with this proposal. We propose that this massive axonal arbourwill put a high energetic demand on the neurons for normal cell biological functions and, more importantly, the generation and propagation of action potentials and the subsequent recovery of the membrane potential. Any stressor, e.g. oxidative stress, genetic mutations, mitochondrial poisons or dopamine neurotoxins, will have a preferential effect on these neurons because they are energetically 'on-the-edge' and the perturbations leading energy demand out-stripping supply, die-back and eventual cell death (Bolam&Pissadaki, 2012). In order to address this hypothesis we generated a biology-based computational model of the axons of individual dopamine neurons and examined the energetic impact imposed on SNc dopamine neurons by their extensive, unmyelinated axonal arbour (Pissadaki&Bolam, 2013). The main finding is that the energy demand associated with action potential conduction is related in a supra-linear manner to the axonal size and complexity. Thus those neurons that show a greater vulnerability have a disproportionately greater energy cost for action potential propagation. This higher energy demand, together with unique molecular and functional features, may underlie their selective vulnerability in Parkinson's disease (Hunn et al., 2015).

C-04

Seizures and epilepsy: from lab to clinics

Çiğdem Özkara

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An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain. Seizure is a symptom with an incidence of approximately 80/100,000 and lifetime prevalence is 9% per year. Epilepsy is a chronic condition of the brain characterized by an enduring propensity to generate epileptic seizures, and by the neurobiological, cognitive, psychological, and social consequences of this condition. Incidence: approximately 45/100,000 per year with point prevalence: 0.5–1% There are several etiological factors that gives rise to epilepsy which may differ according to the age including pre and post natal trauma, metabolic disorders, vascular malformations, tumor, malformations of cortical development, stroke, degenerative diseases, infections etc. Seizures are classified as focal or generalized where epilepsies are classified as focal, generalized and combined focal and generalized. Generalized epileptic seizures are conceptualized as originating at some point within, and rapidly engaging, bilaterally distributed networks. Such bilateral networks can include cortical and subcortical structures, but do not necessarily include the entire cortex. They can be tonic-clonic, absence, myoclonic, clonic, tonic and atonic types and usually have genetic etiology. Focal epileptic seizures are conceptualized as originating within networks limited to one hemisphere and could be genetically determined or related to structural lesions in majority of the cases. They may be related to several structural epileptogenic lesions as well as genetic etiology. From the basic science aspect there are animal models which may simulate different type of seizures. Although they are not completely perfect in terms of human seizures they contribute a lot to the knowledge of the pathogenesis. There are also models resembling acute seizures (ictogenesis) and chronic condition namely epilepsy (epileptogenesis). Attempts to unveil biomarkers which may predict the development of epilepsy with experimental and clinical studies which continue in parallel with studies ongoing to prevent these process. Reciprocal relationship between basic and clinical sciences hopefully will succeed to enlighten the unanswered questions of seizures and epilepsy to create a better life for all sufferers of this disease well known from antiquity.

C-05

The role of dopamine functions in animal models of depression

Daniela Schulz

Department of Psychology, Yeditepe University, Istanbul, Turkey

Depression research has largely focused on serotonergic mechanisms of depression. However, a large number of patients treated with serotonin-based drugs, including selective serotonin reuptake inhibitors (SSRIs) do not remit. The role of dopamine (DA) in the pathophysiology of depression is well established. The functions of DA fall in at least two categories, motivational and cognitive. These functions include effortful decision-making, voluntary behavior, instrumental learning and working memory. Here, I will examine if and how these functions are represented in animal models of depression. I will present data from two models, learned helplessness (LH) and extinction-induced despair (EID). The former views motivational and cognitive deficits as a consequence of the proposed etiology which is uncontrollable stress. The treatment of helplessness is effective when the DA system is recruited. The EID model, by contrast, views operant extinction, a form of instrumental learning, as a source of stress and despair. Operant extinction was shown to recruit the DA system, and extinction-induced immobility, a form of passive floating in water, was attenuated by desipramine, a noradrenergic-based tricyclic antidepressant, but was aggravated by fluoxetine, consistent with evidence that SSRIscan exacerbate motivational deficits. I conclude that a focus on DA functions in the generation of animal models of depressioncould advance our understanding of different subtypes of depression and also pave the way for new treatment approaches.

C-06

Observing the brain-on-task using functional optical brain imaging

Hasan Ayaz, Banu Onaral

Drexel University, School of Biomedical Engineering, Science&Health Systems, Philadelphia, PA, USA

The efficiency and safety of complex high precision human-machine systems such as in aerospace and robotic surgeryare closely related to the cognitive readiness, ability to manage workload and situational awareness of their operators. Subjective operator reports, physiological and behavioral measures are not sufficiently reliable to monitor cognitive overload that can lead to adverse outcomes. A key feature of the concept of mental workload – that reflects how hard the brain is working to meet task demands – is that it can be dissociated from behavioral performance data. Experienced human operators can maintain performance at required levels for a while through increased effort and motivation or strategy changes, even in the face of increased task challenge. Sustained task demands, however, eventually lead to performance decline unlessthe upward trend in mental workload can be used topredict subsequent performance breakdown. Consequently, it is important to assess mental workload independent of performance measures during training and operational missions. Neuroergonomic approaches based on measures of human brain hemodynamic activity can provide sensitive and reliable assessment of human mental workload in complex training and work environments. Functional near infrared spectroscopy (fNIRS) is a field-deployable non-invasive optical brain monitoring technology that provides a measure of cerebral hemodynamics within the prefrontal cortex in response to sensory, motor, or cognitive activation. This presentation will examine the relationship of the hemodynamic response in the prefrontal cortex to expertise development levels, mental workload stateand task performance in a variety of application areas.

C-07

Oxytocin: the sweet hormone?

Gareth Leng

Centre for Integrative Physiology, The University of Edinburgh, Edinburgh, Scotland

Mammalian neurons that produce oxytocin and vasopressin apparently evolved from an ancient cell type with both sensory and neurosecretory properties that probably linked reproductive functions to energy status and feeding behavior. Oxytocin in modern mammals is an autocrine/paracrine regulator of cell function, a systemic hormone, a neuromodulator released from axon terminals within the brain, and a 'neurohormone' that acts at receptors distant from its site of release. In the periphery oxytocin is involved in electrolyte homeostasis, gastric motility, glucose homeostasis, adipogenesis, and osteogenesis, and within the brain it is involved in food reward, food choice, and satiety. Oxytocin preferentially suppresses intake of sweet-tasting carbohydrates while improving glucose tolerance and supporting bone remodeling, making it an enticing translational target.

C-08

Theory and practice of reporting diffuse glial tumors in adults

Tarık Tihan

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Recent discoveries on the biology, behavior and therapy response of glial tumors in adults and children have prompted significant modifications in the diagnosis and management of these tumors. WHO 2016 provides a new blueprint, which is most likely to be modified and advanced in the future iterations of the classification scheme. Currently, the new classification recommends the use of an integrated diagnosis, which requires analysis of the key molecular characteristics of gliomas. This lecture is designed to provide practical and some theoretical knowledge on the use of the new classification scheme and to suggest how to incorporate these changes into daily surgical pathology practice. WHO classification of gliomas: In the past, one of the most critical distinction for gliomas was between diffuse/infiltrating and solid (non-infiltrating) tumors. It was clear from early years that diffuse gliomas had a tendency to progress and acquire more ominous phenotypes and eventually become malignant. The designation of WHO grades II through IV represented this biological tendency for progression and acquisition aggressive genotypic and phenotypic properties. For the crafters of the WHO 2016 classification, it was clear that adult and pediatric tumors (even though the had the same names) were very different genetically even though the morphological features did not imply such dramatic difference. It was also clear that certain molecular pathways among adults were quite distinct and separated themselves from tumors without such genetic alterations, even though the morphological features were quite similar. Thus, WHO 2016 came with the suggestion of an integrated diagnosis that includes histological typing, molecular characterization and designation of a particular grade that provides useful/actionable information to the neuro-oncologists. The quest for the comprehensive and definitive Integrated Diagnosis is by no means over, and there is much to be accomplished before such integrated diagnoses could be used without ambiguities, imprecision and controversies. However, the current scheme is a significant improvement over the old, and paves a path to the next stage, creating much hope for complete elimination of the uncertainties and ambiguities that is often inherent in the "art" of Pathology.

C-09

New insight into the pathogenesis and treatment of psychiatric disorders: inflammation

Feyza Arıcıoğlu

Department of Pharmacology and Psychopharmacology Research Unit, Faculty of Pharmacy, Marmara University, Istanbul, Turkey

At present, the pathophysiology of many psychiatric disorders is still not fully understood. Current treatment approaches that have been used in psychiatric disorders for many years and usually have a single mechanism-based effect are inadequate to control disease symptoms; modulation of dopamine in schizophrenia and monoaminesfor depression. Patients who do not respond to the current treatment options in the majority of psychiatric disorders, mainly diseases such as depression affecting 10% of the population and schizophrenia affecting 1% and resistance casesconstitute about 1/3 of them. Recent studies indicate that the increase in neu-

roinflammatory proinflammatory cytokines is a common pathology in the development of these diseases. Uncontrolled astrocyte / glia activity increases proinflammatory cytokines, causes changes in the levels of neurotransmitters and thus leads to a decrease in the production of neurotrophic factors. This form of neuroplasticity suppresses the ability of the synapses to change, to be structurally and functionally adaptive to certain situations. Treatment is expected to increase the production of neuronal plasticity and neurotrophic factors, including hippocampal neurons, synaptogenesis and neuronal maturation. Increasing evidence suggests that glutamatergic synapses/system plays an important role in the neuropathology and treatment of these diseases. On the other hand, a major problem is that patients have to wait 3–4 weeks for clinical effect. It has recently been shown that ketamine, which acts through glutamatergic N-methyl-D-aspartate receptors, can initiate antidepressant effects within hours and that its mechanism has a role in increasing the production of mTOR pathway and neurotrophic factors. In the treatment of schizophrenia, the effects of many antiinflammatory molecules such as cyclooxygenase-2 inhibitors are discussed. Current research suggests that the recommended medicines for use as adjunctive therapies to existing treatments, as well as future drug targets, should be those that act through the glutamatergic system, enhance neurotrophic support and / or antiinflammatory properties.

Keywords: depression, schizophrenia, cytokine, inflammation

C-10

Understanding the value of alpha-synuclein as a biomarker in Parkinson's disease dementia: a story from animal models to human biospecimens

Deniz Kırık

Department of Experimental Medical Science, Lund University, Lund, Sweden

It is almost exactly 20 years ago that the first evidence of a genetic mutation in the alpha-synuclein gene causing Parkinson's disease (PD) was identified. It has rapidly become clear that the relationship between alpha-synuclein and PD went well beyond the rare familiar forms of the disease, as the protein was found in the intracellular aggregates in idiopathic cases. Since then, alpha-synuclein has become the focus of attention in research aimed at better understanding the disease pathogenesis and development of new therapeutic interventions to achieve disease modification. In particular, development of tools and assays to measure alpha-synuclein levels in biospecimens has been recognized as a key step. This talk will take a 10-year perspective on how work in our laboratory took us from questions we had at hand that related to the involvement of the cholinergic system in the cognitive dysfunction in PD through studies in human post-mortem brains in one axis, and development of new assays and their use not only in animal experiments, where they were first intended but also in patient samples. Taking advantage of this journey, I will aim to illustrate how experimental studies inform clinical work and how needs in understanding the disease in humans triggers the need for further experimental studies making the two parts of the story inseparable from each other.

C-11

Contemporary research and issues on cognitive processes involved in visual word recognition: the case of Turkish orthography

İlhan Raman

Middlesex University, Department of Psychology, London, UK

Although much research has been conducted over the past 60 years with the aim of understanding typical and atypical processes involved in visual word recognition, the focus has nevertheless been Anglo centric. It was not until 1990's that the focus shifted to other orthographies when questions started to be raised about the applicability of theoretical frameworks firmly rooted in English to other writing systems. This is because orthographies differ widely from each other on several accounts. One such variation in alphabetic orthographies is orthographic transparency which, simply put, is the ease with which one can derive phonology from orthography. In this respect ,Turkish orthography is said to be transparent because the relationship between orthography to phonology is extremely predictable. The aim of this talk is to briefly review the literature before turning the attention to research on Turkish orthography which represents a very interesting and unique orthographic medium to test the claims of several models. Findings will be presented from intact and neuropsychological studies in order to establish the current status of research on Turkish orthography and to identify future research directions from a multidisciplinary perspective.

C-12

Promoting brain remodeling and plasticity in ischemic stroke: underlying mechanisms, potential pitfalls and clinical translation strategies

Dirk Hermann

Department of Neurology, Chair of Vascular Neurology, University Hospital Essen, Dementia and Ageing Research/Director NeuroScienceLab, Essen, Germany

Promotion of neuronal plasticity and brain remodeling has recently turned out highly promising in experimental stroke models. By promoting neuronal plasticity, neurological recovery may be induced, which raises hopes for the large number of stroke patients exhibiting long-lasting functional deficits despite successful recanalization therapies. With respect to future clinical application, a number of challenges exist that need to be overcome to ensure that plasticity-promoting therapies do not get lost in the translation from bench to bedside. Based on own investigator-driven experiments, our laboratory presently actively supports controlled clinical studies in the stroke field with the aim of establishing a clinically applicable therapy that promotes recovery once acute sroke injury has occurred. This talk will present an overview on conceptional works in this field, defining scientific needs and challenges, and outlining examples of successful translation into clinical trials.

Symposiums

(S-01 — S-8)

Symposium 1
Brain bank, neurodegeneration and biomarkers

S1-1

Biomarkers of two main types for Alzheimer's disease

Engin Eker

President, The Turkish Alzheimer Society, Istanbul, Turkey

Alzheimer's disease (AD) is a progressive neurodegenerative disorder that slowly destroys learning, memory and thinking skills, resulting in behavioral anormalities. It is estimated that nearly 36–38 million are affected globally with numbers reaching approximately 120 million by 2050. AD can be definitively diagnosed at autopsy since its manifestations of senile plaques and neurofibrillary tangles throughout the brain cannot yet be fully captured with current imaging technologies. Current AD therapeutics have been suboptimal. Besides identifying some biomarkers that distinguish AD from controls, there has been a recent drive to identify better biomarkers that can predict the rates of cognitive decline and neocortical amyloid burden in those who exhibit preclinical, prodromal, or clinical AD. This presentation covers biomarkers of two main types: cerebrospinal fluid-derived, and blood-derived biomarkers. Recent data and researchs have to develop in order to find even better biomarkers for AD that are more predictive.

S1-2

Definitive diagnosis of dementia and brain banking

Derya Kaya, Ahmet Turan Işık

Center for Aging Brain and Dementia, Department of Geriatric Medicine, Faculty of Medicine, Dokuz Eylül University, Izmir, Turkey

Diagnosis of possible or probable diseases with dementia is made with clinical, neuropsychological, and radiological evaluations. For a definitive diagnosis, a histopathological confirmation by autopsy is required.Thus, brain banking is indispensable for making definitive diagnosis of human central nervous system. As the population ages, it becomes more important for uncovering the secrets of the brain diseases with dementia. Brain banks collect post-mortem human brains not only to make definitive diagnosis, but also to investigate susceptibility to disease, explore the mechanisms of patogenesis particularly in neurodegenerative diseases, and to perform research.As the human brain is so complex and there are so many open questions in neuroscience, brain bankscontinue to remain at the heart of brain research.Data from the tissues can lead to make definitive diagnosis, thus the family and/or public-health policycan be informed, treatment strategies can be refined, and new drugs can be developed.The first Brain Tissue Resource Centre of Turkey has been established legally in the Center for Brain Aging and Dementia, in the Department of Geriatric Medicine, Dokuz Eylul University, Izmir in 2015. It is based on scientific, ethical, and legal standards. We aim to become a reference center which collects the human brain specimens for brain research, so that, we could have a chance for increasing the help for our better understanding of the pathogenesis of the diseases affecting the brain tissue, and for reducing the personal, familial, social, and economic burdens of them.

S1-3

Can neurodegeneration be mimiced?

Turgay Çelik

Department of Pharmacolog, Faculty of Pharmacy, Yeditepe University, Istanbul, Turkey

The prevalence of neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD) is expected to rise in the next years. These age-related neurodegenerationsare largely human-specific neurodegenerative diseases. Despite to aspects similar to those of human brain aging can be observed in aged primates, these animals do not readily develop the full neuropathological and clinical phenotypes observed in humans. Although none ofthe existing in vivo and in vitro models fully reproduces the complete spectrum of this insidious human disease, critical aspects of this pathology and disease processes can be experimentallyrecapitulated. Genetic and non- genetic animal models have helped understanding of the underlying mechanisms of disease and have proven to be invaluable in the preclinical evaluation of potential therapeutic interventions. Recently, non-mammalian species, such as *C. elegans*, *D. melanogaster* and zebra fish, may be useful for the dissection of the basic disease mechanisms and the screening of compounds targeting specific mechanisms involved in ND.

Symposium 2
Basal ganglia and deep brain stimulation

S2-1

Computational physiology of the basal ganglia and deep brain stimulation: closing the loop between health, disease and treatment

Hagai Bergman

Department of Medical Neurobiology (Physiology), Institute of medical Research Israel-Canada (IMRIC), Totonro, ON, Canada; The Edmond and Lily Safra Center for Brain Sciences (ELSC), The Hebrew University, Jerusalem, Israel and Department of Neurosurgery, Hadassah Medical Center, Jerusalem, Israel

The basal ganglia (BG) use actor/critic architecture that enables multi-objective optimization of behavioural policy. The BG modulators (critics, e.g., dopamine) encode the mismatch between prediction and reality; whereas the BG main axis (actor) provides the connection between state and action. The striatum and the subthalamic nucleus (STN) constitute the input stage of the BG main axis (actor) network and together innervate BG downstream structures. Our recent studies indicate that subthalamic rather than striatal activity shapes BG downstream activity. This STN modulation of BG downstream activity occurs both before (in health) and after intoxication by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) which leads to striatal dopamine depletion and parkinsonian clinical symptoms. Thus, the divergent excitatory STN projections have a critical role in shaping BG output activity. This explains why the STN (and not the striatum) is such an effective site for deep brain stimulation (DBS) in Parkinson's disease and other BG disorders. Finally, today DBS systems are manually adjusted every 1–3 months. However, the abnormal beta synchronized oscillations in the STN are episodic, and long (>4 seconds) episodes can be detected only after MPTP treatment. We therefore suggest that we can better treat BG disorders by closed-loop adaptive DBS that would inactivate the basal ganglia only when they "misbehave", i.e., following detection of STN long beta events.

S2-2

Deep brain stimulation and target selection in movement disorders

Boran Urfalı

Department of Neurosurgery, Faculty of Medicine, Mustafa Kemal University, Hatay, Turkey

Deep brain stimulation (DBS) therapy for movement disorders depends on a series of interrelated procedures that include precise lead placement and proficient electrode programming. The first and most important step toward consistent DBS outcomes remains careful patient selection, because more than %30 of DBS failures can generally related to the incorrect initial diagnosis or inappropriate indications for surgery. Patient selection for DBS surgery must be based on selection of only appropriate candidates. First step of this selection is neurological evaluation and this must be focused on establishing the correct diagnosis and being sure that all the medical therapy options have been tried. In recent years, DBS is getting more important for treatment of movement disorders without any ablative surgical procedure. There is some approved indications for DBS (by FDA). These indications are Parkinson's disease (PD), essential tremor (ET) and primary dystonia. Tics, choreas, other tremor forms and etc. are also targeting with DBS but these indications are considered off-label targetings. After neurological evaluation (diagnosis and medical management) patient must be referred to the neurosurgeon for neurosurgical evaluation. One of the components of neurosurgical evaluation is surgical options which includes available targets and surgical methodologies, risks, goals etc. Neurocognitive and psychiatric evaluation must be done by neuropsychologists and psychiatrists, because of importance of cognitive assessment and psychiatric screening to decide which nucleus must be targeted by the neurosurgeon. In movement disorders, related to diagnosis there is different target selections. These target selections are subthalamic nucleus (STN), globus pallidus interna (GPi), ventral intermediate (VIM) nucleus of the thalamus, caudal zona incerta (cZI), pedinculopontine nucleus (PPN) and the other thalamic nucleuses. For deciding best surgical target, patients must be evaluate carefully and for getting better postoperative and clinical results micro electrode recording systems must be use preoperatively.

S2-3

Functional physio-anatomical network of basal nuclei: past, present and future

Ilkan Tatar

Department of Anatomy, Faculty of Medicine, Hacettepe University, Ankara, Turkey

The basal nuclei (BN) describes as the feed-forward part of a closed loop connecting all cortical areas sequentially through the BN direct and indirect pathways back to the motor cortex. The motor cortex projects to the spinal level through the corticospinal pathway and controls muscle activation and movements. In the classical D1/D2 direct/indirect model of the BN, "direct pathway" is a monosynaptic GABAergic inhibitory projection from the striatum to the GPi/SNr, whereas the "indirect pathway" projection is polysynaptic and disinhibitory through the GPe and the glutamatergic (excitatory) STN. However, recent anatomical, physiological, and theoretical studies have revealed that the BN connectivity is more complex than the simple connectivity depicted by the D1/D2 direct/indirect model. This model is also having shortages in explaining the dynamic patterns of BN activity and Parkinson's disease and ignores the emerging roles of the BN in reinforcement learning and behavioral adaptions to the changing environment. More modern computational models of the BN treat the BN as an actor/ critic reinforcement learning network. The main axis or the actor part puts into action the behavioral policy or the mapping between states and actions (behavioral policy), and the critic calculates the mismatch between predictions and the actual state (prediction error). The reinforcement actor/critic model of the BN has revolutionized current understanding of physiological mechanisms of model- free (procedural, implicit) learning and may provide insights into certain BN-related disorders such as akinesia and levodopa-induced dyskinesia. The computational goal of the BN might be optimizing the trade-off between the orthogonal goals of maximizing future cumulative gain and minimizing the behavioral (information) cost (i.e., multi- rather than single-objective optimization). General prediction of the next generation of DBS devices will exploit BN actor/critic multi- objective optimization algorithms and will provide even better therapy for human patients.

Symposium 3
Growth factors in neurological and psychiatric disorders

S3-1

Growth factors in neurological and psychiatric disorders

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Growth Factors (GF) are a family of molecules that play vital roles in cell survival, synaptogenesis and maintenance of the newly formed synapses in both developing andmature central nervous system (CNS). GFs are protective againstacute and chronic neurodegenerative diseases; GFs administered after cerebral ischemia decrease the infarct volume. Similarly, increases in FGF2 levels reduce the effect of the neurotoxins inneurodegenerative diseasemodels and promote neuronal survival. Thus, GFs appear to be promising drug targets for treatment of ischemic stroke as well asseveral neurodegenerative diseases. Unfortunately, these large peptides cannot cross the blood-brain-barrier when administered systemically.In order to benefit from their neuroprotective action in clinical practice, they should be administered non-invasively and should be effective mainly in the brain without inducing peripheral side effects. We have recently developedbrain-targeted FGF2-loaded nanoparticles and showed that their systemic administration is protective in stroke. Neuroinflammation has a vital role in the pathophysiology of the CNS diseases such as cerebral ischemia, Alzheimer's disease, Parkinson's disease and depression. Proinflammatorycytokines protect the neuronal integrity under physiological conditions although they may become destructive under pathological conditions. Inflammasome, which is a cytosolic multiprotein complex and a part of the natural immune response are activated by inflammatory signals. The effect of the proinflammatory cytokines that emerge after inflammasome activation on neurogenesis depends on their concentration, the activated cell type and the co-existing factors in the environment. In addition to its various effects, BDNF pathway may also modify the inflammation as it has been shown that BDNF expression decreases upon inflammation. GFs also play a role in the pathogenesis of mental disorders. The first finding that suggestsa role for FGF2 in depression etiopathogenesiscame from postmortem investigations, which showed a decrease in the FGF2 expression in the dorsolateral prefrontal cortex and anterior cingulate cortex of the patients who had major depression. In animal models, the intracerebroventricular administration of FGF2 had antidepressant and anxiolytic effects, and a decrease in the FGF2 expression in the hippocampus has shown to be anxiolytic. We have recently observed that FGF-AS, which is synthesized in the opposite chain of the FGF2 and is involved in the regulation of the FGF2 expression, has also a role in the pathogenesis of affective disorders. In this symposium, the relationship between the GFs and the neuroprotection, neuroinflammationas well as depression will be reviewedbased on the experimental data from our laboratory.

S3-2

Growth factors in neuroprotection: importance of administration route and timing

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Basic fibroblast growth factor (bFGF), also known as FGF2 or FGF-β is a member of the FGF superfamily. bFGF promotes survival of neurons, stimulates axonal outgrowth, synaptogenesis, and stimulates the proliferation of neural progenitor cells in brain. As bFGF couldsuppress cell death by acting at several points on death pathways and, additionally, promote regeneration, it is found to have neuroprotective effects in acute experimental stroke. Although bFGFis a promising agent for treatment of stroke as well as other neurodegenerative diseases this peptide is relatively large (17.2kDa) and cannot penetrate the brain tissue when systemically administrated. bFGF can reduce infarct size when given intracerebroventricularly to animals, but as this method is invasive, translation of theseexperimental findings to clinic is hampered.We recently demonstrated that bFGFcould successfully be encapsulated into a chitosan nanomedicine formulation, rapidly transported across the blood–brain barrier by receptor-mediated transcytosis, and efficiently provide neuroprotection in transient focal cerebral ischemia.This could be a therapeutic approach in stroke. Vascular endothelial growth factor (VEGF) is a 34 to 48kDa homodimeric glycoprotein. It has been shown to have a role in atherosclerosis, arteriogenesis, cerebral edema formation, neuroprotection, neurogenesis, angiogenesis, postischemic brain and vessel repair in experimental stroke. VEGF promotes recovery after strokeby the delayed administration, but also has been shown to provide neuroprotection independently of its angiogenic action. We determined that although, early intravenous injection of VEGF increases blood brain barrier leakage, hemorrhagic transformation and infarct volume; its early intracerebroventricular administration is neuroprotective in acute experimental stroke. This is in accordance with its neuroprotective effect observed with topical application. The potential efficacy of several growth factors for stroke treatment and neuroprotection depends on the routes and time of administrationfor achieving a desirable result.

S3-3

The role of FGF2 and FGF-AS in affective disorders

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The role of growth factors in the pathophysiology of affective disorders has been studied for long. The most replicated finding in this field is that stress decreases brain derived neurotropic factor (BDNF) expression in depression-related brain areas, specifically in hippocampus and prefrontal cortex, while while antidepressants show opposite effect on BDNF. Similar

findings were also reported for other growth factors. The relationship of fibroblast growth factor-2 (FGF2) with depression and anxiety disordershas been demonstrated by both postmortem brain studies and animal studies. FGF2 expression was shown to decrease after chronic stress exposure and increase after antidepressant administration in animal studies. Moreover both acute and chronic FGF2 administration into the lateral ventricule has antidepressant actions in rats. Interestingly acute and chronic injections of FGF2 have opposite effects on anxiety-like behavior: Acute FGF2 administration increases anxiety-like behavior, chronic FGF2 administration, on the other hand, decreases anxiety-like behavior.We showed that decreasing FGF2 expression in the hippocampus increases anxiety-like behavior in rats. Then we studied the role of the natural antisense transcript (NAT) of FGF2, FGF-antisense (FGF-AS),in the pathophysiology of affective disorders.NATs are long RNA molecules that are transcribed from the opposite strand of protein-coding sense transcripts. Once thought to be a rare phenomenon, NATs are now recognized as a widespread feature of mammalian genome.There are findings in the literature that suggest a role of FGF-AS in the regulation and stability of FGF2. We therefore investigated the role of FGF-AS in the neurobiology of affective disorders. We showed that FGF-AS levels changed with stress and manipulations of FGF-AS levels affected anxiety- and depression-like behaviors in rats. Our findings showed that increasing FGF-AS expression increased anxiety- and depression-like behaviors, while decreasing FGF-AS expression in the hippocampus had anxiolytic and antidepressant effects.

S3-4

Neurotrophic factors and para-neuroinflammation

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Neuroinflammation plays a role in the pathophysiology of acute and chronic central nervous system diseases such as cerebral ischemia, multiple sclerosis, Alzheimer's disease, Parkinson's disease and major depression. Neuroinflammation in general is an adaptive reaction that is initiated by noxious stimuli and conditions, such as infection and tissue injury. However, tissue stress or homeostasis change may also cause inflammation. Depending on the trigger, the inflammatory response has a different physiological purpose and pathological consequences. The term para-neuroinflammation refers a tissue adaptive response to noxious stress or dysfunction and has characteristics that are intermediate between basal and inflammatory states (Medzhitov, 2008). The physiological purpose of para-neuroinflammation is to restore neuronal tissue functionality and homeostasis.Cortical spreading depression (CSD), one of these triggers, causes ionic, metabolic and vascular perturbations, nearly all of which are resolved within a few minutes. CSD, putative cause of migraine aura and headache, leads to neuronal megachannel opening and caspase-1 activation. This inflammasome activation triggers high-mobility group box 1 (HMGB-1) release from neurons and NF-κB activation in astrocytes thereby reports homeostasis and functional change to the nervous system. This neuro-parainflammation induced by CSD leads to increase in the expression of neurotrophic factors like brain-derived neurotrophic factor (BDNF), nerve growth factor, basic fibroblast growth factor in neurons and astrocytes.BDNF is an important neurotrophic factor that plays a role in neurogenesis and neuroplasticity.In the literature, it was shown that BDNF expression increases from early time points after CSD. In addition, cerebral ischemia experiments performed after CSD revealed that increased expression of BDNF provide neuroprotection. Neurotrophic factors may also play a role in the migraine headache. In this section I will discuss the relationship between neurotrophic factors, especially BDNF, and para-neuroinflammation in regard to our data and the literature.

Symposium 4
In silico drug design

S4-1

In silico design of novel and selective neuronal nitric oxide synthase (nNOS) inhibitors

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Three closely related isoforms of nitric oxide synthases (NOS) catalyze the nitric oxide (NO) synthesis through oxidation of L-arginine to L-citrulline. Mammalian cells have three different isoforms. These three NOS isoforms takes parts in different tissues for various physiological and pathological processes. Neuronal NOS (nNOS) produce NO in central and peripheral nervous system, endothelial NOS (eNOS) plays role in endothelial cells and NO in macrophage cells is produced by inducible NOS (iNOS). The excessive production of NO, especially by nNOS (in brain) is implicated in various disease states such as neurodegeneration and oxidative stress. This may contribute the occurrence of certain disease such as Alzheimer's, and Parkinson's diseases. The other two isoforms, iNOS and eNOS, are very crucial and not inhibited during the inhibition of nNOS. In endothelial cells, eNOS relaxes smooth muscle causing to decrease blood pressure and in macrophage cells eNOSgenerates NO as an immune defence system to destroy microorganisms and pathogens. In order to control the excessive production of NO in the brain and treat neurodegeneration it is important to inhibit nNOS selectively. Three isozymes show extraordinarily structure similarities hindering the selective inhibitor design. In the literature, there are many outstanding studies, however there has not being developed any drug which accomplished the required potency and selectivity. This is a very challenging task. In this present work, virtual screening techniques *in silico* environment were applied to design selective and potent nNOS inhibitors. Molecular modeling studies were done using already known crystal structures

of all three isoforms. The best candidates showing high inhibition constantsand selectivity towards nNOS over eNOS and iNOS isoforms were determined.

S4-2

Investigation of allosteric coupling in human β2-adrenergic receptor (β2-AR) in the presence of intracellular loop 3

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This study investigates the allosteric coupling that exists between the intra- and extracellular parts of human β2-AR, in the presence of intracellular loop 3 (ICL3), which is missing in all crystallographic experiments and most of the simulation studies. Our 1 μs long MD run has revealed a transition to an alternative inactive state of the receptor, in which ICL3 packed under G protein's binding cavity and completely blocked its accessibility to G protein. Simultaneously, an outward tilt of transmembrane helix 5 (TM5) caused an expansion of the extracellular ligand-binding site. Independent runs with a total duration of 4 μs were carried out to further investigate this inactive state with packed ICL3 and the allosteric coupling event. In all three independent unrestrained runs, ICL3 preserved its initially packed conformation during 500 ns long simulation, suggesting an inhibition of the receptor's activity. Specific bond restraints were later imposed between some key residues at the ligand-binding site, which have been experimentally determined to interact with the ligand. Restraining the binding site region to an open state facilitated ICL3 closure, whereas a relatively constrained binding site hindered ICL3 packing. However, the reverse operation, i.e. opening of the packed ICL3, could not be realized by restraining the binding site region to a closed state. Thus, any attempt failed to free ICL3 from its locked state. Overall, our simulations indicated that starting with very inactive states, the receptor stayed almost irreversibly inhibited, which in turn decreased the overall mobility of the receptor. Bond restraints, which represented the geometric restrictions caused by ligands of various sizes when bound at the ligand-binding site, induced the expected conformational changes in TM5, TM6 and consequently, ICL3. Still, once ICL3 was packed, the allosteric coupling became ineffective due to strong hydrogen bonds connecting ICL3 to receptor's core.

S4-3

Homology modeling of human dopamine transporter: understanding the difference between inhibitors and substrates

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Parkinson'sdisease (PD) is characterized by the loss of dopamine-generating neurons in the substantia nigra (SN) and corpus striatum (CS). Most drugs currently on the market for PD target the symptoms rather than exert neuroprotection of the dopaminergic neuron. Targeting new drugs to dopaminergic neurons by specific uptake through the dopamine transporter (DAT) is a viable strategy for neuroprotection. By means of homology modeling, molecular docking and molecular dynamics (MD) simulations, we have generated 3D structure models of hDAT in complex with amphetamine, cocaine and modafinil. The results reveal differences in binding kinetics of these compounds to the DAT during open and closed conformations, which may be crucial for future drug design.

Symposium 5

Approach to ALS and spinal cord injury patients and improvement of their life quality

S5-1

Diagnosis and new treatment options

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Amyotrophic lateral sclerosis (ALS), motor neuron disease (MND) is a progressive, lethal disease which is characterised by degeneration of motor neurons at the primary motor cortex, brainstem and medulla spinalis. Although clinical progression and prognosis are determined by involvement of the corticospinal tracts, brainstem and spinal motor neurons, respiratory muscle involvement due to degeneration of the spinal motor neurons is the main cause of death. In many European and North American studies, incidence is reported as 1–3/100,000, prevalence is reported as 4–6/100,000 per year. It is predicted that there are 90–100,000 ALS patients all over the world and 3500–5000 in Turkey. Although the cause is not known, in some geographical regions such as Guam Island in East Pacific, Kii Peninsula in Japan and West Papua in New Guinea have higher incidence and prevelance when compared to other regions of the world. Incidence of ALS increases with age; incidence is less before 40 years of age and highest around the age 75. There is a male predominance. Bulber ALS is a disease of elderly and especially more prevalent in women. In many studies, mean age of onset of the sporadic ALS varies between 60 and 65 years. Mean age of onset of the familial one is a decade earlier. Its onset can very seldomly be at 2nd and 3rd decades. Male to female ratio is 1.5/1 in sporadic ALS, whereas it is 1/1 in familial ALS. Familial cases constitute 10% of all patients.The first gene defined in Familial ALS patients is the superoxide dismutase 1 (SOD1)gene located in chromosome 21.SOD1 is responsible from nearly 5% of all familial cases. There seems to be no significant differences regarding clinical presentation and neuropathic signs between familial and sporadic cases. It is anticipated that motor neuron degeneration in both familial and sporadic ALS follows an associated molecular mechanism. Transgenic animal models origi-

nating from genetic studies will contribute to the understanding of the disease ethiopathogenesis and to the formation of treatment perspectives.

S5-2

Acute spinal cord injury and management

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Acute spinal cord injury (ASCI) was defined as "An ailment not to be treated" or "incurable disease" for the first time in Egypt papirus in 17BC by Edwin-Smith. This belief started to change with the facts that Santiago R.y Cajal discovered growth cone and spontaneous regenerative capacity of the central nervous system. Then, in 1911, Alfred R. Allen, at University of Pennsylvania, formed the first animal model of spinal cord injury,in the modern sense, by dropping some weight on the spinal cord of a dog. In this contusion (hitting) type of injury model, Allen also put forward the secondary injury concept. Later, in the 1970s, Tator and Rivlin developed compression model of injury with a clip which was created by themselves. During this period, studies on both brain injury and spinal cord increased rapidly. For example, in 1972, Wylie and Kerr have described apoptosis together. In 1986, Olney and Rothman showed the role of glutamate neurotoxicity in ischemic injury for the first time and the description of never-ending pathways associated with each other started. Thus, a better understanding of the pathophysiology of spinal cord injury started. In recent years, various pharmacological agents have been tested for patients with ASCI (methylprednisolone sodium succinate, trilazad mesylate, GM-1 ganglioside, thyrotropin-releasing factor, gasiklidin, naloxone, and nimodipine), in large, prospective, randomized, controlled clinical trials and contrast to their success in the laboratory have marked no neurological benefit in clinics. However, positive results of the study of NASCIS II (North American Spinal Cord Injury Study) with methylprednisolone sodium succinate (MPSS) have been found, and that has become a ground for new studies in patients with ASCI, but later methylprednisolone have encountered serious criticisms and many centers have abandoned it. While the first half of the 1980s and 1990s passed with the neuroprotective studies and understanding of pathophysiology, subsequent studies have been mainly regeneration studies. Today, the most important problem is that the trials working good in animals do not show the same effect in humans. Many theories and strategies have been proposed to overcome this.

S5-3

Principles of rehabilitation in amyotrophic lateral sclerosis and spinal cord injury

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Amyotrophic lateral sclerosis (ALS) is characterized by progressive muscle weakness, atrophy and fasciculations resulting from the progressive loss of motor neurons. Due to progressive muscle degeneration, the risk of exercise-related muscle damage is high. For this reason, exercise should be prescribed with caution. Main objectives are to provide energy conservation, provide mobility as much as possible, protect communication with alternative and augmentative communication methods in patients who can not speak or write and provide respiratory support. Spinal cord injury (SCI) is the damage of the neural elements of the spinal cord, which is manifested by motor and sensory loss, autonomic and bladder and bowel problems. In the early period of SCI, the aim is to prevent pressure ulcers and contracture formation and minimize the respiratory problems. Medical problems and complications during SCI rehabilitation process include autonomic dysreflexia, orthostatic hypotension, pulmonary complications, deep vein thrombosis and pulmonary embolism, bladder and bowel dysfunction, UTI, sexual dysfunction, hypercalciuria, hypercalcemia, osteoporosis, pain and pressure ulcers. The main goals of rehabilitation in both ALS and SCIs are to maximize physical independence, prevent secondary complications and increase quality of life. Rehabilitation is an interdisciplinary team work and the team leader is the psychiatrist. Other members of the team typically include the person with SCI, family members, physical therapists, occupational therapists, nurses, expert of social services, orthotist, dieticians, psychologists. Other consultant physicians, respiratory therapists, speech and language therapists can also be members of the team depending on the specific injury and rehabilitation goals. Treatment targets in both ALS and SCIs must be established according to International Classification of Functioning, Disability and Health (ICF) model of the WHO based on a framework that includes individual and environmental factors, aiming at revealing a more holistic understanding of health rather than focusing on the patient's health status alone. This model consists of body structure and functions, activity and participation, and environmental and personal factors components. The main factor that leads to inadequate performance and mobility in core tasks in life is changes in body structure and function. However,the main aim of treatments for these changes should be to increase activity and participation.

S5-4

Nutrition management of patients with amyotrophic lateral sclerosis (ALS)

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Amyotrophic lateral sclerosis (ALS) is a neurodegenerative progressive disease of upper and lower motor neurons of the brain and spinal cord which is caused by various factors (genetics, viral infections, glutamate excitotoxicity, autoimmune reac-

tions, heavy metal intoxications such as lead, mercury and aluminium intoxications). Upper and lower motor neuron involvement determines the clinical presentationof the disease which is characterised by stiffness in the extremities, hyperreflexia, emotional lability, asymmetrical weakness of arms and legs, wasting of the muscles, cramps, twitching of the muscles, tiredness, difficulties in speech and swallowing. These complications in combination with weight loss prevent adequatebalanced nutrition and cause protein-energy malnutrition. Nutritional status of the patient should be determined, medical nutrition therapy should be incoorporated. Anthropometric measurements, blood biochemical tests, bowel movements, nutritional habits and physical activity status are evaluated. Energy need, macro and micro diet components, pulp and liquid amounts should be established. Oral intake of the patient should be planned initially, however with the progression of the disease, patients can not supply adequate energy and diet componentsdue to longer eating durations, fatique during chewing and early satiety. Moreover deficiency in swallowing reflex causes aspiration. To overcome aspiration, thickeners should be added to fluids. If the oral intake is inadequate causing weight loss upto 10%, enteral or parenteral nutrition should be started. Parenteral nutrition should not be used for longer periods and replaced by enteral nutrition. The important factors in enteral nutriton are the route of nutrition and type of the product. Percutaenous Endoscopic Gastrostomy (PEG) is recommended in patients who are on continuous tube feeding. The choice of the type of enteral product is consistent with the tolerance of patients and presence of concomitant diseases. Patients who undergo medical nutrition therapy should be followed and appropriate corrections should be managed in any change in the patient's status. Medical nutrition therapy is one of the most important factors which effects the prognosis of the ALS patients. If the nutrition therapy is managed timely and properly, survival is prolonged and quality of life gets better.

S5-5

Effect of diaphragm pacing on quality of life of ALS and spinal cord injury patients, who to pace and when to pace?

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Respiratory insufficiency in amyotrophic lateral sclerosis (ALS) patients is treated by non-invasive mechanical ventilation. In select ALS patients we aim to treat respiratory insufficiency using diaphragm pace implantation. Between April 2014 and December 2016, we prospectively collected data of 10 patients (six female, median age 59) who underwent diaphragm pacing and 15 patients who did not undergo pacing. In pacing group, one patient had trachestomy, 4 patients were using BIPAP. In the non-pacing group, one had tracheostomy, 2 were using BIPAP. Patients who were diagnosed to have chronic hypoventilation (FVC<50%, $pCO_2 \geq 45$ mmHg) preoperatively using lung function tests and blood gas analysis, were checked for diaphragm stimulatability by diaphragm EMG and diaphragm ultrasound. Diaphragm electrodes were inserted laparoscopically. The pacing intervals were set according to the need. Follow-up of the patients were performed using diaphragm ultrasound. Postoperative morbidity was 10% (n=1, pneumothorax). 30-day mortality was 0%, 90-day mortality was 10% (n=1). In acute period, patients had better sleep cycles. The patient with tracheostomy was off the ventilator at the 3rd month. During the follow-up, two underwent tracheostomy, one due to excessive salivation, one due to respiratory insufficiency. None of the patients had pneumonia in the first 12 months. Average follow-up of patients was 20.4 months (range, 3-32 months) after implantation. During the follow-up, one patient died due to a cardiac arrest (3rd month) and one died following a pneumonia attack (15th month). Diaphragmatic thickness calculated by diaphragm ultrasound showed an average of 23% increase in the thickness at the 3rd month postimplant. While the survival was 80% in the pacing group, survival was 0% in the non-pacing group. A 18-year old male who suffered C4-5 spinal cord injury was evaluated for pacing following two septic attacks at the ICU. Diaphragm EMG showed stimulatable diaphragms, pacing was done through laparoscopy. Patient was off the ventilator at the end of 3rd month. Pacing was shut down at 9th month. He is scheduled for tracheostomy closure. Survival is increased in ALS patients with chronic hypoventilation following pacing. In spinal cord injury patients pacing supports ventilation by recruiting diaphragm physilogy and ventilation functions if the phrenic nerve functions were intact.

Symposium 6
High technology methods in neuroscience research

S6-1

In vivo micro sensor technology in the diagnosis and treatment of brain diseases

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Strong imbalances in neurotransmitter levels have been associated with many mental disorders. These include Parkinson's disease, depression, anxiety, memory loss, hepatic encephalopathy and addictions. In most cases, it was impossible to measure the neurotransmitter levels in the brain at any moment in seconds, due to the chemical transmission and the nature of the structures. Minimally invasive techniques for monitoring brain chemistry in vivo provided better understanding of neuropharmacology of CNS disorders. For monitoring and sampling brain chemistry; voltammetric electrodes, microdialysis and related analytical techniques had been used. Microdialysis, compared to voltammetry, offers lower temporal and spatial resolution. Glutamate is a principle neurotrans-

mitter. But it also has neurotoxic effects. In our studies, we used voltammetric electrodes for detecting glutamate activities in synaptic area. We did silicon based multisite biocompatible microelectrode fabrication for detection neurotransmitter concentrations. These electrodes were implanted with stereotaxy-to different brain areas for each experimental disease models. We used glutamate transporter activator or inhibitor drugs to change glutamate levels in synaptic area in different animal models. Also our understanding on drugs and their action mechanisms are increasing by this method. We are producing new biocompatable barriers for selectivity and we are obtaining longer duration times for enzymes on electrodes in animals. We can detect neurotransmitters by our own wireless medical device. Our studies are supported by TUBITAK (The Scientific and Technological Research Council of Turkey) projects # 107S067 and 113S083

S6-2

Acute impact of VMH activity on appetite

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Ventromedial hypothalamus has long been implicated in energy homeostasis and glucose regulation. Despite the wealth of research on VMH function in energy balance, direct link between acute VMH activity manipulation and appetite has not been demonstrated. Here we used chemogenetic tools SF1-Cre mice to selectively alter VMH activity in behaving mice. Our results suggest that while acute VMH activation rapidly inhibits food intake, VMH inhibition is not sufficient to drive food intake.

S6-3

Dissecting myelination and demyelination at high resolution

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Myelin sheet enables neurons to transmit information efficiently and it is critical for neuronal function and survival. Improper development of or damage to myelin leading to disruption of electrical impulse conductivity, atrophy of neurons, and permanent functional deficitsand is suggested to underlie many neurological disorders, such as multiple sclerosis (MS). We focus on the investigation of myelin and myelin formation to explore the dynamics of myelination, cellular mechanisms that lead to demyelination and the cause-and-effect-relationships between neurodegenerative diseases and perturbations to myelin. Besides unraveling the basic biology of myelin, we are particularly interested in translating this knowledge to the development of therapeutic approaches for demyelinating diseases and to functional recovery of the nervous system following injury. In order to accomplish these goals, weinitiated a big data approach to construct anatlas of protein-protein interactions between oligodendrocytes and axons and immune cells. These maps, or myelin interactomes, point toward pathways involved in myelin formation and in the immune attack to myelin. Functions of the newly identified pathways will be explored using in vitro assays developed by us. The in vitro assays are also suitable for testing candidate drugs both to promote remyelination and to protect against demyelination.

Symposium 7
Understanding neural circuits

S7-1

Pea3 family of transcription factors in neural circuit formation and selectivity

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Neural positioning and selectivity in wiring are important aspects of neural circuit formation, which were shown to be regulated by Pea3 subfamily of transcription factors, however the transcriptional targets of Pea3 in this process were so far unclear. It was shown for some time that Pea3 subfamily members, namely Pea3, Erm and Er81, were expressed in a mutually exclusive manner in motor neuron subpopulations, that GDNF, FGF and Met signals regulated expression of Pea3 proteins, in the absence of which motor neuron body positioning and / or connectivity pattern was either affected or compromised. The known downstream transcriptional targets of these highly homologous proteins, however, were unclear, with only a few targets such as matrix metalloproteases (MMPs) being identified. In our laboratory, we have focused on genome-wide profiling of transcriptional targets for Pea3, Erm and Er81 in different neuronal cell model systems, including SH-SY5Y, mHippo and mHypo cells. Our results indicate context-dependence of Pea3 family targets, with far less genes being responsive to Pea3 or Erm, but more genes being affected by Er81 in hypothalamic neurons. Data also shows overlapping genes for all three family members, as well as transcription factor-specific targets, which may well explain the selectivity of circuit formation. Our analyses of potential target genes are ongoing. Our ultimate aim is to use a microfluidic-based system to analyze further how Pea3 family members might regulate directed-circuitry in different primary neuron types, and to study whether our findings can be applied to neuroregenerative approaches. Some of the targets identified in microarray analyses also indicate that this circuit selectivity may not be confined to neuron-neuron interactions, but rather may be applicable to neuron-glia and neuron-vascular interactions, which are crucial in forming the microniches of the developing nervous system.

S7-2

A grave dilemma: get connected, we die

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In culture, neurite extension from and synapse formation among the primary isolated neurons is a common phenomenon. Even the primary sensory neurons that have relavitely limited number of synapses in vivo tend to form synapses of various permutations (neurite – neurite, neurite-soma). Electrical network activity can be detected in such cultures, especially in long term incubation.While this signaling is thought to support survival of neurons, the connectedness also allows spread of a potential threat over the network. With an in vitro axotomy model we have been using in our lab, we observed that axotomy of a single neuron in the network leads to death of several other uninjured neurons. We have found thatthis effect, which represents the in vitro model of secondary damage and transneuronal degeneration, comes out with a complex scenario where chemical synapses between neurons, electrical synapses between glial cells, purine receptor activation by ATP and inflammatory mediators like TNF alpha all take part. This study was supported by TUBITAK (Project no 107S358)

S7-3

Molecular control of motor neuron circuitry development

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The formation of proper neuronal circuits is critical for normal functioning of the nervous system. While major failures in early developmental events give rise to more profound defects, subtler-alterationson later developmental processes might be underlying cause of a wide range of neuropsychiatric disorders. Therefore, understanding the molecular mechanisms of neural circuit formation become a major goal in neurobiology today for the functional cellular repair of complex brain circuitry. Substantial progress towards this goal has been made over the past decade by using variety of cellular and molecular biological techniques together with novel neuroimaging approaches. The main aim of this talk is to highlight molecular mechanisms of motor neuron circuitrydevelopment by giving a special emphasize on corticospinal motor neurons. These neurons play very important roles to initiate and modulate voluntary movement by receiving, integrating, and relaying cerebral cortex's input toward spinal targets. A number of genes are implicated in the key developmental processes of these neuronal populations. Some of these genes, such as Fezf2, regulate subcerebral projection neuron identity while others, such as Ctip2, regulate the fasciculation, outgrowth and pathfinding of corticospinal axonal projections. Molecular control of these genes over the motor neurons will be demonstrated by giving examples from loss-of-function and gain-of-function experiments. In addition, influences of other factors including environmental factors, medications, neuronal activity on the cellular structure of these neurons will be discussed by giving examples not only from morphological studies, but also from large-scale "-omics" studies. We hope that this symposium will provide multidisciplinary a forum for critically reviewing recent progress in diverse parts of neural circuit formation.

S7-4

A biochemical view to axonal outgrowth

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In the developing nervous system, proper axon outgrowth and pathfinding are essential for neurons to reach their final destination and establish precise neuronal circuits. Injured neurons in the adult mammalian central nervous system have a very limited capacity for axonal regeneration and neurite outgrowth. Both conditions require molecules to axonal growth and/or reorganization. Extracellular guidance signals including growth factors/neurotrophic factors, cell adhesion molecules/integrins and canonical guidance proteins. These proteins are responsible for directing the navigation of the growth cone of an extending axon. Another important point in axonal growth is the cellular energy metabolism. Elevated levels of cAMP in nervous system are important for the guidance and stability of growth cones. Hypoxia/ischemia or mitochondrial poisoning that rapidly results in energy failure and neuronal network dysfunction. Chronic effects of mitochondrial dysfunction increased oxidative stress which results with neurodegeneration. Our experimental studies are focused on two various model. One of them is an in vivo experimental rat toxicity model. Rats received acrylamide, which induced axonal degeneration. The second model is an ex vivo organotypic hippocampal slice culture model. Axonal transport is inhibited with colchicine treatments. Results of our studies are focused on antioxidant treatment strategies and energy metabolism. The link between axonal growth/regeneration and biochemical results will be discussed.

Symposium 8

Multimodal personalized treatments in neurodegenerative diseases and neuromodulation

S8-1

Neuromodulatory treatment approaches in neurodegenerative diseases

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Alzheimer's disease is the leading cause of mortality in the world leading to serious economic/social burden in the society. Drugs that are still in use today are developed more than 20 yearsa go, but, unfortunately, despite the large budgetary research investment current treatment strategies are failed. Current research indicates that in Alzheimer's disease, early diagnosis and recognition at the level of mild cognitive impairment are possible. Another important finding is that the disease involves different pathophysiological properties for each of the patient. The presence of such phenotype based differences in the pathophysiology opens a new window for the individualization of the therapeutic approach that can be applied to the patients. Non-invasive neuromodulation techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) have emerged as a promising individualized therapeutic approach in recent years. In this context, neuromodulation techniques have been shown potentially to interfere with brain plasticity processes and key compensatory mechanisms that lead to an increase in brain reserve. Accordingly, appropriate protocols have been shown to cause long-term modulation of brain plasticity. This above-mentioned individualized neuroplasticity based therapeutic properties of transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS) may allow us to modulate the progression of clinically relevant cognitive and behavioral symptoms of neurodegenerative diseases. In this study, we have evaluated the neuromodulatory treatments including also individualized treatment approaches in neurodegenerative diseases which were carried out in our dementia unit at Istanbul Medipol University.

S8-2

The role of neuromodulation in neurodegenerative diseases: *in-vivo* and *in-vitro* studies

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Increasing human data suggest the therapeutic and neurorestorative role of transcranial magnetic stimulation in various neurological and psychiatric disorders in humans. However, there are limited experimental studies in the literature enlighting the possible neuroprotective role of this method. In our presentation, we aimed to summarize the neuroprotective effect of rTMS in various animal studies that can help us to understand the underlying mechanism of the repetitive transcranial magnetic stimulation (rTMS).

S8-3

The human microbiome in Alzheimer's disease as potential source of biomarkers and therapy

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AD is a progressively deteriorating neuropsychiatric disease which attacks the brain leading to impaired memory, thinking, and judgment, disorientation, depression and behavioral changes. AD accounts for 60 to 90% of all forms of dementia and its prevalence throughout the world is rapidly increasing. AD is predicted to affect 1 in 85 people globally by 2050. The cause and developmental stages of AD has not been understood well. About 5% of AD cases have genetic or familial cause but the vast majority (~95%) of the cases are sporadic and do not have a known cause. The hallmark of the disease is abnormally accumalting tauproteins and secereted beta-amyloids by neuroncells. It has been hypothesized that microbes could directly or indirectly contribute to pathogenesis of AD. We live in association of trillions of microbes in and on our bodies that are collectively called "the human microbiome". Because these microbes can directly or indirectly impact on our health they make up the humans' extended genome. For example, the microbiome helps us digest the food we eat, supply vitamins and essential metabolites (e.g. vitamins, short chain fatty acids, etc.) for our cells, produce small molecules (e.g. amyloids, lipopolysaccharides), train our immune system and keep the pathogenic microbes in check. Host microbiomes are remarkably unique, thereby lending itself to forensic studies and personalized medicine. Diet, genetics, and environmental insults such as pesticides and excessively used antibiotics, are important factors capable of shifting healthy state microbiome to disease state, which is also called perturbed microbiome or "dysbiosis". A line of evidence have recently suggested that gut microbiome in chronic diseases is in dysbiosis state. Disbyotic microbiome is implicated in systemic inflammation in the body contributing to progression of the chronic disease. This dichotomy of the microbiome states in health and disease presents opportunity for developing prognostic biomarkers (microbial species, molecules, pathways). Also, recently healthy diet including prebiotics, probiotics and fecal transplantation and microbiome engineering are being intensely investigated as to be part of multimodal approaches in reducing the risk of complex diseases such as AD.

S8-4

EEG brain oscillation abnormalities in neurodegenerative diseases

Bahar Güntekin

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Research on EEG Brain Oscillations invaded the literature in the last decade. Cognitive and affective processes of the brain were investigated with the methodologies of EEG Brain Oscillations in healthy subjects and also in different patient groups. The methodologies of EEG-Brain Oscillations include: power spectrum analysis, digital filtering, phase locking factor and coherence analysis between different electrode

pairs. The cognitive impairment of the patients with neurodegenerative diseases was represented with the abnormalities in the EEG-Brain Oscillations. Alzheimer's disease (AD) patients and Parkinson disease (PD) patients both have reduced delta responses during cognitive tasks in comparison to healthy controls. While the AD subjects had decreased theta phase locking during cognitive task, this was not the case for PD patients. In this panel talk we will represent abnormalities of EEG Brain Oscillations in AD and PD subjects. We will discuss the common and distinct abnormalities found in the EEG of these different patient groups. In the first part of the talk we will briefly represent the properties of different EEG frequency bands (delta, theta, alpha, beta and gamma) during cognitive processes and then we will discuss what could the EEG abnormalities seen in these neurodegenerative diseases mean?

S8-5

Role of steroids and neurosteroids in the pathogenesis and treatment of neurodegerative disorders

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Brain is an steroidogenice ndocrine organ and is also the target of circulating steroids due to its abundant steroidreceptors. It's continiously shaped and modified by steroid milleu that changed by enviromental or endogenous stimuli. Aging and related neurodegenerative disorders are characterized by impaired repair and self-renewal, but some capacity of regeneration persist till advanced ages. Neurosteroids can stimulate both repair and regenerative processes. Among these, allopregnanolone being most studied one, may decrease hippocampal neuronal loss – even promote new neurons - by aging or Alzheimer disease in animal models. This steroid is produced from the progesterone but not can not stimulate to progesterone receptor. Allopregnanolone activates GABA-A receptors and has a promising potential in the treatment of not only neurodegenerative diseases but also neuropatic pain and mood-anxiety disorders. DHEA and DHEA-SO4 producedfromadrenal cortexof primates have very good correlations with aging and related diseases. Non-primates have very low DHEA, except songbirds. In songbirds, exogenous DHEA promotes singing behavior and causes enlargement of the song center of their brain. DHEA seems to be important in neuronal plasticity. It may have neuroprotective effects by MAO inhibition. DHEA levels are low in Alzheimer Disaese but not in Parkinson. Parkinson disease is more frequent and starts earlier in man compared to women. This gender difference may be attributable to estrogen. In Parkinson models, estrogen has a protective effect against dopaminergic neuronal loss. On the other hand, estrogen exposure may be a risk factor for Alzheimer Disease. The pleomorphisms of the enzymes involved in biosynthesis, metabolism or signaling of the estrogens have been found to be related to increased risk of Alzheimer disease. Endocrinological investigations and hormonal replacements / modifications should be integrated into thet reatment algorithms of neurodegenerative diseases. This can only be achieved with extensive efforts of the clinicians dealing with these patients.

Panels

(PS-1 — PS-2)

PS-1

Neuroscience in Turkey and Future Perspectives, 2017

Emel Ulupınar[1], Gülgün Şengül[2], Metehan Çiçek[3], Tamer Demiralp[4], Gürkan Öztürk[5], Ertuğrul Kılıç[5], Bayram Yılmaz[6]

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Similar to the progress in neuroscience in the World, this multidisciplinary area of research has been developing in Turkey as well. Increasingly more universities have been launching graduate programs in neuroscience throuought Turkey. These programs appear to attract both newly graduated students as well as post-graduates like PhD holders and clinical residents from neurological sciences. With participants of neuroscientists, scientific meetings have been organized for about 20 years under the name of Turkish Neuroscience Congress. Turkish Higher Education Council recognized and launched an academic qualification area (associate professorship) with the code of 10105.06 in 2015. Increasingly more brain research labs and research centers are being established at universitites and institutes in the neuroscience research area. The Scientific and Technological Council of Turkey (TUBITAK) and related government departments have been providing more funds and launching new porgrams dedicated to brain research. Turkey has established a new national funding agency (Health Institutes of Turkey, TÜSEB) under the act # 6569 with the vision of supporting health science and technology to serve to the country and World at large. TÜSEB is expected to provide funds and starts programs in the neuroscience area. The European Union launched a new program (Horizon 2020) in 2014 following the 6th and 7th framework programs. Horizon 2020 offers significant number of grants for neuroscience under several programs. At this panel, progress in neuroscience will be evaluated and discussed with interactive participation of the audience.

PS-2

The publishing of scientific papers

Paul Bolam

Oxford University & Editor-in-Chief of the European Journal of Neuroscience

Topics to be covered:

1. How a paper is handled once submitted to a journal
2. What happens to your paper?
3. The ‘peer-review’ system
4. What the Editors do
5. What the Reviewers do
6. Exercise: Looking at ‘good’ and ‘bad’ reviews
7. How to respond to Reviewers’ comments
8. Ethics of publishing (human subjects, animals, plagiarism)
9. What we expect in a good paper
10. Exercise: Looking at ‘good’ and ‘bad’ abstracts

Oral Presentations

(O-01 — O-60)

O-01

Comparision of the effect of medium supplements on dopamnergically induced human dental pulp stem cells

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Objective: In this study the effect of serum, serum supplement, enzymes and cell attachment proteins on the viability of dopaminergically induced adult human dental pulp mesenchymal stem cells (hDPSCs) was investigated.

Methods: Human DPSCs (approved by Non-invasive Clinical Research Ethic Committee of Hacettepe University, LUT 12/94–09) were cultured in α-MEM + 15% (v/v) FBS + 0.4% (v/v) antibiotic solution and characterization studies were carried out with the cells at passage 4. Dopaminergic induction studies were performed in Neurobasal A medium and B-27 supplement was used as serum supplement. The cells were induced for 14 days in SHH, FGF-8, bFGF, BDNF, GDNF and TGFβ-3 containing Neurobasal A medium which includes 1% (v/v) FBS or 0.5% (v/v) B-27. Induced hDPSCs were detached from the surface either by applying Trypsin/EDTA or Papain enzymes and transferred onto the laminin or fibronectin/laminin coated Petri dishes. Cell viability was investigated by F-actin/DAPI staining and invert phase contrast microscopy images.

Results: No morphological differences were observed between these two groups before detachment. Human DPSCs induced in serum containing medium maintained their viability for 8 days after detachment. The cells induced in the presence of B-27 were able to attach onto the surfaces at very low cell density, most of the attached cells were detached from the surfaces in the late cell culture and cell nucleus was contracted and became indistinct.

Conclusion: Cell deaths observed for the cells cultured in B-27 containing medium was determined to follow a pathway independent from the enzyme and attachment proteins. In the following studies, detailed investigation on the effects of serum and B27 components on the dopaminergic induction of hDPSCs in the molecular level to explain cell death mechanism is aimed.This study was supported by the projects TUBITAK 113S285, Hacettepe University 10875. Merve Çapkın Yurtsever was also financially supported by TUBITAK-BIDEB 2211/A.

Keywords: human dental pulp stem cells, dopaminergic induction, serum, B27

O-02

Investigation of the resistance to glutamate-induced excitoxicity in mouse motor neuron-like NSC-34 cells on graphene oxide films

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Objective: Excess glutamate release leads to glutamate-induced excitotoxicity and neuronal death by overstimulating NMDA-type glutamate receptors. Graphene, a monolayer of sp2-bonded carbon atoms, is a quasi-two-dimensional (2D) material with unique electrical and chemical properties, enabling electrical activity of the cells and facilitating the integration with neural tissues. The aim of this in vitro study was to investigate the behavior of motor neurons on graphene oxide (an oxidized form of graphene) substrates and under glutamate-induced excitotoxicity which has been shown to be an important factor in many neurodegenerative diseases. For this purpose, we used graphene oxide film as a substrate for motor neuron-like NSC34 cells, a hybrid cell line produced by fusion of mouse neuroblastoma with mouse motor neuron-enriched primary spinal cord cells. Results: SEM results showed that the cells were attached on GO films. Cell viability (MTT) and toxicity (LDH) assays suggested a resistance to glutamate-induced excitoxicity in NSC-34 cells on graphene oxide films.

Methods: NSC-34 cells were grown in DMEM high glucose formulation containing 10% fetal bovine serum. Cells were seeded on GO films and cells cultivated on tissue culture polystyrene were used as control. DMEM-F12 with reduced serum concentration (1%) and 1% non-essential amino acids as used for differentiation. After 6 days of differentiation, L-glutamic acid induced excitotoxicity was applied on NSC-34 cells on both surfaces. Following stress, morphologies of cultured neurons were examined by scanning electron microscopy (SEM) and immunostaining. Cell viability was measured by MTT assay. SEM results showed that the cells were attached on GO films. Cell viability (MTT) and toxicity (LDH) assays suggested a resistance to glutamate-induced excitoxicity in NSC-34 cells on graphene oxide films.

Results: The effective L-glutamic acid dose was found 5mM and after 1h exposure as measured by MTT and LDH assays for cell viability and toxicity. After 1 h L-glutamic acid exposured, D- NSC-34 cells are more resistant on GO film by toxicity (LDH) assays. As SEM and immunostaining results, the

cells are overlapped on GO film and have less neurites than polystyrene surfaces. In MTT results, GO had a protective effect on undifferentiated NSC-34 cells after 24h. In LDH assay, after 1h L-glutamic acid exposure, the cells were more resistant to cytotoxicity on GO films.

Conclusion: These findings suggest that GO is biocompatible with NSC-34 cells and has a protective effect against cytotoxicity.

Keywords: differentation, graphene oxide film, motor neuron-like cell

O-03

Identification of HRP3 as a neuron derived factor that regulates glial cell biology and myelination

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Objective: Myelin, produced by oligodendrocytes in the central nervous system (CNS) and by Schwann cells in the peripheral nervous system (PNS), envelops the axons and enables fast and efficient action potential transmission. It also provides metabolic support to the neurons. Myelination is regulated by signals between neurons and glial cells. Our preliminary studies suggested that Hepatoma-derived growth factor-related protein 3 (HRP3) is a neuron derived factor that regulates myelination. HRP3 expression was increased during both myelinization and remyelination following cuprizone induced myelin damage. In the current study, we aim to examine the effects of HRP3 on the regulation of glial biology and to compare its effects on CNS and PSS myelination.

Methods: Immunohistochemical and Western blot analyzes on rats showed that the amount of HRP3 was increased during myelination and HRP3 localized to the axons during myelination while localizing to the nucleus post-myelination. We used dorsal root ganglion (DRG) neuron-Schwann cell co-cultures as an in vitro PNS model and embryonic stem cell-derived neuron-oligodendrocyte co-cultures in microfluidic chambers as a CNS model. Short-distance signals between neurons and glia were examined in co-culture, whereas feeding Schwann cells and oligodendrocytes with neuron conditioned medium was used to examine the long-distance signals.

Results: Immunohistochemical analyses revealed that neuron specific overexpression of HRP3 via GFP-labeled lentivirus in DRG neurons significantly increased Schwann cell number and myelination. In the CNS model, although the number of mature oligodendrocytes increased significantly, there was no significant increase in myelin levels.

Conclusion: We observed that HRP3 shuttles to the axons during myelination and stimulates glial cells. Our ongoing experiments using conditioned medium, aim to resolve whether the effect of HRP3 is caused by direct cellular contact or by HRP3-mediated signals released to the environment. The part of study that is conducted in Turkey was supported by TUBITAK and Turkish Academy of Sciences.

Keywords: myelination, hepatoma-derived growth factor-related protein-3 (HRP3), schwann cell, oligodendrocytes

O-04

Axolotl cell suspension application as a method for treatment of mouse spinal cord injury

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Objective: Spinal cord injury is a condition leading to the death of neurons and disruption of axons in the injury site. It is well known that human central nervous system (CNS) has a very limited capacity for regeneration and injuries occurring in CNS cause progressive dysfunction. Tissue regeneration is a well known event observed after spinal cord injury in semenders and these processes could also be seen in the adult stage. In this study, our aim is to investigate the regeneration observed in spinal cord injured SCID mice to which cells isolated from eGFP transgenic axolotl's spinal cord have been transferred, by using immunohistochemical and behavioral analyses.

Methods: There are 3 experimental groups; control group (n=7), spinal cord injury group (n=7) and regeneration group (n=7). Spinal cord injuries were done in mice of the second and third groups. Spinal cord injury was also done in axolotls and formed blastema was transferred as a cell suspension into the mice of third group.

Results: The effects of transferred cell suspensions were observed by using open field tests and immunohistochemical analyses. The motor capacity of mice was evaluated by open field test. Total distance travelled and the average speed of the SCID mice in the second group decreased 75.42% and 75%, respectively, with respect to the control group ($p<0.05$). The same parameters in the third group increased by 78.7% and 80%, respectively, with respect to the second group ($p<0.05$). Tissue sections of the mice in the third group were taken from their spinal cords. Samples were labeled by neuronal and glial markers and visualized by confocal microscopy. Immunohistochemistry

revealed that neurons derived from eGFP axolotls remain alive in SCID mice after 3 weeks.

Conclusion: Our findings altogether indicated that signs of neuroregeneration in spinal cord injured mice were observed after transferring axolotl's cell suspensions.

Keywords: spinal cord injury, neuroregeneration, axolotl, SCID Mouse

O-05

Investigation of muscarinic receptors in mesenchymal stem cells obtained from different sources in vitro and influence of receptor blockadge on differentiation

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Objective: For regeneration purposes, it may be possible to demonstrate the functional activities required for transplantation of new stem cells, where stem cells are placed by exspressing appropriate receptors on cell surfaces. At present, information on receptor expression of mesenchymal stem cells is not sufficient. This study was aimed to demonstrate muscarinic receptors in mesenchymal stem cells and to determine the effect of differentiation of receptor blockade.

Methods: mRNA expression of muscarinic receptors subtypes in osteogenic and adipogenic differentiation, and the effect of atropine in the 1st, 2nd and 3rd passages of mesenchymal stem cells obtained from human placental fetal membrane (FM) and bone marrow (BM) were shown by RT-qPCR. To determination the effect of blockers on differentiation Bone Morphogenetic Protein (BMP-6) and Peroxisomeproliferator-Activated Receptor gamma (PPAR) from osteogenic aspect, and PPARγ from adipogenic aspect were investigated via RT-qPCR.

Results: Significant increase in FM groups was detected in M1 mRNA expression compared to the control group. This situation was seen as a decrease for the BM groups. No significant changes were found in the FM groups compared to the control for M5 mRNA expression but significant decrease was seen in the BM groups. BMP-6 mRNA expressions in the FM and BM groups in the osteogenic differentiated cells increased significantly compared to the undifferentiated cells. In the groups derived from both sources, PPARγ mRNA expressions in adipogenic differentiated cells were showed the significant increase compared to the control group.

Conclusion: These results indicate that the cells obtained from different sources in that the expressions of muscarinic receptors behave differently and indicate that there are also some changes derived from the same sources cells according to the passage and differentiation. It is important to investigate muscarinic receptors from different sources and passages with in-vivo studies to obtain more effective clinic results.

Keywords: cell differentiation, mesenchymal stem cells, muscarinic receptors, RT-QPCR

O-06

Characterization of mouse cardiomyocytes and sensory neurons interactions

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Objective: Cardiovascular diseases are contributed to 30% of all deaths worldwide despite attempts to develop new therapies. To this end, the main aim of this study is revealing the molecular and cellular interaction between myocardium and the innervating sensory neurons. The sensory nervous system transmits signal about the homeostatic statue of the heart to the brain through dorsal root ganglion (DRG) and Nodose ganglion consisting of their cells bodies. Even though the motor control of autonomic nerves over the heart is well characterized in the literature, the role played sensory neurons is unclear. To this end, cardiomyocyte and neuron co-culture provides to investigate interaction between cardiac specific sensory neurons and cardiomyocyte.

Methods: In this study, fluorescence and voltage sensitive dye moving through membrane lipids retrogradely was injected in to hearts of adult Balb-C mice to label cardiac specific sensory neurons. At the end of 1, 3, 7, 14th days after injection, nodose ganglion and DRG located in cervical and thoracic levels were dissociated enzymatically and observed under fluorescent microscopy.

Results: As a result of investigation, it was observed those fluorescence labeled cardiac specific neurons were obtained. For further analysis, obtained heterogeneous sensory neurons were analyzed in detail using quantitative reverse-transcriptase PCR (qRTPCR) and immunocytochemistry methods. In addition, optogenetics tools are used to investigate the way of electrical signal and molecular mechanism between cardiomyocytes and interacted sensory neurons. Membrane potential of cardiomyocyte and neurons transfected with blue light sensitive channel rhodopsin 2 are examined. In the following step, purified cardiac specific sensory neurons and cardiomyocytes interaction in co-culture will be investigated.

Conclusion: With the knowledge gained from this study, novel therapeutic approaches can be developed for heart disease by modulating nervous system together with cardiac muscle. This study is supported by TUBITAK under 1001 Scientific and Technological Research Projects Funding Program by project no: 115S381.

Keywords: cell culture, sensory neurons, cardiomyocytes, optogenetics, live imaging

O-07

Evaluation of EEG records in acute experimental *Toxoplasma gondii* infection in rats

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Objective: *Toxoplasma gondii* is a zoonotic, neurotrophic parasite with intracellular localization. Depending on its location in the brain, it causes epileptic like symptoms. It is reported that approximately 1/3 of the people in the world are infected with this parasite. The spreads in Turkey are recorded to ranging from 30 to 70%. Epilepsy is one of the most common diseases of the central nervous system. The source of cryptogenic and idiopathic epilepsy is thought to be parasitic, such as *T. gondii* and *Cysticercus cellulose*. This study; is designed to detect EEG records in experimental rats with acute *T. gondii* infection.

Methods: In this study 2–4 months male Wistar albino rats were used. The rats were divided into four groups, one control and three treatment. Primarily, all animals were under anesthetized were placed on permanent eklektrod. The control group (PTZ (40 mg/kg) groups) (G1) (n=4) were awake an hour EEG recordings. The animals in experimental group were injected with *T. gondii* strain 1x104 / 1 ml via intraperitoneal (IP) than after were awake with one hour EEG recordings on 8 (G2) (n=9), 15 (G3) (n=6) and 30 (G4) (n=6) days of the infection.

Results: The number of seizures, seizure duration, DDD (spike wave discharges) data were evaluated by one way ANOVA and Tukey test. The number of seizures in the control group (G1, PTZ group) was found to be significantly higher than the infection group (F (3.21)=4.51 p=0.014 n2=0.39). The duration of seizure (F (3.21)=0.95 p=0.43 n2=0.12) and DDD (F (3.21)=1.13 p=0.39 n2= 0.14) It was seen that there was no significant difference between the groups.

Conclusion: As a result, it has been shown that acute experimental *T. gondii* infection in rats can cause seizures and epilepsy. This study is supported by The Scientific and Technological Research Council of Turkey (TUBITAK) given project number as 115S223

Keywords: *T. gondii*, rats, epilepsy, EEG, PTZ

O-08

Correlation between the fluid intelligence and the quantitative EEG delta frequency response in women and men

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Objective: Fluid intelligence could only be measured through neurocognitive tests. Although the relationship between the quantitative electroencephalography (EEG) and neurocognitive tests is known, researches about this relationship are insufficient. The aim of this study was to investigate the relationship between the EEG delta frequency responses and the fluid intelligence.

Methods: Resting state-EEG recordings were taken from 16 healty volunteer participants (ages 19–38; mean =22.25) with a 32 channel recording system in the Faraday cage. The delta power values were obtained from the EEG recordings with the BESA program. For quantitative analysis, artifact free 45 epochs (30 eye-open, 15 eye-close) lasting 2 s each were selected and the delta frequency power values were calculted for each brain lob. The same volunteers participated in the Raven's Standart Progressive Matrices (RSPM) test. RSPM test was carried out as 5 sets (A, B, C, D, and E) and each set includes 12 questions. Volunteers answered a total of 60 questions and duration of the test was recorded. The relationship between the fluid intelligence test scores and the delta frequency was evaluated.

Results: Women have the highest delta frequency power value in the occipital region (24.75) while men have it in their frontal region (30.6). Both groups have the lowest delta frequency power values in their temporal region (women=20.16; men=22.25). Women averaged a score of 49.88 RSPM points while men avaraged 48.42. The participants scored highest on set A and lowest on set E. Men had an intellectual activity speed faster by 148.2 seconds compared to women.

Conclusion: Compared to men, women have higher positive correlation between the delta frequency and fluid intelligence, while the duration of the RSPM test was shorter for men. A Positive correlation between brain regions responsable for highest delta responses and the regions includes fluid intelligence was determined.

Keywords: intelligence, RSPM test, EEG delta frequency

O-09

Correlation between the visual perception and the EEG theta frequency response

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Objective: Because of biological, neurophysiological, psychological characteristics of visual perception,researches must involve an interdisciplinary approach. There are studies explain the effects of electroencephalography (EEG) waves on the visual perception. However, neurocognitive and neurobiological relationships of these studies are not clear. The aim of this study was to explore the relationship between the EEG teta frequency responses and the visual perception.

Methods: Known the best visual perception test the Raven Progressive Matrices (RPM) test was applied to 16 healty vol-

unteers. The questions consisted of missing parts from visual matrices where the volunteers were requested to correctly choose, from the given options, the correct part. Resting-state EEG recordings were taken from the same volunteers. Theta frequency power values were obtained from artifact free epochs (30 opened-eye and 15 closed-eye) lasting 2 s each. EEG theta frequency values and RPM test scores were correlated.

Results: According to RPM test, average scores of women are 5% higher than men. Duration to finish the test is 2.4 min longer for women with respect to men. In men EEG power values are; Occipital 19.67, Parietal 18.63, Temporal 15.68, Frontal 18.19. In women EEG power values are Occipital 30.81, Parietal 23.96, Temporal 15.68, Frontal 19.43.

Conclusion: Relationship between the theta bad and the visual perception was found higher in women compared to men. EEG Theta power value is the highest in the Occipital region of brain and the lowest in the Temporal region. A gender-independent positive correlation between the RPM test results and the EEG Theta frequency findings was determined.

Keywords: visual perception, RSPM test, EEG theta frequency

O-10

Accurate and inaccurate feeling of knowing in semantic memory: an event-related potential study

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Objective: Feeling of knowing (FOK) is a metacognitive process which allows individuals to predict the likelihood that they will be able to remember information which they currently cannot recall. Studies revealed that the accuracy of FOK judgments increases when people's confidence level increases during semantic memory process. Although FOK provides evidence for the mechanisms of metacognitive systems, the neurobiological basis of FOK is still unclear. In this study, effects of the accuracy of FOK judgments were investigated during semantic memory using event-related potentials (ERPs).

Methods: 74 undergraduate students (48 female) aged between 18 and 26 (M=21.10, SD=1.50) participated in the study. 30 general information questions and the classical recall-judgment-recognition (RJR) paradigm were used to measure FOK judgment during semantic memory task. Stimulus presentation, recording, storage and analysis were carried out by using 32 Channel EEG NeuroScan system. The stimulus onset ERP averages (amplitude and latency) were calculated for central, frontal, parietal electrode sides separately.

Results: Greater negativity peaking between 70 and 120 msec (N100) followed by a positive peak between 160 and 240 msec (P200) were recorded at all electrode sites. The amplitude values of these components were high for fronto-central electrode locations. The biggest N100 amplitude was recorded at midline electrodes (Cz, Fz, Pz) whereas P200 amplitude at left fronto-central electrodes (C3, F3) was bigger than other electrodes. Significant N100 amplitude differences were observed between accurate and inaccurate FOK judgments at all electrode locations. The results supported the previous literature showing that frontal areas have a crucial role for memory-monitoring processes.

Conclusion: Accurate FOK judgments were associated with greater negativity for N100 component that may support the familiarity-based effect. On the other hand, P200 component at frontal and central electrodes may be related with assessing familiarity of stimulus in order to guide a successful recognition.

Keywords: event-related potentials, feeling of knowing, semantic memory

O-11

Auditory linguistic stimuli causes lateralized physiological and haemodynamic responses in brain

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Objective: Changes in the tympanic membrane temperature (TMT) reflect the ongoing activity of the ipsilateral hemisphere and can produce lateralized responses to hemisphere specific stimulation. The present study investigated whether verbal stimuli have a lateralized effect on TMT.

Methods: Thirty-three undergraduate students were asked to listen to a word list consisting of 20 monosyllabic Turkish and 10 monosyllabic English words in a randomized order while simultaneous TMT recordings from both ears and fNIRS recording from forehead were performed. Furthermore, to increase the difficulty of the task and engage the subjects, the English words were choosen to be phonetically ambiguous to denote Turkish words as well (such as "car", "boy"). The subjects were assigned to one of three groups with individual instructions given at the beginning of the experiment. The control group was asked to simply listen to the list; the word group was asked to remember and write down the English words at the end of the experiment; the number group was asked to remember the number of English words presented in the list.

Results: The highest temperature increase in both ear during the experiment was found in word group ($p<0.01$). A verbal stimulation caused the highest temperature difference between left and right ear in word group ($p<0.05$) (calculated as left-right). Correlations between the (left-right) TMT difference between the two ears and the flowing total blood volume (calculated by adding oxy-hemoglobin and deoxy-hemoglobin signals) difference to the hemispheres were found to be statistically significant in all three groups ($p<0.01$). Haemodynamic response function(HRF) for Turkish and English words in each group was calculated and it was found that word group's HRF has shorter time-to-peak values than other groups ($p<0.01$).

Conclusion: Findings show that a verbal task can produce lateralized effect on TMT, and TMT is strongly correlated with haemodynamic activity.

Keywords: functional near infrared spectroscopy, language, lateralization, tympanic membrane temperature

O-12

Reliability of Turkish version of Flinders Handedness Survey

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Objective: The aim of this study to investigate the reliability of Turkish version of Flinders Handedness Survey.

Methods: 149 healthy subjects (120 females and 29 males; ages between years 18–31) were included in the study. Flinders Handedness Survey is a 10-item questionnaire to assess hand preference. The questionnaire was applied with one-week interval for all these participants to evaluate the test-retest reliability. This questionnaire requires subjects to indicate which hand they usually use for various actions as follows: writing, using a spoon, using a toothbroush, holding a match when striking it, using an eraser on paper, holding a needle when sewing, holding a knife when buttering bread, using a hammer, holding a peeler when peeling an apple and drawing. Instructions for participants were given at the top of the questionnaire together with some questions relating to basic demographic data. Responses of 'left', 'either' and 'right' are assigned scores of -1, 0 and +1, respectively. These scores are then summed to give a test score that ranges from -10 to +10. Individuals with scores ranging between -10 and -5 are deemed to be left-handed whereas individuals with scores ranging between +5 and +10 are right-handed. Individuals with scores between these ranges are mixed handed. The test-retest reliability was assesessed with Cohen's Kappa coefficient.

Results: Scores for all items ranging from 0.53 to 0.95. The best scores indicate excellent agreement were found for these items: writing (0.95) and drawing (0.86). The least scores indicate substantial levels of agreement were found for these items: holding a needle when sewing (0.60), holding a knife when buttering bread (0.55), holding a peeler when peeling an apple (0.53).

Conclusion: Our results suggest that Flinders Handedness Survey is reliable in measuring handedness in Turkish population. Further studies with larger sample size are needed to assess hand preference.

Keywords: hand preference, cerebral asymmetry, reliability

O-13

The neuroprotective effect of regular swimming exercise programme on the dopaminergic neurons localized in the striatum of Parkinsonian rat models

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Objective: Parkinson disease is the second most common neurodegenerative disease characterized by the loss of dopaminergic neurons found within the substantia nigra pars compacta.The physical therapy is being involved as one of the mainstay in the treatment options.We aimed to observe the effect of the regular exercise on the dopaminergic and CR(calretinin) positive neurons found in the striatum of the rats with PD by applying immunohistochemistry protocols.

Methods: 6-OHDA is injected unilaterally to the medial forebrain bundle of the wistar rats (n=8) by using the stereotaxic method. Rats are divided into two as sedentary and exercise groups. Rats found in the exercise group are made to swim regularly for 30 minutes for 5 days (6 weeks). At the 21st day following to the injection, by apomorphine injection (0.05 mg/kg/sc), rotation test is applied. The brain excision is done by the transcardiac perfusion method after the exercise program. Samples are stained with TH (thyrosine hydroxylase) and CR protocols.

Results: The number of rotations in the exercise group decreased (p=0.0058) with a statistically significant difference. The density of TH positive neurons were not to be seen different noticably in the striatum and substantia nigra. The sum of CR positive neurons showed a statistically significant difference (p=0.009).The number of CR positive neurons obtained from the ipsilateral (lesioned) and contralateral side of the samples belonging to exercise group were shown to have a significant increase (p=0.028), (p=0.008) respectively.

Conclusion: As a conclusion, we would like to show a direction to the further clinical studies in order to show the neuroprotective effects of the vigorous and prolonged periods of exercise in the treatment.

Keywords: Parkinson, exercise, 6 OHDA, thyrosine hydroxylase, calretinin

O-14

Effect of experimental diabetes on proteins associated with Alzheimer's disease

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Objective: Diabetes mellitus (DM) which is one of the foremost health problems enhances the risk of Alzheimer's disease (AD) although the molecular mechanisms behind this association remain unclear. In the present study, we aimed to elucidate these molecular mechanisms.

Methods: Selective Alzheimer's disease indicator-1 (seladin-1) and insulin degrading enzyme (IDE) expressions were investigated in rat primary cultured neurons under diabetic conditions and in the brains of streptozotocine (STZ)-induced diabetic rats. Besides, how neuroprotective 17β-estradiol and the antidiabetic drug metformin affect these proteins has been investigated.

Results: We found that lack of insulin rather than high glucose levels decreased seladin-1 and IDE protein (18.2% $p<0.001$ and 16.8% $p<0.05$ respectively) and mRNA levels (58.4% $p<0.001$ and 16.3% respectively) in cultured rat primary neurons after 5 days incubation. If the diabetic conditions were intermittent, neuronal seladin-1 levels were unaffected whereas IDE levels were reduced (34.8% $p<0.001$). Beta secretase-1 (BACE-1), which has an important role in AD pathogenesis, increased (15.7% $p<0.001$) accompanying to decreased seladin-1 levels after 5 days of insulin deprivation. Metformin incubation in the presence of insulin increased seladin-1 protein (35.9% $p<0.001$) and mRNA (32.4%) levels. Metformin and estradiol incubation with intermittent glucose treatment caused IDE to remain at control levels. STZ-induced diabetes reduced seladin-1 and IDE (14.3% $p<0.01$ and 28.3% $p<0.001$ respectively) but did not change BACE-1 levels in the cerebral cortex of rats. Ovariectomy, which performed in order to clarify the effect of estradiol on mentioned proteins, did not change seladin-1 and IDE levels whereas STZ-induced diabetes in ovariectomized rats caused reduced seladin-1 (24.8% $p<0.001$) and IDE (34.6% $p<0.001$) levels compared to control.

Conclusion: Taken together we suggest that seladin-1 and IDE may represent new treatment targets for DM patients to prevent AD onset.

Keywords: Alzheimer's disease, diabetes mellitus, insulin degrading enzyme, seladin-1.

O-15

The effect of a Rho kinase enzyme inhibitor Fasudil on seizures in rats with absence epilepsy

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Objective: There is evidence that the Rho/Rho-kinase pathway may play a role in convulsive type epilepsy. Besides Rho proteins are upregulated in a pentylenetetrazole model of temporal lobe epilepsy. But this pathway and its specific inhibitors have not been investigated in absence epilepsy. We examined the effects of RhoA/Rho kinase pathway on the mechanisms of absence epilepsy for the first time by using a potent and specific RhoA/Rho kinase inhibitor, Fasudil.

Methods: In the experiments, 4–7 months old Genetic Absence Epilepsy Rats from Strasbourg (GAERS) were used. Under Ketamine (100 mg/kg, ip.) and Xylazine (10 mg/kg, i.p.) anesthesia, an intracerebroventricular (icv) cannula was implanted in the lateral ventricle of the rat and the electrodes were implanted over the right and left frontoparietal cortices. After one week of recovery, EEG recordings were done to measure the number and duration of basal spike-and-wave discharges (SWDs). Three different doses (10, 30 and 100 nmol, the number of rats are n=4.5 and 7, respectively) of Fasudil were given by an icv injection within 5 µL volume. Consequently, EEG recordings were performed for three hours. EEG signals were processed by Powerlab 8S EEG recording system and analyzed. The number and the total duration of SWDs in the cortical EEG recordings of the basal and post-injection periods were evaluated.

Results: Fasudil suppressed SWDs in a dose dependent manner. The concentration of 100 nmol, decreased the total duration and the number of SWDs significantly. Especially in the first 20 minutes after injection, the total duration was 63.71± 64.18 s and the number of SWDs was 3.57±8.35, and for both the statistical significance was $p<0.001$.

Conclusion: The current results show that Rho/Rho-kinase pathway may take a role in the etiopathogenesis of the absence epilepsy, and Fasudil may have the potential to be an anti-absence drug. This project is supported by TUBITAK (113S221).

Keywords: Rho kinase, RhoA, Fasudil, GAERS, absence epilepsy

O-16

The role of soluble guanylate cyclase activator BAY41-2272 in the analgesic effects of extremely low frequency electromagnetic fields in rats

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Objective: The analgesic mechanism of low-frequency electromagnetic fields is not enough scientifically enlightened. Our aim of this study is to investigate the role of soluble guanylate cyclase activator BAY41-2272 in the analgesic effect of very low frequency electromagnetic fields in rats.

Methods: In this study were used 72 adult male Wistar albino rats (250±15 g). Rats were randomly divided into 4 groups (Sham, magnetic field (MF), L-arginine and MF+L-arginine). The rats were provided environment where is at 22±2 °C room temperature, 12-hour light/dark cycle and insulated from sound. 50 Hz magnetic field application, the same times for 30

minutes each day for 15 days, and a total of four times every 15 minute intervals. The analgesic effect measurement was performed by tail-flick and hot-plate test equipment. Prior to analgesia test, BAY 41-2272 (10 mg/kg) and nitric oxide donor L-arginine (300 mg/kg) was injected intraperitoneally in rats. The resulting data was converted to % analgesic effect (% MPE). In the statistical analyzes of the data, analysis of variance (two-way ANOVA) was used and the multiple comparison determined by Tukey tests. The level of statistically significant was expressed $p<0.05$.

Results: Analgesia test results showed that maximum analgesic effect of magnetic field produces in 5 mT and on day 7 (tail-flick: 25.89±3.00 and hot-plate: 61.73±2.95). Administration of BAY41-2272 in rats (tail-flick: 42.11±4.45 and hot-plate: 62.42±4.67) exposed to a magnetic field the analgesic effects were significantly higher than magnetic field group rats (tail-flick: 32.11±3.45 and hot-plate: 51.13±4.78) ($p<0.05$). Similarly, the analgesic effect of L-arginine group (tail-flick: 42.11±3.45; hot-plate: 61.13±4.78) were found to be significantly higher compared to the magnetic field group ($p<0.05$).

Conclusion: The findings indicated that soluble guanylate cyclase activator BAY 41-2272 and nitric oxide donor L-arginine increase the analgesic effect produced by the magnetic field.

Keywords: magnetic field, analgesia, soluble guanylate cyclase, BAY41-2272, L-arginine

O-17

Modeling dynamic behavior of striatal medium spiny neurons with point neuron models

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Objective: The aim of this work is to investigate the role of network organization and the neuron model type on the collective dynamic behavior of striatal population. For that purpose, two different scale neuron models which are phenomenological Izhikevich and conductance-based Hodgkin-Huxley (HH) type are used to investigate the dynamic behavior of MS neurons.

Methods: Two network architectures are proposed with inhibitory and excitatory synaptic currents. In these networks, while all MS neurons affect each other with inhibitory synaptic currents, an excitatory current is applied to all medium spiny (MS) neurons in the first layer, to represent the cortical inputs. A mathematical model of a medium spiny neuron of striatum based on HH type neuron model is proposed using different calcium channels and its dynamical behavior is investigated.

Results: It is observed that when the original HH model is used, regular spiking type behavior is observed. Including the high threshold calcium current, after hyperpolarization calcium current and voltage gated potasium current into the model improves the modeling capabilities. With extended ion channels, in addition to regular spiking behavior, bursting with resting stage are obtained. Then, Izhikevich neuron model is used in the network structures to compare the dynamic behaviors and computational time.

Conclusion: It is shown that, using simple neuron model gives almost the same results as the complicated neuron model. Thus, to form larger networks, it is convenient to use simple neuron model. Also, the organization of neurons do affect the collective behavior.

Keywords: straitum, medium spiny neurons, Hodgkin-Huxley neuron model, Izhikevich model

O-18

A basal ganglia model formed with spiking neurons *via* Brian2 and a user interface to test the model

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Objective: A user interface is designed for basal ganglia circuit to create an experimental environment so that researchers who are interested in neuroscience from different disciplines can benefit the opportunities computational neuroscience provides.

Methods: Firstly a basal ganglia model is realized with Brian2 simulator to create the user interface.

Results: The model consists of spiking neural network where single neuron models are modified Izhikevich neuron model. While forming the network, neuron model and synaptic connections are considered to be dynamical systems. The model consists of cortex, striatum with three parts, globus pallidus interna and externa, subthalamic nucleus and thalamus. Each group has 100, 230, 100 and 100, 100, 100 neurons respectively. Striatum contains D1 and D2 regions and interneurons. D1 and D2 regions in striatum represent the spiny neurons which have D1 and D2 type receptors and these neurons are modelled to be sensitive to dopamine level. These neurons behavior change with dopamine level and the level can be adjusted manually. Therefore, the dopamine effect on the activity of basal ganglia circuit can be observed with the model. The model also includes direct, indirect and hyper-direct pathways. With the interface, action initiation performed with different dopamine levels and the effects of some disorders resulting from improper dopamine level can be easily observed. Outputs of the model are raster plots, firing rates and frequency responses. Frequency responses of the model are compared with earlier work in the literature and we obtained frequency responses with different dopamine levels that are close to the results in the literature for all groups except thalamus.

Conclusion: Synaptic weights, connection probabilities and dopamine level can be changed via user interface, and the model can run directly on the interface. Consequently interface outputs are raster plots, firing rates and frequency responses of each group.

Keywords: basal ganglia circuit, dopamine effect, action initiation, Brian2

O-19

The impact of pyramidal neuron excitability changes on a Jansen and Rit neural model of temporal lobe epilepsy

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Objective: The large-scale brain network modelling has a promising application in the diagnosis and treatment of neurological disorders such as epilepsy, depression, brain injury i.e. The Jansen & Rit is a neural population model which consists of excitatory, inhibitory interneurons and a pyramidal neuron that is made up of assembly of biophysical parameters of neural masses by changing region. This model allows us to generate an exceptional brain network model which is able to oscillate alpha-band. In this project, a large scale brain network model was simulated for mimicking temporal lobe epilepsy (TLE) with electroencephalogram (EEG) data based on Jansen & Rit neural model using *the virtual brain* (TVB) as a neuroinformatics platform.

Methods: In Jansen & Rit neural model, temporal lobe epilepsy model with biophysical parameters (mean firing rate and mean membrane potential values) that determine the excitability of pyramidal neurons in neural mass model was generated using pyramidal neuron hyperexcitability conditions which are obtained by regional modulation in the temporal lobe.

Results: EEG datasets which were achieved from this model at temporal lobe were compared with EEG datasets from patients. How to reflect especially the changes in hippocampal pyramidal neuron excitability over function and parameter ranges that can generate epileptic waveforms were analyzed in the obtained EEG results. Responses to local stimulation which may be able to suppress epileptic activity were examined on this model.

Conclusion: The results obtained from simulations emphasize that pyramidal neuron hyperexcitability can play an important role in temporal lobe epilepsy, and it also reveals how this system will respond to methods such as electromagnetic stimulation.

Keywords: hyperexcitability, Jansen & Rit neural model, large-scale brain network, temporal lobe epilepsy

O-20

Thermodynamic analysis of metabolic energy utilization during action potential generation in neurons

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Objective: Signal transmission in the nervous system is achieved by electrical signals in the form action potentials. During signal transmission, neuron membrane becomes depolarized and a fraction of sodium and potassium ions are relocated. In order to return to resting membrane potential (polarized state), Na+/K+ pump is activated and utilizes 1 mole of ATP for the exchange of 3 Na+ to 2 K+. The energy requirement is regarded as a limiting factor for initiation and transmission of action potential in neurons. The energy utilization and efficiency by Na+/K+ pump for neuronal function can be estimated by referring to the fundamental principles of thermodynamics.

Methods: In this study, Na ion load, ATP consumption, entropy generation and exergy loss were evaluated under the physiological conditions of pH=7, T=298.15 K and I=0.25 M in the giant squid axons and tiger salamander retinal ganglion. In addition, metabolic energy required for ionic pumping was analyzed in terms of the first law of thermodynamics and exergy efficiency. Hodgkin and Huxley quantitative approach reported in the studies with the squid giant axons was employed in the present analysis with the squid giant axon and tiger salamander retinal ganglion.

Results: In our analyses, during an action potential Na+ ion load was estimated as 1401 nC/cm^2 and 1902 nC/cm^2 in the squid giant axon and tiger salamander retinal ganglion, respectively. In addition, energy utilization per unit area was found as 2.92×10e12, 3.98×10e12 ATP in the squid giant axon and tiger salamander retinal ganglion, respectively. Thermodynamic approach utilized in the present study has revealed that entropy generation and exergy loss was higher in tiger salamander retinal ganglion. Also, squid giant axon was more efficient both energetically and exergetically in conversion of glucose to work.

Conclusion: The metabolic efficiency of action potential of a neuron is highly dependent on sodium channel kinetics and can show variations among different neurons. This variation of sodium channel kinetics affects metabolic energy cost for information transmission along axons. In future studies, thermodynamic approach in different neuron types remains to be employed for more and comparative data.

Keywords: ATP, entropy, exergy, efficiency

O-21

Network properties of dorsal root ganglion neurons *in vitro*

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Objective: Dorsal root ganglion (DRG) cells expressing GCaMP in glutamatergic neurons were cultured on Multi Electrode Array (MEA). The responses of the network to vari-

ous electrical stimuli were simultaneously observed and analyzed under microscope.

Methods: Cre dependent GCaMP expressing Ai96 (RCL-gCamp6s) transgenic mouse line was crossed with Vglut2-ires-cre mouse line expressing cre recombinase enzyme. Hence, double transgenic mouse line with GCaMP expressing glutamatergic neurons were generated. DRG neurons from adult mice in this line were cultured after ganglia were dissected, neurons and glial cells were separated using a Percoll gradient. Experiments started after neurite-extending neurons formed network like structures. Calcium activity of DRG neurons cultured on polyethylenimine (PEI) and laminin coated MEA was monitored with 488 nm laser attached Spinning Disk microscope. Various electrical stimuli patterns applied to the cells by altering amplitude and frequency settings through the electrodes. In order to distinguish the signals that emerge as response to these stimuli, the spontaneous activity observed in advance. Evoked responses were recorded from single cells as well as from cells connected with each other. Besides, changes in amplitude and frequency of stimulation have resulted as axonal activities or long-term response patterns.

Results: Experiments for testing known evoked responses via varying chemical blocking methods are being continued. To the extent that it is intended to determine cell connection types and functional network structures.

Conclusion: It is estimated that various cell types form characteristic network structures, and connections in these network structures are thought to take place through different mechanisms. This work is supported by Boğaziçi University Research Fund under the Project Code 8080D.

Keywords: dorsal root ganglion (DRG), multi electrode array (MEA), GCaMP

O-22

Effects of gold nanoparticles and their polyethylene glycol modifications on hippocampal neurons

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Objective: In the past decade, gold nanoparticles (AuNps) have been suggested to use in biomedical applications and neurology such as cancer treatment and contrast agent in computer tomography. In this study, bioelectrical effects of 5nm AuNps and polyethylene glycol modified (PEG) AuNPs on hippocampal neurons were investigated.

Methods: Nanoparticles were characterized by UV/Vis spectroscopy, Dynamic Light scattering (DLS), Transmitted Electron Microscope (TEM) and Atomic Force Microscope (AFM). Four to six weeks old C57BL/6 male mice were used to prepare brain hippocampal slices (300 μm). Spontaneous and evoked actions potentials were recorded by patch-clamp whole-cell configuration from control, AuNp or PEG-AuNp administered slices.

Results: DLS, TEM and AMF analysis showed that hydrodynamic size of AuNPs was around 5nm, their shapes are sphere-like and their surface modifications were successful. Mean amplitude of spontaneous action potentials was significantly decreased after administration of AuNps (Control: n=20, 50.2±3.9mV vs AuNp: n=11, 34.4±4.6mV, $p<0.05$). There was no difference after administration of PEG-AuNp compared to control (n=5, 42.1±6.4 mV). Similarly, firing rate of action potentials (peak/second) was significantly increased in AuNp group, while there was no difference in PEG-AuNp group compared to control (9.6±1.7peak/sec, 6.9±1.7peak/sec; 4.6±1.7peak/sec; $p<0.05$). Properties of evoked action potentials were determined by current-voltage (I–V) analysis. Both AuNps and PEG-AuNps decreased the mean voltage at low current intensity [(-150 pA) - 0 pA, $p<0.05$]. Increase in mean voltage amplitude was determined in AuNP and PEG-AuNP groups compared to control at 0–150 pA.

Conclusion: In this study the properties of spontaneous action potentials recorded from hippocampal brain slices were affected by AuNps but not PEG modified AuNps. The possible effects of AuNps on neurons should be considered in medical applications and biocompatible modifications of AuNps might be determined. Supported by TUBITAK (215S052)

Keywords: electrophysiology, patch clamp, gold nanoparticles, polyethyleneglycol

O-23

The effectivness of spreading depression wave on long term potentiaion magnitude on dentate gyrus

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Objective: Spreading depression (SD) is a pronounced self-propagating depolarization of neurons and glia with a transient massive redistribution of ions between intracellular and extracellular compartments. In the brain, spreading depression (SD) is characterized by a large extracellular DC shift, a massive failure of ion homeostasis and a transient cessation of neuronal function. In this study we aimed to find a relationship between SD wave which sometimes happen in hippocampus and hippocampal long-term potentiation (LTP) magnitude.

Methods: LTP was induced using high-frequency stimulation (HFS; 100Hz, 1 sec, 4 times) at the perforant patway – dentate gyrus synapses in anesthetized male rats aged 2–3 mo. A bipolar tungsten electrode was used to stimulate the medial perforant path (PP, from bregma, in mm: anteroposterior: 6.5; mediolateral: 3.8; dorsoventral: 2–2.5 below the dura) of the

right hemisphere. A double-barrel glass micropipette was inserted into the granule cell layer of the DG in the right hemisphere (in mm, from bregma: anteroposterior: 3.0; mediolateral: 2.13; dorsoventral: 2.5–3 mm below the dura) to record the field potential. After recording, hippocampii were extracted and some MAPKs proteins were measured using western bolting technique.

Results: HFS induced a transient depression of field potentials in nine of healthy rats (depressed rats), but remaining rats (non-depressed) showed an obvious potentiation of field potentials. The magnitude of LTP at the end of recording was higher in non-depressed rats compared to that in depressed rats ($p<0.05$). Total and phosphorylated levels of P38 MAPKs were found to be higher in depressed rats ($p<0.05$).

Conclusion: Our results showed that SD wave induced by HFS can be related to increase in activation of p38 MAPKs. This work was supported by the Scientific Research and Project Unit (TDK-2016-6628).

Keywords: cortical spreading depression, MAPK, ionic distribution, long term potentiation

O-24

Spontaneous synaptic transmission in the entorhinal cortex of BDNF heterozygous mice

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Objective: In the central nervous system, Brain Derived Neurotropic Factor (BDNF), promotes the morphological and functional maturation of synapses. BDNF has differential effects on excitatory and inhibitory synaptic transmission. The aim of this study was to characterize the effects of reduced levels of BDNF on spontaneous excitatory (glutamatergic) and spontaneous inhibitory (GABAergic) currents in the entorhinal cortex.

Methods: To address the physiological effects of BDNF, we used BDNF heterozygous mice which exhibits reduced BDNF concentrations in the cortex. Transvers entorhinal cortex – hippocampus acute brain slices of 24–30 days old mice were used for patch clamp recordings. Under voltage clamp whole-cell mode, spontaneous Excitatory Postsynaptic Currents (sEPSCs) and spontaneous Inhibitory Postsynaptic Currents (sIPSCs) were obtained from layer 2/3 pyramidal neurons of entorhinal cortex. The amplitudes and the frequencies of spontaneous currents were compared between heterozygous mice and wild type litters.

Results: In the entorhinal cortex of heterozygous mice, the amplitude of sEPSCs was similar to wild type but the frequency of sEPSCs were significantly smaller. The effects of reduced levels of BDNF on inhibitory synaptic transmission were robust. Both the amplitude and frequency of sIPSCs were suppressed in heterozygous mice. As a result the excitatory/inhibitory balance was significantly higher in heterozygous mice in the favor of excitation.

Conclusion: Reduced levels of BDNF in the cortex effects both, excitatory and inhibitory neurotransmission in the entorhinal cortex. Effects of reduced BDNF levels on excitatory system were confined to presynaptic release and modest. Yet, the changes were more prominent in the inhibitory system and which involves both pre- and post-synaptic changes. Recordings from the same neuron also revealed an altered excitation/inhibition balance in heterozygous mice. This study was supported by TUBITAK (115S149)

Keywords: BDNF, entorhinal cortex, synaptic transmission, patch-clamp

O-25

Investigating the protective effects of NAD+ precursors against neurotoxicity

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Objective: Nicotinamide adenine dinucleotide (NAD) is a dinucleotide metabolite which serves as a rate-limiting co-substrate for the sirtuin enzymes, NAD+-dependent protein lysine deacetylases in all living cells. In addition to nicotinamide (NAM), nicotinic acid (NA), and nicotinamide mononucleotide (NMN), nicotinamide riboside (NR) which is a pyridine-nucleoside form of vitamin B3 also acts as a precursor of NAD+. Supporting data have been accumulated to indicate the important roles of NAD+ and its precursors in protecting cells against oxidative and chemical stress and preventing cell degeneration.

Methods: In our study, we wanted to compare NAD+ precursors in terms of their capacity to increase intracellular NAD+ levels and to protect neurons against toxicity. We cultured SHSY neuroblastoma and PC12 cell lines and exposed them to chemical stress by treatment with manganese(II) chloride (MnCl2).

Results: NAM treatment was not able to protect the cells against MnCl2 toxicity. High doses of NAM become toxic to the cells itself, most likely due to inhibition of sirtuin activity in the cell.

Conclusion: We plan to include additional in vitro cell toxicity assays (treatment with 6-OHDA, amyloid , fragment, etc.) and compare the extent of beneficial effects of other NAD+ precursors than NAM. Most interesting of all, nicotinamide riboside has been widely studied in neurodegenerative disease models and can be a potential and valuable tool to fight against these diseases.

Keywords: NAD, neuroprotection, nicotinamide

O-26

Neurite inhibition in neurons differentiated from stem cell as a modified method for neurotoxicity screening test

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Objective: Neurotoxicity Screening Test is an important method by which medical and industrial products can be analyzed through neurite inhibition. In the classical method, neuron differentiation is achieved in mouse NB2a neuroblastoma cells and maximum neurite outgrowth is obtained. Test which is a marker of moderate toxic effect, provides an idea of the neurotoxic effect of these agents in the clinical use. It is known that statins cause peripheral neuropathy and their effect can be revealed in culture medium. In this study, neurons differentiated from stem cells and NB2a cells were compared using neurotoxicity screening test. NB2a cells were differentiated into neurons in dibutyryl cyclic-AMP containing serum-free media. Adipose tissue-derived mesenchymal stem cells were differentiated into neurons in the presence of fibroblast growth factor and epidermal growth factor. Neuronal differentiation was characterized by glial fibrillary acidic protein, nestin, oligodendrocyte-4 and tubulin. Cell viability and proliferation were analyzed by 3-(4.5-dimethylthiazol-2-yl)-2.5-diphenyltetrazolium bromide test. 24 hours after neuronal differentiation, neurons were treated with a high dose of 100 µM statin, which reflects the clinical condition. After application, neurons were stained with Commasie blue to make neurites visible. Neurite outgrowth and inhibition per neuron observed in the microscopic area were determined as percent inhibition by software-mediated measurements.

Results: It was observed that cell proliferation and fragmentation decreased in both NB2a and stem cell-derived neurons. Similarly, high statin concentration have been shown to significantly inhibit neurite outgrowth. However, these inhibitions were found to be more prominent in stem cell-derived neurons.

Conclusion: Neurotoxicity Screening Test is an important method to give an idea of the possibility of harming health in human and related medical and industrial products. The use of the mesenchymal stem cell instead of cell line as a modification in this method has made an important contribution to increase the sensitivity of the test. This was thought to be important in products that affect human quality of life.

Keywords: neurotoxicity screening test, mouse neuroblastoma cell line, mesenchymal stem cell, neuronal differentiation, in vitro

O-27

Effects of stem cell and niche on apoptotic cell death of retinal ganglion after axotomy

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Objective: Damage in the adult mammalian central nervous system causes apoptosis of cells through irreversible degeneration. Cell death has been shown to be reduced by the addition of the protein synthesis inhibitor cycloheximide. Endothelial cells that do not reach target retinal ganglion cells die from apoptosis, especially growth factors that maintain survival in spite of not disturbing the blood vessels in the optic nerve axotomy, especially without causing retinal artery injury. The contributions of stem cells and niche to healing in damaged tissues are known, and this property is also seen for nerve cells. In this study, after optic nerve axotomy by applying stem cells and niches to the damage region, their effects on apoptosis in retinal ganglion cells were investigated.

Methods: In male rats, the optic nerve was excised by incision made by pulling the eye sphere to the front, was cut through a pair of scissors and the wound in the eye was closed. Mesenchymal stem cells originating from adipose tissue and their conditioning culture media 24-h were used alone or in combination. After 1 week of application, the eyes dissected with optic nerves were fixed and were hardened in 30% sucrose solution. Histological sections were taken from optic nerve level. Sections were stained with TUNEL method for apoptosis. Differences between groups were analyzed statistically.

Results: While no apoptosis was observed in the control group, a significant apoptosis was observed in the axotomized groups. Stem cell, niche and co-use significantly reduced apoptosis.

Conclusion: The apoptosis reduction of retinal ganglion cells in optic nerve injury of stem cells and niches indicated that they may also be useful in central nervous system damage. It was thought that the mechanisms used by stem cells and niches would make their clinical use much more meaningful in nervous system diseases which are difficult to be understood and improved.

Keywords: axotomy, optic nerve, apoptosis, stem cell, niche, retinal ganglion cells

O-28

Layer-specific distribution of muscarinic receptor M2 in the sensorimotor areas of rat brain

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Objective: We aim to understand attentional modulation in the sensorimotor areas of the rat cortex. This study focuses on the distribution of M2 in the barrel field (S1BF) and hindpaw

representation of SI cortex (S1HL), and primary motor cortex (M1). The results will be useful for computational models of vibrotactile neurons in the brain.

Methods: Coronal sections (thickness: 50 µm) were obtained from seven Wistar rats. Layer thicknesses and number of cells were determined on slides stained with ethidium bromide (λp(exc): 526 nm, λp(em): 605 nm). M2 receptors were localized by a monoclonal antibody. For fluorescence imaging, Alexa Fluor 594 conjugated to a secondary antibody was used. Receptor complexes were manually counted, and statistical analyses were performed on three dependent variables: average number of M2 receptor complexes in a layer (N), average number of M2 receptor complexes normalized with layer thickess (D), and average number of M2 receptor complexes per total number of cells in a layer ©.

Results: Two-way ANOVA showed significant main effects of layer on N, D, and C ($p<0.001$, $p=0.002$, $p=0.053$, respectively). Area and layer interaction was found for N ($p<0.001$). However, area did not have a main effect on all three variables. The number of M2 receptor complexes was highest in layer V for M1, S1HL and in layer VI for S1BF. Highest M2 receptor density was observed in layer II and III for all tested areas. Thicker layers had relatively more number of M2 receptor complexes. Furthermore, layers II and III had the densest staining. There was no difference among the three cortical areas, which is consistent with the cholinergic innervation in the brain.

Conclusion: Our previous data showed that atropine had stronger effects on single neurons in deep layers. According to the current results, supragranular layers seem to have more widely distributed, but weaker muscarinic inputs.

Keywords: somatosensory, attention, cortex, immunofluorescence, cholinergic

O-29

Bioinformatic analysis of transcriptome data to identify perturbed metabolic pathways of Alzheimer's disease and Parkinson's disease

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Objective: Mapping of transcriptome data on cellular networks is important to document pathway-level molecular effects of diseases.

Methods: To this aim, transcriptome data belonging to the postmortem brain tissues of 11 Alzheimer's Disease (AD) and 12 Parkinson's Disease (PD) patients were obtained from the online Gene Expression Omnibus Database. The data were computationally integrated with the genome-scale human metabolic network with 143 pathways involved in 2051 unique reactions controlled by 2535 genes, derived from HumanCyc Database.

Results: The applied bioinformatic method scores each metabolic pathway based on a novel algorithm which better represents the cross-talk among different pathways, enabling a more global and realistic cataloguing of network-wide perturbation effects in response to diseases. The algorithm computes a list of significantly affected metabolic pathways for each disease. Of 143 pathways considered, several pathways were found to be affected by AD and PD. The pathways which were perturbed in only one of the diseases and in both diseases were separately analyzed to point to commonalities in the considered neurodegenerative diseases.

Conclusion: Among the identified pathways are heparin sulphate, retinol and triacylglycerol biosynthesis, mevalonate, sphingosine and 3-phosphoinositide pathways, which are all reported to be disease-related in the literature. This research was financially supported by TUBITAK (Grant Number: 305S302).

Keywords: metabolic pathways, transcriptome, neurodegenerative diseases

O-30

Investigation of expressions of certain microRNAs in patients with Alzheimer's disease

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Objective: Alzheimer's disease (AD) that is a progressive neurodegenerative disorder is the most common dementia among the elderly. Recently, accumulating cases have demonstrated a direct link between microRNAs (miRNAs) and AD. miRNAs regulating post-transcriptionally gene silencing are short, single-stranded RNA molecules approximately 22 nucleotides in length. It was aimed to determine the expression levels of some miRNAs predicted to contribute to AD progression and clarify the relationship between AD and their expression profiles.

Methods: The blood samples derived from 84 AD patients and 67 control subjects were composed and total RNA isolation was performed by Trizol Reagent method from whole blood. The expression levels of selected miRNAs (hsa-miR-132–3p, hsa-miR-186–5p, hsa-miR-195–5p, hsa-miR-219a–5p, hsa-miR-3163 and hsa-miR-3613–3p) were analyzed by using comparative ΔΔCT method in Real-Time PCR. Data obtained from molecular analyze were statistically evaluated.

Results: It was identified that hsa-miR-186–5p were markedly downregulated in AD patients, while hsa-miR-3163 and hsa-miR-3613-3p were significantly upregulated in AD patients compared to controls ($p<0.05$). However, no significant differences in expression levels of hsa-miR-132–3p, hsa-miR-195–5p and hsa-miR-219a–5p were observed between groups ($p>0.05$).

Conclusion: It has been indicated that altered expression levels of hsa-miR-186–5p, hsa-miR-3163 and hsa-miR-3613–3p

may contribute to AD-related neurodegeneration. These miRNAs might use as bioinformative molecules and play a part among potential biomarkers in AD pathogenesis and so might help diagnostic and therapeutical progress.

Keywords: Alzheimer's disease, miRNA, expression

O-31

Detection and characterisation of the breast milk induced cells in brain

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Objective: Breast milk is an indispensable food source which contains all the energy and nutrients necessary for growth and development of infants from birth. There are different types of cells in breast milk as well as a variety of carbohydrates, fat and protein. In our study the passage to the nervous system of baby through breast-feeding and differentiation properties of these cells are investigated.

Methods: With the purpose of investigation of the passage of cells in breast milk to infant, transgenic mice with the ability of producing green fluorescent protein in all cells were used as mothers. On normal type mice breast fed by these mice, the cells producing green fluorescent protein that were passed from milk through breast-feeding were investigated by microscopic examination, flow cytometry and quantitative polymerase chain reaction techniques.

Results: At the end of the examinations with various waiting times after breast-feeding, it was seen that the cells passed from the mother through breast-feeding entered the bloodstream of the baby by passing through the digestive system. It was determined that these cells were passed from the bloodstream to brain tissue of the baby. It was seen that these cells detected in brain tissue differentiated into neurons and glia cells which express NeuN and GFAP.

Conclusion: As a result of our study, it has been demonstrated that maternal cells could pass from breast milk to brain tissue of the baby through breast-feeding. It was determined that these cells are capable to differentiate to the cells of brain tissue. Through further studies, the identification of the relationships of these cells with physiological and pathological processes on the baby will be an important step in this area. This project was supported by TUBITAK (The Scientific and Technological Research Council of Turkey, Project No: 114R078).

Keywords: breast milk, brain, microchimerism

O-32

Interpretation of differently localized neuroendocrine tumors by samples

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Objective: In this study, it was aimed to discuss neuroendocrine tumor (NET) and related syndromes on three cases, where localized to stomach and appendix.

Methods: In our study, three cases who underwent surgical intervention and diagnosed NET were investigated.

Results: Three patients, who were 23, 47 and 71-years-old women, came to the policlinic due to abdominal pain. A 23-year-old female patient was operated because of a lesion at the size of 135×85×70 mm on the ovarium. During the operation, a prophylactic appendectomy was performed for the suspicion of appendiceal tumor. The patient's AFP is low; CA 19–9, LDL, cholesterol levels were high. Attention was drawn to the fact that the mass lesion located in the ovary was mature cystic teratoma. In apendiks surprisingly noticed the presence of neuroendocrine tumors. A 47-year-old female patient underwent hysterectomy due to uterine multiple myomas. At the time of operation, the swallowed appearance of the appendix was noticed. For this reason, appendectomy was added to surgery. The diagnosis of a neuroendocrine tumor of the appendix was established. He noted that the patient had thyroidectomy due to hyperthroid and the glucose level was high. The patient had thyroidectomy due to hyperthyroidism and the high level of glucose was noticed. A 71-year-old female patient with abdominal pain was diagnosed with a neuroendocrine tumor grade 1 by endoscopic biopsy. Subsequently, the patient underwent subtotal gastrectomy. Gastrectomy had multiple foci on the corpus and 3 cm hyperplastic polyp on the antrum. It is noticed that Vitamin B12 low, iron binding capacity, glucose, VLDL, TG, cholesterol, BUN, free T4 levels were high in the biochemical results of the patient.

Conclusion: Neuroendocrine tumors may show very different clinical behavior in terms of location, diameter, cellular characteristics and hormonal activity. Handling with all parameters will be the right approach.

Keywords: hysterectomy, gastrectomy, appendectomy, neuroendocrine tumor

O-33

Effects of nesfatin-1 on necrotizing enterocolitis-induced brain damage in newborn rats

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Objective: Necrotizing enterocolitis (NEC) is an acute gastrointestinal disorder leading to neurodevelopmental disorders. Nesfatin-1 has demonstrated anti-inflammatory, anti-apoptotic and neuroprotective effects on oxidative brain damage due to subarachnoid hemorrhage. This study was aimed to investigate antioxidant and neuroprotective effects of nesfatin-1 on NEC-induced newborn rats.

Methods: Following their births, Sprague Dawley pups were separated from mothers, fed 3 times/day orogastrically with a hyperosmolar formula (Esbilac+Similac), and injected with saline (n=21) or nesfatin-1 (0.2 µg/kg/day; n=20) for 3 days. On 3rd day, pups were exposed to hypoxic chamber (5% O_2+95% N_2) for 45 sec. Control group (n=16) was left with their mothers. On the 4th day, after the scoring of clinical disease (general appearance, activity, response to touch, body color) newborn rats were decapitated and gut was macroscopically scored. Brain levels of malondialdehyde (MDA; indicating lipid peroxidation), antioxidant glutathione (GSH) and myeloperoxidase activity (MPO; showing neutrophil infiltration) were measured. Data were analyzed by ANOVA and Student's t-test.

Results: Mortality rate in nesfatin-1 treated NEC group (10%) was lower than that in saline-treated NEC group (23%). Clinical sickness and macroscopic intestinal scores were higher in saline-treated NEC group compared to control group ($p<0.001$); while both scores were decreased in nesfatin-1-treated NEC group ($p<0.001$). In response to colonic inflammation, MDA level and MPO activity were elevated in brain tissues of saline-treated NEC group. In nesfatin-1 treated NEC group, elevated MPO activity in brain tissue was decreased ($p<0.05$), but decline in MDA level was not significant. When compared with control group, brain GSH content was increased in both NEC groups ($p<0.01$).

Conclusion: In neonatal NEC model, nesfatin-1 reduced mortality, improved intestinal damage and clinical appearance, and alleviated NEC-induced oxidative brain damage by inhibiting neutrophil infiltration. Further research is required to elucidate the impact of this neuroprotective effect on long-term cognitive functions.

Keywords: necrotizing enterocolitis, nesfatin-1, oxidative stress.

O-34

Effect of different glutamate doses on NMDR subgroups in hypothalamic-prefrontal pathways in subchronic dose of ketamin-induced NMDAR hypofunction model

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Objective: N-methyl-D-aspartate receptor (NMDAR) hypofunction model created with subchronic dose of ketamine is widely used in schizophrenia studies. It is expressed in the model; NMDAR ion channel is blocked by ketamine and undergoes receptor hypofunction. There is not sufficient information about how receptor hypofunctions, is that with the increase in expression levels, is it by selecting different subunits with different affinity or is the receptor hypofunction created with ketamine relevant steric effects or structure which is composed of different subunits?

Methods: Clarification the molecular mechanisms of disease, primarily in controlling the symptoms, in advancing the process, we think that for the benefit of early diagnosis. For this reason, to examine the dynamics of the disease process, we designed the animal experiments in our study; we created a model based on the NMDAR hypofunction in 6 week old Wistar male rats. We did isotype comparison and determined the expression levels by Western Blotting method in groups: subchronic doses of ketamine, two different doses of L-glutamate, and subchronic dose of ketamine with two different doses of L-glutamate.

Results: We evaluated the statistical significance of the data SPSS 20 program. NMDAR responded with subunit exchange against hypofunction. This effect has not been shown on any sub-units in modeling tissue hippocampus, it suggests hippocampus regeneration ability. Different doses of glutamate in schizo-mimetic models, NR2A subunit with decreased caused neurodevelopmental-transformation and it is caused to take place different from NR2A/B sub-units. Full agonist of the NMDAR glutamate showed the effects with changing receptor kinetics.

Conclusion: It is possible to say glutamate application in healthy models and models of schizophrenia can change the intensity and direction of impact and also support different neuro-biochemical processes in sickness and in health.

Keywords: NMDAR, ketamine, glutamate, hippocampus, prefrontal cortex, schizophrenia

O-35

A2A receptor dependent hypoxic ventilatory response in rats exposed to acute intermittent isocapnic and chronic sustained hypoxia

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Objective: Intermittent hypoxia (IH) elicits long-term facilitation (LTF) known as a persistent augmentation of respiratory motor output and ventilatory acclimatization to hypoxia (VAH) occurs during chronic sustained hypoxia (CSH) due to augmentation of hypoxic ventilatory response that both two mechanisms cause ventilatory neural plasticity. The pathways leading to LTF activated by protein coupled metabotropic recep-

tors (Gq and Gs) as serotonin and adenosine-dependent. We tested the hypothesis that adenozin 2A receptor activation which is induced by acute intermittent isocapnic hypoxia (AIIH) have a main role in constitution of VAH.

Methods: In our study, three experimental groups were formed using Sprague Dawley, male, 3–4 months old rats. Normoxic group is kept in normoxic conditions. Ventilation (V), respiratory frequency (fR) and tidal volume (Vt) of the group, which is exposed to AIIH (FIO_2= 10, $FICO_2$= 4), is measured by using Whole Body Plethysmography (WBP). Arterial blood gas is measured to determine the intensity of hypoxia. CSH group has exposed to hypobaric hypoxic conditions (380 mmHg, FIO_2= 10) for a week. Each group's brain stem and spinal cord sections are used for immunofluorescence staining method for 5-HT2A and A2A receptors and BDNF proteins. $p<0.05$ significance level is accepted for statistical analysis.

Results: In AIIH protocol (n=11), preAAİH60' basal fR, Vt and V values compare to postAAİH6o' there is no significance ($p > 0.05$) results for vLTF. According to CSH results, in brain stem and spinal cord sections, AIIH groups' 5-HT2AR and BDNF immunoreactivity is higher than A2AR ($p<0.05$).

Conclusion: Results show that Gq pathway, which activated after moderate AIIH, causes constitution of pLTF but there is no evidence that causes constitution of vLTF. VAH formation was similar to plasticity thought to be due to A2AR activation. Understanding these mechanisms contributes to prove new methodes on treatments of respiratory failure or motor dysfunctions.

Keywords: adenosine, hypoxia, hypercapia, serotonin, ventilatory neuroplasticity

O-36

Pericytes in brain and retinal capillaries express alpha-smooth muscle actin, which can be down regulated by small interfering RNA

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Objective: Pericytes, which encircle capillaries in the brain and retina, are crucial for regulation of microcirculatory blood flow by their contractile properties. Alpha-smooth muscle actin (a-SMA) present in large arteries and arterioles play a central role in vascular smooth muscle contraction, and may be a strong candidate for contractile response of pericytes, which regulate blood flow at microcirculation. We aimed to detect expression of a-SMA in pericytes by immunohistochemistry using pericyte-specific markers and by knocking down a-SMA expression via small interfering RNAs in mice brain and retina.

Methods: With a novelimmunohistochemical method a-SMA protein was detected even in the smallest capillaries of retina and brain of adult mice. a-SMA protein was co-labeled with 3 different pericyte specific markers; neural/glial antigen 2 (NG2), platelet-derived growth factor receptor beta (PDGFR-beta) and CD13 to show a-SMA existence in cerebral and retinal pericytes. a-SMA gene targeted custom and scrambled siRNAs were designed. Intravitreal injection to retina and intracerebroventricular injection to brain with a cannula via the specific in vivo transfection reagents were used for siRNA delivery. The down-regulation of a-SMA gene expression after siRNA delivery was validated with both quantitative RT-PCR and immunohistochemistry.

Results: We showed co-localization of a-SMA protein with NG2, PDGFR-beta and CD13 proteins in cerebral and retinal pericytes surrounding microvessels. a-SMA gene expression was significantly down regulated in retina and brain of siRNA injected mice compared to scrambled siRNA injected mice.

Conclusion: We detected a-SMA expression in cerebral and retinal pericytes around capillaries. The expression of a-SMA gene was successfully down regulated with specific siRNA, confirming that pericytes did indeed express a-SMA as suggested by immunohistochemistry. a-SMA may play a role in pericyte contraction. The Scientific and Technological Research Council of Turkey (TÜBİTAK) project no:114S190 (M.Y.) and 7th Framework Programme EU Marie Curie Actions (L.A.M.) – Co-funded Brain Circulation Scheme, TÜBİTAK

Keywords: alpha smooth muscle actin, siRNA, pericyte

O-37

Cross-talk inhibition between 5-HT7 and A2A receptors depends on severity of acute intermittent hypoxia

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Objective: Recent studies show that long term facilitation (LTF) of phrenic nerve activity with acute intermittent hypoxia (AIH) involves two signaling pathways. In addition to the well-known serotonergic (Gq) pathway, a Gs pathway can be activated by adenosine receptor 2A (A2AR) or serotonin receptor 7 (5-HT7R) cause LTF. Gs pathway can inhibit the Gq pathway and increase LTF by causing a cross-talk inhibition between A2AR / 5-HT7R and 5-HT2AR's. We tested the hypothesis that Gs pathway contributes to plasticity in ventilatory control during severe acute intermittent hypoxia (sAIH) dependent on A2AR activation in adult male rats.

Methods: In this study we used two experimental groups. Ventilation measured in adult, awake, unrestrained rats with

whole body plethysmography (WBP) by exposing moderate (FIO_2=10) and/or severe (FIO_2=7) AIH, which was five 5-min exposures to FIO_2= 0.10 or 0.07 interspersed with four 5-min exposures to FIO_2= 0.21. Staining was performed by incubating tissue sections (30 µ) with anti 5-HT7 and anti A2A on spinal cord (C3-C5). For statistical analysis $p<0.05$ significance level was adopted.

Results: In mAIH group (n=8) ventilation (Vi) was increased comparing m_preAIH60 (207.6±46.22) and m_postAIH90 (240.2±38.71). Also in sAIH group (n=10) ventilation was increased comparing s_preAIH60 (194.9±35.41) and s_postAIH90 (232.00±37.35). These results show that both moderate and severe AIH enhance ventilation and cause LTF. In evidence that immunoreactivity of A2AR activation found higher than 5-HT7R activation on spinal cord in sAIH group. In contrast immunoreactivity of 5-HT7R activation found higher than A2AR activation in mAIH group.

Conclusion: The results support a role for Gs in the increased ventilatory drive and hypoxic responses with severe AIH.

Keywords: serotonin, adenosine, long term facilitation, acute intermittent hypoxia

O-38

Changes in miRNA levels of rats in different environments after experimental subarachnoid hemorrhage

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Objective: Nowadays, patients with subarachnoid hemorrhage (SAH) are kept in isolated environments. Studies show that environmental conditions have effects on post-SAH recovery. This study aimed to observe the effects of different environmental conditions on the levels of some miRNAs that may be associated with cognitive dysfunction after SAH in the frontal lobes of rats.

Methods: Adult female Sprague-Dawley rats (n=21) are divided into 3 groups: control group (n=7), post-SAH isolated environment group (n=7) and post-SAH enriched environment (n=7). Experimental subarachnoid hemorrhage was created by cisterna magna double injection method. The control group was housed in standard rat cages without SAH formation while others were in enriched and isolated environment cages for 14 days after SAH model was constructed. The levels of miR-132, miR-134 and miR-138 in the left frontal lobe tissues obtained after craniectomy and dissection were determined by Real-Time PCR and statistical differences were calculated using one-way ANOVA test.

Results: There was a statistically significant increase of miR-132, miR-134 and miR-138 levels in the frontal lobes of the rats that were housed in enriched environment after SAH ($p<0.001$). The levels of miR-132, miR-134 and miR-138 in the frontal lobes of the animals kept in the isolated environment after SAH were not statistically different compare to the control group ($p>0.05$).

Conclusion: The observation of the miRNA levels in the frontal lobes of animals from the enriched environment conditions after SAH being elevated and expression levels being higher than those in isolated environment suggests that the expression of these miRNAs in the frontal lobes may be affected by different environmental conditions. These increases in miRNA expressions may be the result of synaptic remodeling process.. Further studies to support these results have major implications.

Keywords: enriched environment, isolated environment, miR-132, miR-134, miR-138, SAH

O-39

Interpretation of motor overflow in children with attention deficit hyperactivity disorder in relation to cortical functional couplings

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Objective: Impairments in neural networks related to motor, sensory and cognitive functions, and neuronal deficits in attention and executive function networks are found in children and adolescents with attention deficit hyperactivity disorder (ADHD). Structural and functional anomalies in the brain have been associated with impaired cognitive, affective and motor behaviors in ADHD. Even though repetitive motor task related motor overflow is significantly decreased among healthy school aged children, is frequently continued among children with ADHD.

Methods: In this study, 30.8–12 years old, right-handed children (15 ADHD, 15 age and sex matched healthy control) with a total IQ score of 75 or more on Wechsler Children's Intelligence Scale for Children (WISC-IV) were enrolled. They were asked to move for at least 30 seconds and then rest for at least 30 seconds. EMG of bilateral forearm extensor muscles and 20 channel EEG data were acquired in a separate session for each hand. Coherence values were calculated to obtain cortical coupling measures. In order to access motor overflow parameters the ratio of the mean rectified EMG data during rest to movement was calculated.

Results: The motor overflow in both groups was higher during the non-dominant hand compared to dominant hand movements. This difference was statistically significant in the ADHD group. We observed that intra- and interhemispheric cortical functional couplings were higher during non-dominant hand movement compared to dominant hand in both groups in both alpha and beta frequency bands. We also found that in the beta-band, interhemispheric coupling between primary motor cortices was decreased in healthy controls while increased in ADHD group.

Conclusion: Asymmetric development of callosal inhibition can be a possible candidate for the explanation of the increase in motor overflow during non-dominant hand movement. The increase in interhemispheric coupling between primary motor cortices might be an indication of decreased in transcallosal inhibition in ADHD.

Keywords: ADHD, EEG, motor overflow, coherens, callosal inhibition

O-40

The impact of attention deficit hyperactivity symptoms on mindfulness

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Objective: Mindfulness involves concentrated attention to present experience. Mindfulness has been found to be affected by emotional states. A better understanding of what factors are associated with mindfulness would be useful in exploring the efficacy of mindfulness-based therapies. We hypothesized that besides depression, anxiety and stress levels, attention deficit hyperactivity disorder (ADHD) symptoms would negatively correlate with the level of mindfulness since attention has the primary role in both ADHD and mindfulness. In order to test this hypothesis, the aim of this study is to investigate the association between mindfulness and attention deficit hyperactivity symptoms.

Methods: Seventy-six subjects with a mean age of 29.61 years who applied to the outpatient unit Acibadem Maslak Hospital, Psychiatry Department, Istanbul for the first time participated in this cross-sectional study. The control group consisted of 32 healthy gender and age matched participants. Mindful Attention Awareness Scale, Depression Anxiety Stress Scale, Adult ADHD Self-Report Scale, Wender Utah Rating Scale were given to the participants. A probability level of $p<0.05$ was used to indicate statistical significance. Independent sample t test was used to compare the mean scores between groups. Categorical data was analyzed by chi-square test. In order to explore whether ADHD symptoms, depression, anxiety, stress, age and sex were related to mindfulness levels, linear regression analyses were used. SPSS 16 Programme for Windows was used for statistical analysis.

Results: Our findings showed that childhood ADHD symptoms, depression levels, and adult attention deficit symptoms significantly predicted low levels of mindfulness. The results also demonstrated that besides emotional symptoms such as depression, anxiety and stress, ADHD symptoms also had a significantly negative impact on mindfulness.

Conclusion: The results of this study indicated that there is a significant negative relationship between ADHD and mindfulness. Improving mindfulness through practice offers a novel approach in multimodal treatment of ADHD.

Keywords: attention, anxiety, depression, hyperactivity, mindfullness

O-41

Investigating the relationships between the nomophobia, attachment characteristics, mental and behavioral problems on a group of adolescents in Istanbul

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Objective: The aim of this study is to investigate the relationships between the nomophobia, attachment characteristics and mental and behavioral problems in adolescents.

Methods: This research is conducted on 844 adolescents (n=441 female, and n=393 male) obtained from 3 schools in Istanbul. The short form of Inventory of Parent and Peer Attachment (s-IPPA), nomophobia scale, and strengths and difficulties questionnaire (SDQ) self report and parental versions were used in this study. Participants who scored ≥60 on nomophobia scale (moderate or severe nomophobia) are considered as nomophobia group, and ≤60 (mild nomophobia or absent) considered as control group. The categorical variables were analyzed by chi square test. The comparison of the scale scores was done by independent sample t test. Logistic regression analysis was used to determine if there is a significant relationship between the nomophobia and SDQ, s-IPPA scores and sociodemographic data. The statistical analysis was performed with SPSS 16 for Windows Programme and statistical significance level was set at $p<0.05$.

Results: The results indicated that 63.6% of the adolescents were experiencing moderate or severe nomophobia. The prevalence of the moderate or severe nomophobia in girls (73.7%) was significantly higher than boys (%55.6). Both mother and father attachment scores were significantly lower in nomophobia group compared to the controls. Hyperactivity, emotional and behavioral problems subscale scores and total problem scores of the self-report SDQ were significantly higher in nomophobia group. The results of the logistic regression analysis suggest that s-IPPA and SDQ scores significantly influence the probability of the nomophobia development in adolescents.

Conclusion: The attachment level shows the quality of the relationship between the child and caregiver. The results suggest that the prevalence of the insecure attachment are higher, and mental and behavioral problems are lower in adolescents with nomophobia compared to their non-nomophobic peers.

Keywords: adolescent, attachment, behavior, nomophobia

O-42

The modulation of prepulse inhibition response with orexin A on a sleep deprivation model

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Objective: Orexin is a peptidergic neurotransmitter released from lateral hypothalamus. In addition to playing a key role in sleep / wakefulness system, it also plays important roles on regulation of eating behavior, mood, reward system and energy homeostasis. In recent years, it became a current issue that orexin may have a relation with the positive and negative symptoms of schizophrenia and this neuropeptide can be examined as a therapeutic target.

Methods: In our study, the role of orexin A, that pass through blood-brain barrier, is investigated on the prepulse inhibition (PPI) response using 72 hour REM sleep deprivation (SD) rat psychosis model. The impact of 5 µg/kg (n=8), 10 µg/kg (n=8) and 20 µg/kg (n=8) orexin A injections on the impaired prepulse inhibition responses after sleep deprivation are compared with the rats that received saline injection (n=8).

Results: The analysis of the data with one-way ANOVA revealed that 20 µg/kg orexin A injection improved prepulse inhibition response to 78 dB prepulse intensity compared to the control group (p=0.01). Furthermore, a dose-dependent increase pattern has also been observed for all prepulse intensities.

Conclusion: These results indicate that orexin A may play a role in SD-related psychotic sypmtom neuroethology, however, detailed follow-up studies are still required.

Keywords: orexin A, sleep deprivation, prepulse inhibition reponse, psychosis, schizophrenia

O-43

Behavioral and molecular effects of different types of perinatal music exposure on rats

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Objective: Previous studies on music both on human and non-human animals showed that prenatal music have behavioral, cognitive, and molecular effects on newborn. The aim of the present study is to measure effects of perinatal music on behavioral parameters such as anxiety, motor-coordination, learning, memory, and depression on rats.

Methods: Mother Wistar albino rats (n=8) were divided into four music groups; rock (120 dB), classical (80 dB), and sufi (40 dB) music and they were exposed to music for one hour/day during pregnancy. After giving birth, music exposure continued together with pups till weaning period. At the weaning period and at the adult age, pups were applied to anxiety, motor coordination, depression, learning and memory tests, respectively. Animals were sacrificed hippocampus were removed. Corticosteron level, total oxidant status, total antioxidant status, oxidative stress index were analyzed by ELISA. Also, the hippocampal proteins whose expressional alterations determined by microarray analysis were validated with RT-PCR and Western blot. Mean values and standard deviations were compared with One-way ANOVA and LSD test using SPSS.

Results: Rock music group had decreased anxiety and depression level, and low level of learning and memory abilities ($p<0.05$). Also, rock music group had increased level of oxidative stress compared to other groups ($p<0.05$). Classical and sufi music groups had increased level of anxiety-like behaviors and depression, however increased level of learning and memory compared to control group. While sufi music group had high level of anxiety and depression, they had high performance in short term memory ability ($p<0.05$).

Conclusion: It was found that being subjected to different types of music is no related with motor coordination skills in rats. Furthermore, the expression of proteins like Klotho, transthyretin, Kjcn-1, BDNF, NGF, and synaptophysin were affected from different type perinatal music exposure. Exposing to different types of music has several effects on behavior in rats.

Keywords: anxiety, learning, memory, perinatal music, microarray, RT-PCR

O-44

Investigation of a marker for the quality of transcardial perfusion in frozen brain sections in mice

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Objective: The goal of fixation via transcardial perfusion is to preserve the tissue in a life-like state without allowing hypoxia / hypoperfusion-induced changes. Given that frozen sections are commonly preferred when using florescent-tagged antibodies, in co-localization studies or transgenic animals expressing florescent proteins, there is a need for markers of imperfect perfusion in frozen sections to ensure that the observed changes were not caused by non-optimal tissue fixation.

Methods: In this study, adult naive Swiss albino mice were anesthetized with chloral hydrate, followed by transcardial perfusion in different flow rates of heparinized saline and 4% paraformaldehyde (PFA). Extracted brains were prepared according to the type of immunostaining for Nissl, NeuN, Hoechst, YOYO-1, or HMGB1. Images were taken with fluorescent, phase-contrast, DIC and laser scanning confocal microscopes in cortical and subcortical regions of 7 perfect perfusion fixed brains of mice and 9 imperfect perfusion fixed brains of mice.

Results: There was significant increase in swollen 'donat-shaped' neurons in imperfect perfused brains in NeuN, Hoechst and HMGB1 ($p<0.05$). Astrocyte end feet swelling around dark neurons in cortical and subcortical regions of imperfect perfused brains were also increased in Nissl staining compared to perfect perfused one ($p<0.05$). These findings suggest that deformed 'donat-shaped' neurons in Hoechst, YOYO-1, NeuN and HMGB1 immunostaining can be a neurohistological marker of imperfect PFA perfusion in frozen sections. This result was further supported by the classical

histopathologic images by colocalizing astrocyte end feet swelling around dark neurons with donat-shaped neurons in phase-contrast and DIC confocal microscopes. Average quantity of the donat-shaped neurons in Hoechst was less than 5% in perfectly fixed PFA perfused mice.

Conclusion: his percentage may be considered as a threshold to recognize imperfect perfusion in Hoechst staining which is an easily applicable and widely used DNA marker.

Keywords: transcardial perfusion, paraformaldehyde fixation, astrocyte end feet, neuron, neurohistological marker, microscopy

O-45

Investigation of pathophysiological processes with advanced microscopy techniques after cerebral ischemia

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Objective: According to the data from World Health Organization, 15 million people suffer from stroke world wide and 5 million of these suffer from permanent handicaps while around 5 million people die as a result of stroke. With this study, identification of the plasticity following brain ischemia in a more detailed manner will lead to the development of new treatment options.

Methods: In this study, it is aimed to investigate the changes in the time-dependent development of plasticity following experimental brain ischemia with the use of advanced imaging and molecular techniques. To this end, in order to examine the changes in the axonal projection in mice subjected to brain ischemia, cellular structures in the motor cortex were targeted by GFP and tdTomato fluorescent protein expressing viral injection. The anterograde progression of the fluorescent vectors from cell soma through axons and the transition between hemispheres were monitored. Changes in the axonal projection following brain ischemia were identified in 3D.

Results: FN and RN are the assessment centers of plasticity in the brain and it has been reported that in the case of an injury axonal transitions occur from the contralateral region to the ipsilateral region. Clarity and subsequent advanced imaging are recently being used as one of the most effective methods for analysis of fluorescently labelled cells in 3 dimensional structures. For standard histological applications, to analyze a thick and opaque tissue, laser scanning microscopes can image at a maximum of 20-50 µm tissue depth. Using clarity techniques, laser permeability of not only whole brain tissue but also other whole organs can be increased and much deeper images of the tissue can be taken.

Conclusion: This study will contribute to the studies which aim to identify new pharmacologic target molecules and develop new drugs to promote brain plasticity following ischemia in humans.

Keywords: cerebral ischemia, plasticity, clarity, axonal projection

O-46

Effects of rottlerin and genistein on proliferation, invasion and cell death in neuroblastoma cells

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Objective: Neuroblastoma (NB) is the most common extracranial solid cancer in childhood and the most common cancer in infancy in the world. Tyrosine kinase signaling networks play a major role in governing cell differentiation, including in neuroblastoma. Rottlerin, a naturally occurring polyphenolic compound derived from *Mallotus philipinensis (Euphorbiaceae)*, appears to have great potential in cancer therapy because of its effects on several cellular processes such as proliferation and apoptosis. Genistein is a phytoestrogen and acts as tyrosine kinase inhibitor. Genistein have been found to inhibit the uncontrolled cell growth of cancer, most likely by inhibiting cell division and cell survival in several cancers such as prostate, cervix, brain and breast. After all of these knowledge, we investigated the effects of rottlerin and genistein on cell proliferation, invasion and cell death in neuroblastoma cells.

Methods: In this study, the human neuroblastoma cancer cell lines (SH-SY5Y, Kelly) were used. Rottlerin and Genistein were also employed for therapy. As in vitro experiments, cell proliferation, colony formation, invasion, wound-healing tests and apoptosis analysis by flow cytometry were performed.

Results: Our results showed that rottlerin and genistein treatments caused a significant reduction in cell proliferation, colony formation, invasion/wound-healing capacity in Neuroblastoma cells at concentrations of 5 µM and 30 µM, respectively ($p<0.001$). The combination of these doses also empowered the level of inhibition in these analysis ($p<0.0001$). Additionally, these drugs also increased the level of apoptosis in Neuroblastoma cell lines ($p<0.001$). Western blot analysis after these drug treatments are planning to perform for revealing related protein pathways.

Conclusion: The present results have shown that rottlerin and genistein have important effects on cell proliferation, metastasis and cell survival on neuroblastoma. Taken these in vitro findings together, treatment with rottlerin and genistein in combination may be a viable approach and beneficial to neuroblastoma patients.

Keywords: Genistein, invasion, neuroblastoma, proliferation, Rottlerin

O-47

Simulated noisy epileptic EEG signal source localization with minimum norm method

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Objective: Epilepsy is a neurological disorder that results in sudden and unusual changes in the electric potentials of some

nerve regions on the brain. Many patients heal with the help of the developed antiepileptic drugs and seizures can be reduced considerably. It is important to find epileptic foci before surgery for epileptic drug-resistant patients. Simulated EEG signals with AWGN are generated for active regions created by user and then locations of these regions are found with the inverse problem solution in this study.

Methods: Simulated data is obtained using Brainstorm software for ICBM152 headmodel in this study. A 64-channel Biosemi electrode set is selected. The BEM headmodel is used for forward problem solution. Two patches are placed on the brain cortex and EEG signals are computed at electrodes. Then WGN is added to the measurements and inverse problem solutions are obtained using Minimum Norm method in which sLORETA, dSPM and Current Density Map are used as measures.

Results: User selected patch zones are successfully obtained for EEG signals without noise as shown by MRI maps generated for the headmodel. Patch locations are placed outside the relevant regions for noisy EEG signals with SNR=1. Since epilepsy is expected to have an active cortical area of about 5 cm^2 to observe on electrodes, it is ensured that the size of the patches produced is at least 5 cm^2. Despite the noise, sLORETA points to the nearby region.

Conclusion: Epileptic EEG signals are computed by simulation and source localization success is tested for both signals without noise and signals with AWGN in this study. Noninvasive detection of the relevant region on EEG signals guide physicians in surgery. sLORETA metric locates foci from noisy signals successfully compared to other metrics as shown in the results. We thank Brainstorm's team for providing Brainstorm software (http://neuroimage.usc.edu/brainstorm).

Keywords: EEG, simulation, inverse problem solution, minimum norm

O-48

Thymoquinone increases the survival rate of peripheral sensory neurons on laser microdissection axotomy model

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Objective: Thymoquinone is the most effective bioactive molecule of Nigella sativa seeds. It has potent immunomodulator, antioxidant, anti-inflammatory and antiapoptotic effects.

Methods: Dorsal root ganglion neurons were isolated from adult Balb-C mice. Neurons were obtained by enzymatic and mechanical separation of ganglions. The cells were seeded petri dishes and incubated for 48 hours in the incubator. Then, Thymoquinone (TQ) was dissolved in DMSO, added to the experimental groups at 1000 nM and 750 nM concentrations. DMSO was added to the control group. An hour later, the axon cut was made in a laser microdissection system with UV laser unit equipment. For the cutting point, a distance of 150 µm from soma is calculated. The axons of 311 neurons were cut in three groups (1000 nM TQ: 105, 750 nM TQ: 105, control: 101). After axotomy, The petri dishes was transferred to the a computer controlled time-lapse microscopic system. Digital images were taken with a 40× objective every 5 minutes for 24 hours. Propidium iodide (7.5 µM) and Calcein AM (1 µM) were used to visualize dead and living neurons. The phase-contrast and fluorescence images of the cells were recorded before the axotomy and after the axotomy (24th h. and 48th h.)

Results: Thymoquinone increased Schwann cell proliferation significantly. After 48 hours, maximal neuron survival was determined in the group of 1000 nM TQ (45/105–43%), second in the group of 750 nM TQ (34/105–32%), third in the control group (23/101–22%). The neurons continued to extend the neurite after gone through the acute stress process. In the statistical comparison of the control group with 1000 nM TQ Group, the number of live neurons was found to be high at a significant level in the 1000 nM TQ group ($p<0.05$).

Conclusion: Thymoquinone increases the survival rate of peripheral sensory neurons in vitro. We believe that the this effect derives from Schwann cell proliferation and NGF excreted from Schwann cells.

Keywords: thymoquinone, dorsal root ganglion, neuron culture, axotomy, regeneration

O-49

The effects of salmon calcitonin on the calcitonin gene-related peptide levels and dural mast cells in glyceryltrinitrate induced migraine model and *ex-vivo* meningeal preparations in the rats

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Objective: Meninges and cerebral vessels are mainly innerved by calcitonin gene-related peptide (CGRP) containing trigeminal sensory nerves. CGRP released as a result of activation of trigeminal sensory nerves plays key role in pathophysiology of migraine by causing vasodilation of cerebral and meningeal vessels, nociception, also dural mast-cell degranulation (DMCD). It has been known that recently frequently studied salmon-calcitonin exhibits antinociceptive effects in various pain conditions. We aimed to investigate effects of salmon-calcitonin on CGRP levels and dural mast cells (DMCs) in glyceryltrinitrate (GTN)-induced migraine model and *ex-vivo* meningeal preparations in rats.

Methods: Forty-two male wistar-rats (8–10 weeks) were used in in-vivo and *ex-vivo* studies. In in-vivo groups, control (n=7) received vehicle (0.2 ml, i.p.), GTN group (n=7) glyceryltrinitrate (10 mg/kg, i.p.), SF+GTN group (n=7) saline (0.2 ml, i.p.) and GTN (10 mg/kg, i.p.), Calc+GTN group (n=7) salmon-calcitonin (50 µg/kg, i.p.) and GTN (10 mg/kg, i.p.) together, after four-

hours, blood samples and duramaters were taken. In *ex-vivo* meningeal preparations, skull cavities in first-group (n=7) were applied first artificial-cerebrospinal-fluid (ACSF,300 µl, control) then GTN (100 µM, 300 µl) for 15 min, second-group was applied first ACSF (300 µl, control) then salmon-calcitonin (50 µM) and GTN (100 µM) together (300 µl), respectively, and superfusates were collected. CGRP contents of plasma and superfusates were measured using enzyme-immunoassays. Duramater were stained with toluidine-blue and DMCs were observed. Data were analyzed with one-way ANOVA-Tukey using SPSS_17.0 software.

Results: In in-vivo groups, while GTN increased plasma CGRP levels (22.9±0.5 to 45.7±1.4 pg/ml, p<0.001) and DMCD (8.4± 1.0% to 28.9±4.3%, p<0.05), didn't change MCs number (p>0.05); in *ex-vivo* groups, while GTN increased CGRP levels in meninges (41.1±1.0 to 45.3±0.8 pg/ml, p<0.01), didn't change DMCs number and degranulation (p>0.05). Salmon-calcitonin decreased increased plasma (45.7±1.4 to 28.9±1.2 pg/ml, p<0.001) and meningeal (45.3± 0.8 to 42.8±0.9 pg/ml, p<0.05) levels of CGRP and DMCD (28.9 ±4.3% to 18.8±0.7%, p<0.05) induced by GTN.

Conclusion: Our findings suggest that GTN exhibits its migraine-like effects by increasing CGRP release in plasma, and from trigeminal sensory nerve terminals innervating meninges, and by inducing DMCD. Moreover, salmon-calcitonin reversed effects induced by GTN showing that salmon-calcitonin may be new therapeutic choice in migraine headache. Study was supported by AIBU-Scientific-Research-Fund, Grant-number: 2016.08.02.1060

Keywords: migraine, CGRP, salmon calcitonin, dura mater, mast cell

O-50

Effects of hypoxia and induced hypothermic pre-conditioning on iron homeostasis and iron regulatory proteins in cell culture

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Objective: Fetal and neonatal inflammatory response may contribute towards neonatal brain injury and developmentally related disabilities, such as cerebral palsy as well as cognitive deficits later in life. Damage related to hypoxia is an important factor in pathogenesis of these lesions. Microglia and astrocytes do not only initiate, but also contribute to the growth of lesions within the white matter as well as representing a potential strategic target for treatment in hypoxic disorders. The proper homeostasis of cellular iron transporters and storage are important to prevent excess iron overload or starvation in cells. In this study we aimed to show the effects of hypoxia on iron related proteins and cytokine production in glia, and assess if hypothermic preconditioning has any effect on iron homeostasis.

Methods: Cell cultures were prepared from the neonatal C57BL/6 mice and incbated in hypoxic conditions for 12 hours. Protein expressions were evaluated at the mRNA level by quantitative RT-PCR. Cellular iron accumulation was evaluated using Perl's histochemistry. Cytokine levels have been studied using ELISA.

Results: Hypoxia stimulated the expression of ferritin in both cells. Histochemical staining of accumulated iron in the cells correlated with ferritin expression. In addition to proinflammatory cytokine levels, TfR1 and DMT-1 increased especially in microglia, but ferroportin increased in both cells after hypoxia. Hypothermia increases the expression of transport protein levels differently.

Conclusion: In conclusion, hypoxic conditions accompanying glucose deprivation stimulated iron accumulation in microglia and astrocytes and triggered the expression of iron homeostasis proteins differently. Increased cellular iron and its efflux from glial cells might create a signal to trigger inflammatory responses which play role in hypoxic ischemic injury. Hypothermic preconditioning decreased the ferritin expression especially in microglia, which might suggest that the hypothermic control of cellular iron transporters and iron levels has a regulatory effect especially on the inflammatory pathways stimulated with hypoxic injury.

Keywords: hypoxia, microglia, astrocyte, iron, inflammation, hypothermic preconditioning

O-51

The relationship of NUDT6-overexpression with dendritic spine density

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Objective: Anxiolytic and antidepressant effects of FGF2 are well-established, however, its tumorogenic and angiogenic potential is an obstacle for its clinical use. To overcome this obstacle the role of FGF2 regulating molecules in affective behaviour can be assessed. NUDT6 is a natural antisense RNA transcribed from the opposite strand of FGF2 and it regulates FGF2 expression. In our previous studies we showed that NUDT6-overexpression leads to an increase in depression-like and anxiety-like behaviours. In this study we investigated whether depression- and anxiety-like effects of NUDT6 are mediated by changes in synaptic plasticity by determining the dendritic spine density (DSD) in prefrontal cortex (PFC) and dentate gyrus (DG).

Methods: 8–10 week old male Sprague Dawley rats were used for all experiments (n=4 for NUDT6-overexpression, n=5 for control). NUDT6 was overexpressed by daily intracerebroventricular injections of a rat NUDT6 plasmid for 14 days. Neurons and their processes were stained with rapid Golgi stain and images from third order dendritic branches were taken from PFC and

DG. We selected 3–4 slices/rat and counted dendritic spines in 4 neurons/slice. The difference in DSD (number of dendritic spines per micrometer) was compared between groups by t-test.

Results: In PFC, mean DSD was 1.42 in the control group, whereas it was 1.44 for NUDT6-overexpression group (p=0.94). In DG, mean DSD was 1.22 for the control group and 1.23 for NUDT6-overexpression group (p=0.95).

Conclusion: There were no DSD differences between groups in PFC and DG. However, this finding does not give any information about dendritic spine turnover. The turnover may be higher for one of the groups. This project is supported by Career Development Program of The Scientific and Technological Research Council Of Turkey, SBAG 110S481.

Keywords: FGF2, NUDT6, antisense, dendritic spine

O-52

Propolis treatment in experimental spinal cord injury: neurobehavioral and histopathological consequences

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Objective: Mechanical impact to spinal cord induces primary injury which is unavoidable. But, secondary injuries develop in time and may be preventable. Neuroprotective potential of various agents has been investigated against the secondary injuries in the past. Propolis is a complex natural product with proven beneficial activities in biological abnormalities, but its mechanism of action has yet to be understood. A recent study explored the prospect of propolis treatment in spinal cord injury (SCI). The current study was initiated as a follow up, but using a mild injury model with extensive supplemental data concerning behavior and histopathology.

Methods: The experiments were conducted with 30 male Wistar Albino rats weighing between 200–250 g. The rats were randomly divided into 3 groups (laminectomy control, SCI and SCI+Propolis Treatment, n=10). Laminectomy was performed at T10 level. The exposed SC was contused using an injury device http://www.hatterasinstruments.com/pinpoint.shtml). The contusion bit had 2 mm diameter and the injury settings were 85ms of duration, 1.5 m/s of impact veleocity and 0.5 mm of penetration depth. The rats were allowed to recover and survive for 28 days post-injury. Propolis was administered daily at oral dose of 300 mg/kg. Neurobehavior during the recovery was evaluated using Basso, Beattie, Bresnahan (BBB) test. Histopathological analysis involved H&E staining.

Results: Injured rats all had nearly the same level of BBB scores in the acute phase. But, in the following days, the rats treated with propolis exhibited higher scores than the untreated ones. This meant that they recovered better by regaining their lost functions and lesser tissue damage was present in the cords of the treated rats.

Conclusion: These improvements collectively indicated that propolis prevented or interrupted the cascade of secondary events following the injury, and ultimately protected the SC tissue from further damage. Considering the broad basis of its biological activities, the results suggest that propolis provides neuroprotectivity in SCI.

Keywords: spinal cord, spinal cord injury, propolis

O-53

Cerebellum and implicit contextual learning

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Objective: Contribution of cerebellum to implicit motor learning and its traditional role in motor functions are well known. However, findings on cerebellum's involvement in non-motor implicit contextual learning (ICL) are quite limited. In this task, reduced reaction time in repeated images with training is regarded as implicit contextual learning effect. The present work is the first study that investigates implicit contextual learning in patients with spinocerebellar ataxia (SCA) and aims to reveal cerebellar contribution to ICL.

Methods: 20 patients with SCA (10 females, age: 38.35±13.03, education: 9.1±3.8) and age, gender and education level matched 20 healthy controls (HC) (age: 37.55±13.08, education: 9.5±3.7) underwent ICL task which consisted of 16 blocks, each block had 24 trials of 12 novel and 12 repeated images throughout the experiment. 4×2×2 repeated measures ANOVA with epoch (1–4) and type (repeated-novel) as within-factors and group (patients-healthy controls) as between-factor was performed. Additionally, separate 4×2 repeated measures ANOVAs performed for each group.

Results: Epoch ($F(3.114)=36.34$, $p<0.001$), type ($F(1.38)=252.00$, $p<0.001$) and group ($F(1.38)=15.36$, $p<0.001$) main effects with group-type ($F(1.38)=6.72$, $p=0.013$), group-epoch ($F(3.114)=3.54$, $p=0.017$) and type-epoch ($F(3.114)=4.98$, $p=0.003$) interactions were significant. Separate ANOVAs revealed that type-epoch interaction was significant at HC ($F(3.57)=5.281$, $p=0.003$) while no significant interaction was found at SCA ($p>0.05$).

Conclusion: Compared to HC, SCA patients were slower throughout the experiment. Reaction times reduced more in repeated images, compared to novel trials with training. Post-hoc tests revealed that this effect was based on HC. In SCA group, training effect between repeated and novel trials was identical. In conclusion, ICL effect was found in HC, patients with SCA

showed deficit despite preserved skill learning ability. These findings indicate that cerebellum is involved in ICL processes and this type of learning is impaired in SCA. This study is supported by TUBITAK project #115S437 and IU-BAP project #42362.

Keywords: implicit memory, implicit contextual learning, cerebellar degeneration, spinocerebellar ataxia

O-54

Investigation of the effects of acute and chronic stress factors on working memory in healthy individuals

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Objective: Working memory disorders are core manifestations of psychiatric disorders. However, there are limited research on environmental factors that lead to working memory dysfunctions. In this study, it was aimed to investigate the effect of acute and chronic stress factors on working memory functions.

Methods: 39 volunteers without any lifetime psychiatric disorder and did not have any psychiatric complaints were recruited to the study. Beck Depression Scale, Beck Anxiety Scale, Chronic Stress Scale, Childhood Trauma Scale, Perceived Stress Scale, and Traumatic Life Events list were asked to be completed in the initial assessment by each participant. After the baseline measurements, letter-n-back task for working memory functions and stress thermometer were performed. 19 participants were exposed to Trier social stress tests and 20 participants were exposed to sham social stress tests. Positive and negative emotional changes were assessed by participants' salivary cortisol levels, pulse and blood pressure values, and PANAS scale before and after stress exposures. After applying the stress, the letter-n-back test was reapplied and the memory functions were evaluated.

Results: No difference was found in terms of clinical assessment scales and sociodemographic data between acute social stress and acute sham stress groups. Acute social stress was associated with increased salivary cortisol levels, increased stress scores, being higher stressful, unhappy, nervous, embarrassed, nervous and frightened, less enthusiastic feelings scores compared to the acute sham stress group. However, there was no difference between the acute social stress group and the sham stress group in terms of working memory task results. Both groups showed increased performance compared to pretreatment and there was no difference between the groups. No statistically significant relationship was found between baseline stress measurements and baseline cortisol levels and study memory task outcomes.

Conclusion: Although acute social stress may cause rapid emotional changes, it seems not to be changingworking memory functions.

Keywords: acute social stress, trier social stress test, working memory, chronic stress

O-55

Bipolar patients in neuroinflammation and neuronal destruction of markers comparative analysis of different disease subgroup

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Objective: Bipolar disorder(BD) is a mood disorder. Mood of manic and depressive feelings to come across the situation of people living with the disorder is defined as a BD. BD is not fully known etiology. In surveys conducted in recent years it has demonstrated that immunological factors play a role in BD.The complement system is part of the immunological system with this information BD have examined the relationship of the complement system.Research has underwent the first episode of BD diagnosed with chronic BD diagnosis have received and healthy control subjects collected blood samples. Granted permission from Istanbul Medicine Faculity Ethics Committee for Clinical Investigations (No: 1545).

Methods: Samples collected from the patients were examined by using ELISA and RT-PCR methods.BD to investigate degradation products level of the complement system which complement factor Bb, C4d, and C5b-9 proteins was investigated by ELISA.The complement system proteins which are named C1q, C3, C4, Factor B and the CD55 proteins are used by RT-PCR methods for detection of the activation on peripheral blood mononuclear cell sample.

Results: Investigated by ELISA methods, chronic BD complement degradation product it was significantly lower in the study group compared to other groups. RT-PCR method to investigate the expression of genes C1q, C4 and Factor B's first episodic group of BD and healthy control group were found to be significantly lower level compared to the chronic bipolar study group.In the CD55 expression levels in patients with chronic BD group level it was found to be significantly lower than other groups.The results of the researches showed inverse correlation with disease duration.The C1q and Factor B Barratt impulsivity testing parameters and C5b-9 was found to be correlated with the YMRS.

Conclusion: The complement system showed that it plays a role in the pathophysiology of BD. In light of this information, complement activity has been identified could be a diagnosis of BD. No additional research has been done.

Keywords: neuroscience, bipolar disorder, immunology, psychiatry

O-56

Investigation on the phenotypic and genotypic characteristics of lysosomal storage diseases in Sakarya city

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Objective: Lysosomal storage diseases (LSD) are rare genetic disorders which are frequently hard to diagnose. In our study, we examined the LSD patients in the city of Sakarya in terms of phenotype and genotype.

Methods: From 2011 to 2017, LSD patients from the Sakarya University Training and Research Hospital neurology clinic who were diagnosed by electrophysiological evaluations, muscle biopsy, enzyme analysis and genetic studies were included in our study. All of the cases were examined retrospectively in terms of sex, onset age and clinical findings

Results: In our study, all of the 6 index patients were male. Two of them were diagnosed with Fabry, four with late-onset Pompe disease and one with unidentified LSD. All of them had a family history and except the index patients, 24 new cases were identified among their family members. In this patient cohort, a disease-related gene mutation was detected in 4 of the index patients. These three mutations were c. [680G>A] - p. [R227Q], c. [1025G>A] as hemizygous GLA mutations, and homozygous c. 664G>A (V222M) and c271GA missense mutation as GAA mutations respectively. The most frequent mutation was the heterozygous c. [680G>A]-p. [R227Q] in the GLA gene. The other three index patients were presented with muscle weakness. It is interesting that one of the family members was presented with hoarseness. A 54-year-old Pompe patient with low GAA enzyme died in the follow-up period and mutation analysis could not be performed. The sixth index patient was presented with muscle weakness and was diagnosed with LSD as a result of his muscle biopsy revealing vacuolar myopathy. However, no mutation was detected.

Conclusion: As being a rare disease, it is still important to figure out the demographic and clinical characteristics of the patients. Our study demonstrates that genotypic and phenotypic characteristics of the LSD patients in the city of Sakarya are consistent with the literature.

Keywords: lysosomal storage diseases, Sakarya city, mutation

O-57

The effects of silymarin supplementation on cognitive functions in experimentally-induced diabetic rats

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Objective: The aim of this study is examine the effects of silymarin supplementation on cognitive functions in experimentally-induced diabetic rats and determine levels of brain-derived neurotropic factor (BDNF) and histon deacetylase (HDAC) in the process.

Methods: In this study 38 male Wistar rats, weighing between 400-450 g were used. Rats were randomly divided into the control, silymarin, diabetes and diabetes+silymarin groups. Diabetes and diabetes+silymarin groups were injected single dose streptozotocin (50 mg/kg) intrapertioneally. After seven days of the injection, diabetes was confirmed by measuring blood glucose concentration. Having been solubilized in corn oil, 200 mg/kg silymarin was given via orally to the silymarin and Diabetes+silymarin groups for 35 days. On 30th–35th days of the study, Open Field (OF) test, Elevated Plus Maze (EPM) Test and Moris Water Maze (MWM) test were performed. At the end of 35th day, brain and blood samples were taken from the rats under anesthesia. Brain-derived neurotrophic factor (BDNF) and histone deacetylase (HDAC) levels were measured in these samples.

Results: Blood glucose levels were higher in diabetic groups than control and silymarin groups. In OF test, the number of defecation was lower in the silymarin and diabetes+silymarin groups than diabetic group ($p<0.05$). In EPM test, the number entrance to closed arms were higher and time spent in open arms was lower in the diabetic group than the control group ($p<0.05$). In MWM test, the number of platform crossing and time spent in the platform area was lower in the diabetic group than control group ($p<0.05$). In the diabetic group, plasma and brain tissue BDNF levels were lower than the other groups ($p<0.05$), in (the) HDAC levels there was no difference among the groups ($p>0.05$).

Conclusion: The results obtained from this study suggest that, silymarin supplementation could improve cognitive functions in diabetic rats by increasing the BDNF levels.

Keywords: anxiety, cognitive function, diabetes, learning, silymarin

O-58

Anxiolytic and antidepressant-like effects of resveratrol in streptozotocin-induced diabetic rats

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Objective: Resveratrol is one of the most potent natural polyphenols with antiinflammatory and antioxidant properties. It has been demonstrated to have benefits against diabetes and its complications. In the current study, we investigated the effects of resveratrol on depression and anxiety-like behaviors in diabetic rats.

Methods: Adult male Wistar albino rats were randomly assigned for control, diabetic, resveratrol and imipramine treated diabetic groups (n=10). Diabetes was induced by a single intraperitoneal injection of streptozotocin (STZ) (50 mg/kg), and 2 days after the STZ injection the rats having hyperglycemia (>300 mg/dl) were assigned to be diabetic. Rats in treatment groups were injected intraperitoneally with resveratrol (20 mg/kg) and imipramine (10 mg/kg) for 4 weeks. Imipramine was used as a reference drug. At the end of the 4-week-period, forced swimming and elevated plus maze tests were used to evaluate diabetes induced depression- and anxiety-like behaviors. Significant differences were determined using one-way ANOVA followed by Tukey's post hoc tests.

Results: In the forced swimming test, there were significant differences among the groups in terms of immobility time dur-

ing second day of testing. Persistent hyperglycemia increased immobility time in the diabetic group compared to control group ($p<0.05$). However, resveratrol and imipramine treated groups exhibited significant reduction in immobility time as compared to the diabetic group ($p<0.05$). In the elevated plus maze, diabetes caused an increase in anxiety-like behavior compared to control group ($p<0.05$). Resveratrol and imipramine treatment significantly reduced anxiety-like behavior in diabetic rats ($p<0.05$).

Conclusion: The results of this study revealed that resveratrol exhibited anxiolytic- and antidepressant-like effect in diabetic rats, suggesting that chronic resveratrol treatment may able to prevent the development of comorbid anxiety- and depressive-like behaviour in diabetes. However, further investigations are needed to discover the overall mechanisms underlying this relationship.

Keywords: resveratrol, diabetes, anxiety, depression

O-59

Effects of kisspeptin on depression-like behaviors in male rats

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Objective: Kisspeptin, a novel hypothalamic Arg-Phe-amide-related peptide, is suggested to be important regulators of reproductive axis. In addition to its known effects on puberty and reproduction, kisspeptin may be plays an important role in depression-like behaviors. The aim of this study was to investigate of the possible antidepressant effects of kisspeptin in male rats using force swimming test (FST).

Methods: The male rats were randomly divided into six groups as control, sham, kisspeptin (10 nmol/10 mL), kisspeptin antagonist peptide 234 (1 nmol/10 mL), yohimbine (β2 adrenergic antagonists, 5 mg/kg), cyproheptadine (serotonin receptor antagonist, 3 mg/kg) (n=7 for each group). Kisspeptin and peptide 234 were administrated to animals by an icv injection. FST were carried out 30 minutes after the injection

Results: Kisspeptin treatment significantly decreased immobility time (floating) in male rats ($p<0.01$). We used the peptide 234, the kisspeptin receptor antagonist inhibited kisspeptin mediated antidepressant activity. Yohimbine (β2 adrenergic antagonists), and cyproheptadine (serotonin receptor antagonist) have similar effect with P234 ($p<0.05$). Climbing time was statistically increased by kisspeptin treatment. This effects of kisspeptin was significantly decreased by yohimbine and cyproheptadine.

Conclusion: The results of this study demonstrate for the first time that kisspeptin significantly decreased depression-like behaviors in male rat. We show that adrenergic and, serotonergic pathway are involved in kisspeptin induced antidepressant activity. These data provide new insights for the antidepressant action of kisspeptin which has potential therapeutic value as an endogenous antidepressant agent. This study was supported by TUBITAK-113S193

Keywords: kisspeptin, antidepressant, peptide 234, yohimbine, cyproheptadine

O-60

Evaluation of binge size after deep brain stimulation of the prelimbic medial prefrontal cortex in a rat model of binge eating

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Objective: Binge eating (BE) involves the consumption of a large amount of food in a short period of time and a loss of control during the binge episode. It is a key feature of the major subtypes of eating disorders like bulimia nervosa, binge eating disorder, anorexia-nervosa binge/purge type. Alterations in the mesocorticolimbic pathway has a crucial role in its pathophysiology. We hypothesized that BE rats receiving low-frequency deep brain stimulation (DBS) in the prelimbic cortex, which is a functional analog of dorsolateral prefrontal cortex in humans, would have reduced binge sizes compared with sham stimulation.

Methods: Eight male Sprague-Dawley rats were implanted with a DBS electrode in each rat's left prelimbic cortex. A 2-hour/day limited access to sweet-fat diet (contains %18.1 protein, %35.8 carbohydrates and %46.1 fat by calories) protocol was used to achieve a chronic BE state in the rats. After reaching a stable binge size level, each rat had experienced sham, low frequency (60 Hz) and high frequency (130 Hz) stimulation for three 2 hours long sessions each and two consecutive treatments were separated by at least 2 empty sessions to allow a washout of the effects. c-Fos immunoreactivity was assessed as a marker of DBS-mediated neuronal activation. Data was analyzed using one-way ANOVA and post-hoc Bonferroni test.

Results: Rats acquired binge state in 6 days. There was an overall significant treatment effect. After comparing the groups, we found that low frequency (60 Hz) stimulation of the prelimbic cortex significantly reduced the binge size compared to the sham stimulation ($p<0.0001$). High frequency DBS (130 Hz) had no significant influence on this behaviour compared to the sham stimulation ($p=0.9$).

Conclusion: This study suggests low frequency prelimbic cortex stimulation would be useful for correction of prefrontal hypofunction which is strongly associated with addiction pathogenesis.

Keywords: deep brain stimulation, prelimbic cortex, binge eating, rat model

Poster Presentations

(P-001 — P-105)

P-001

Distribution of GABAergic neurons in ventral midbrain in the mouse brain

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Objective: The mesolimbic system participates in the reinforcing and motivational effects of several rewarding stimuli. This system consists of dopamine neurons that project from the ventral tegmental area (VTA) to various forebrain areas including the accumbens as well as the central amygdala, prefrontal cortex, hippocampus and hypothalamus. Dopamine system in VTA is responsible for reward-related behavior. The VTA is a region rich in not only dopamine neurons but also GABA neurons. Both sets of neurons are known to receive similar inputs from different brain regions, and have different behavioral outcomes. GABAergic neurons receive more inputs from paraventricular hypothalamus and lateral hypothalamus, and they are known to inhibit the VTA dopamine system. To better understand the GABA system within the VTA, we investigated the distribution of GABAergic neurons in the VTA and neighboring regions.

Methods: 6 transgenic mice created by crossing tdTomato Cre reporter Ai14 and GAD2-Cre mice were used to visualize GABA neurons in different regions, based on Franklin and Paxinos atlas. The number of positive cells were mapped, counted and analyzed by SPSS for statistical evaluation. GABA neuron density in VTA and neighboring regions such as substantia nigra pars compacta (SNC), mammillary and supramammillary nuclei were compared in Bregma -2.92 to -3.08. VTA region were also compared to ventral tegmental decussation (vtgx), rostral linear nucleus of the raphe (Rli) and interpedincular nuclei (IPL, IPC, IPI, IPR) in Bregma -3.40 to -3.64.

Results: VTA has higher number of GABA neurons, followed by SNC and then mammillary and supramammillary nuclei in Bregma -2.92 to -3.08. In Bregma -3.40 to -3.64, rostral sub nucleus (IPR) had higher GABAergic neuron population then VTA, followed by IPC, IPI, Rli, IPL and finally vtgx.

Conclusion: IPR and VTA regions are the most densely occupied regions in terms of GABAergic neurons, in Ventral Midbrain in mice.

Keywords: GABA, ventral tegmental area, gad2, mesolimbic system

P-002

Modulation of endoplasmic reticulum-mitochondria interactions by tauopathy-associated mutations

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Objective: Neurodegenerative diseases are a major cause of disability in aging population afflicting both physical and mental health having a high cost for economy globally. Neurodegenerative diseases like Alzheimer's disease, Parkinson's disease, ALS/FTD are characterised by disruptions to cellular processes and functions in different forms throughout the brain. Though these diseases have clinically distinct progressive degeneration impacting different brain regions, several studies demonstrated that many perturbed functions seem to be regulated by a specific interaction site titled mitochondria-associated ER membranes (MAM), and many cellular dysfunctions take place when ER-mitochondria contacts are perturbed, suggesting a common mechanism to be investigated to understand the progression of neurodegeneration. This project investigated whether pathogenic tau impacts on ER-mitochondria interactions, focusing on tethering proteins in MAM site vesicle-associated membrane protein-associated protein B (VAPB) and protein tyrosine phosphatase interacting protein 51 (PTPIP51).

Methods: Methods used to look into VAPB-PTPIP51 binding with luciferase assay on Human Embryonic Kidney (HEK293) cells, colocalisation of VAPB-PTPIP51 with immunocytochemistry analysis and Immunofluorescent microscopy imaging on Human Neuroblastoma SH-SY5Y cells, VAPB-PTPIP51 protein expression with western blotting on both cell lines. In all experiments, cells were transfected with empty vector (control), WT tau, P301L and R406W tau mutations.

Results: Results showed increased binding between VAPB-PTPIP51 in mutant cells, especially R406W tau condition ($p<0.05$). However, no change in colocalisation of VAPB-PTPIP51 has been detected in immunocytochemistry experiments. VAPB and PTPIP51 protein expressions in western blots demonstrated no change in presence of tau mutations, being consistent with the findings of previous studies on other disease inducing proteins.

Conclusion: Though these results do not show very solid evidence for tau-induced changes in ER-mitochondria interactions, with modulations in experimental methods, it can be a step forward in investigating tau-induced changes in a common mechanism disrupted in many neurodegenerative diseases, presenting a promising target for developing future therapeutic approaches.

Keywords: ER-mitochondria interactions, MAM, neurodegeneration, tau protein, taupathies

P-003

Aquaporin-4 expression in ependymal cells of the rat third ventricle

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Objective: Aquaporins (AQP), are water channel proteins and involved in the regulation of water in the organisms. It was reported that, AQP4 is expressed on the basolateral membrane of cerebral ventricular ependymal cells, which play an important role in the water transport between brain parenchyma and cerebrospinal fluid (CSF) compartments. In addition, astrocytic endfeets directly in contact with the cerebral blood vessels and tanycyte cells are also expressed AQP4. In our study, it was aimed to show the expression of AQP4 protein in ependymal cells surrounding the rat third ventricle.

Methods: Adult female Wistar Albino rats were sacrificed by cervical dislocation under ketamine/xylazine anaesthesia. Brain tissue was removed and taken into fresh prepared 4% paraformaldehyde. After at least 24 h of fixation, brain tissue was taken into 30% sucrose solution. Then, sections with a thickness of 5 µm were taken with a frozen microtome and stained with immunohistochemical methods using anti-AQP4 antibody.

Results: AQP4 showed an intense immunoreactions in ependymal cells of the ventricular wall. Intensive staining especially in the basolateral membranes of ependymal cells is remarkable. Aquaporines allow unilateral matter passage controlled by osmotic gradient or hydrologic gradient. The excretion of soluble proteins, waste products and excess extracellular fluid in the interstitial fluid is accomplished in a manner which is facilitated by AQP4 channels. AQP4 proteins are intensely stained in ependymal cells which are surrounding brain ventricles containing cerebrospinal fluid. Thus, the presence of AQP4 channels in ependymal cells, which seem to be a bridge between CSF and brain parenchyma, suggests that these cells are involved in providing fluid balance and removing the excess molecules.

Conclusion: In summary, the investigation of the presence of AQP4 channels in astrocytes and tanycyte cells, as well as in ependymal cells, will contribute to the understanding and treatment of many brain related diseases such as neurodegenerative disorders.

Keywords: Ependymal cells, Aquaporin-4, rat

P-004

Bioinformatic analysis of transcriptome and proteome data for Parkinson's disease by using metabolite-centered reporter pathway analysis approach

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Objective: This study aims at the identification of metabolites and metabolic pathways affected in Parkinson's Disease by using a novel computational approach called metabolite-centered reporter pathway analysis (RPAm).

Methods: The genome-scale data used in this study were obtained from the public database Gene Expression Omnibus (GEO). The data were derived from RNA-seq and proteome analysis of prefrontal cortex tissue samples of Parkinson's Disease patients. The dataset includes 29 transcriptome samples, and 12 proteome samples. Both data types were computationally analyzed using several statistical tools included in R programming language. The statistical results were later mapped on a genome-scale human metabolic network obtained from humanCyc database by using RPAm approach.

Results: he results give significantly altered pathways between healthy and diseased states. The comparison of the results obtained by RNA-seq data with previously obtained results using microarray data enabled the comparison of the two transcriptome data types. Moreover, the set of altered pathways obtained by transcriptome data processing was compared to the results of proteome data.

Conclusion: This lead to documentation of possible post-transcriptional modifications at pathway-level in Parkinson's disease patients. This research was financially supported by TUBITAK (Grant Number: 315S302).

Keywords: genome-scale data, metabolic pathway, metabolite-centered reporter pathway analysis, neurodegenerative diseases

P-005

Doxorubicin-induced alterations of NF-κB expression in the hippocampus of metastatic breast tumor-bearing mice

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Objective: Doxorubicin (Doxo) is an effective chemotherapeutic agent that is used for the treatment of various cancers. It is commonly used for breast tumors which is resistant to endocrine therapy. Despite its effectiveness, it has toxic side effects on multiple organs such as heart, liver, kidney and brain. It has been reported that some breast cancer patients who underwent chemotherapy treatment had significant cognitive function impairments. DNA damage induced by Doxo may activate the transcription factors such the nuclear factor kappa B (NF-κB). Therefore, the aim of this study was to determine Doxo-induced alterations of NF-κB expression in hippocampus.

Methods: The cells that were metastasized to brain from 4THM murine breast carcinoma were previously obtained. The cells was named as 4TBM and were injected orthotopically into the mammary pad, Doxo (4TBM+Doxo) treatment (i.p once in a week, for 3 weeks, 8.75 mg/kg cumulative dose) started two days after inoculation of tumor cells. Necropsies were performed 25 days after tumor injection. Hippocampal sections were evaluated for NF-κB expression by using immunohistochemistry. Lastly,

primary tumor development and peripheral blood smears were evaluated. The findings were interpreted by comparing tumor-bearing and vehicle injected animals (4TBM+vehicle).

Results: Doxo administration decreased the growth of primary tumors. NF-κB expression was increased in hippocampal CA3 regions in both 4TBM+Doxo and 4TBM+vehicle groups. However 4TBM+Doxo group's NF-κB expression was significantly higher than 4TBM+vehicle group's. Also, increased number of neutrophils in the peripheral blood of 4TBM+Doxo group was observed.

Conclusion: NF-κB immunoreactivity is most likely allied to inflammatory process that caused by metastatic breast cancer. However our findings made us think that Doxo treatment may have an enhance effect on cancer related inflammatory process in hippocampus. Further detailed studies on this subject are required. This project is supported by TUBITAK 3001 The Scientific and Technological Research Council of Turkey (Project number 315S181).

Keywords: Doxorubicin, breast cancer, hippocampus, NF-κb

P-006

Neuroprotective effect of melatonin decreases NF-κB immunoreactivity in central nervous system: a model of murine metastatic breast carsinoma

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Objective: Melatonin is considered as an ideal neuroprotective agent such that it has both anti-oxidative and anti-inflammatory effects. Inflammation is an important step to promote tumor development, progression and metastatic dissemination. Inflammation induced NF-κB activation by increasing translocation of the NF-κB (p65) subunit into the nucleus. Thus, the aim of this study was to find out whether melatonin alters the NF-κB expressions as an inflammatory response to aggressive breast carcinoma in brain.

Methods: The cells that were metastasized to brain from 4THM murine breast carcinoma were previously obtained. The cells were named as 4TBM. 4TBM cells were injected orthotopically into the mammary pad and melatonin (4TBM+MLT) or vehicle (4TBM+V) treatment (ip.15 mg/kg 2 times a day; at 10.00 a.m and 1 hour before turning the room light off) started two days after inoculation of tumor cells. Necropsies were performed 25 days after tumor injection. Sections from cerebrum and cerebellum were evaluated for NF-κB expression as a marker of inflammatory response by using immunohistochemistry. All the findings were interpreted by comparing control animals that were not injected with tumor cells. Lastly, the changes were determined in primary tumor growth and phenotypes of immune cells in peripheral blood smears obtained from tumor-bearing mice.

Results: Melatonin administration induced regression of primary tumors. It is observed that melatonin partly prevented tumor-induced systemic increases in neutrophils. In accordance to decrease peripheral inflammatory response, melatonin also decreased enhanced p65 immunoreactivity in neocortex and cerebellum of tumor injected animals. These findings suggested that melatonin decreases tumor-induced peripheral and central inflammation.

Conclusion: These results demonstrated for the first time that the systemic effects of melatonin may have a role of central nervous system protection on highly aggressive metastatic breast carcinoma. This project is supported by TUBITAK 3001 The Scientific and Technological Research Council of Turkey (Project number 315S181).

Keywords: Breast cancer, melatonin, neuroprotective effect

P-007

The response of the neurovascular unit components to amyloid beta 1-42 injection into the rat hippocampus

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Objective: Alzheimer's disease (AD) is a neurodegenerative process which is characterized by the formation of neurofibrillary tangles and senile plaques, neural death and disruption of blood-brain barrier (BBB) integrity. Accumulation of amyloid beta (Aβ) peptide in the brain is one of the reasons for progression of AD. In this study, the animal model of AD was induced by injection of Aβ1–42 protein into hippocampal region of the rat brain, and the changes of neurovascular unit components such pericyte, astrocyte, microglia, neurons and endothelial cells of BBB were investigated at functional and ultrastructural levels.

Methods: In this study, adult female Wistar albino rats were used, Aβ1-42 protein (4 µl/4 µg)was injected into hippocampus intracranially, and until 30 days, they were followed up, and at the end, they were decapitated. Evans blue (EB) and horseradish peroxidase (HRP) were used to measure BBB permeability. Electron microscopy was used to show structural changes in endothelium, pericyte, astrocyte, microglia and neuron as a part of the neurovascular unit.

Results: 30 days later of Aβ1–42 injection, HRP reaction was not detected and no EB dye increase in brain parenchyma. Even there is no abnormal morphology seen in barrier type of endothelial cells at Aβ1–42 applied hippocampal region of rats, many electron dense lipofuscin granules in different sizes were

observed in cytoplasmic regions of astrocyte, microglia and neighborhood neurons.

Conclusion: The results of this study show that Aβ1–42 induced overproduction of lipofuscin which is a toxic substance,in astrocyte, microglia, and neuron of hippocampal region. At the same time, the accumulation of lipofuscin in neurovascular unit cells can cause dysfunction of these cells. Therefore, the indication of the changes in neurovascular unit caused by Aβ1–42 can contribute to the development of new experimental studies to eliminate the symptoms of Alzheimer's disease.

Keywords: Amyloid beta-1–42, blood-brain barrier, lipofuscin, electron microscopy.

P-008

Effects of axonal injury on neuron membrane mechanics

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Objective: After peripheral nerve injury, neurons in dorsal root ganglia and motor neurons of spinal cord and brain stem undergo fundamental morphological and molecular changes. Axonal degeneration and pain are arised from results of nerve injury. For improving treatment of peripheral nerve injury, these morphological and molecular changes must be detected. Axonal degeneration can be done in vitro (axotomy). Volume of cell bodies decreases with axotomy. Calcium influx into the cell body and calcium dependent molecules like calpeins are important in axonal injury. Cytoskeletal-related proteins are important in nerve injury. Caspase-3 dependent cleavage of actin and tubulin is a marker for axonal degeneration. There are less informations about mechanical changes of neurons in nerve injury. With this study, measurements and analysis of cellular tensions like lateral and cortical tensions in dorsal root ganglion neurons seperately and molecular mechanisms of peripheral nerve injury have been aimed.

Methods: Cortical and lateral tensions were measured in dorsal root ganglion neurons from Balb-c mice using optical tweezer system. For understanding molecular mechanisms of nerve injury, addition to lateral and cortical measurements before and after axotomy, intensities of Caspase-3/7 were measured using time-lapse flourescence microscope.

Results: According to results of our study, volumes of cells are significantly changed ($n=48$, $p<0.01$). Lateral tension before axotomy, cortical tension before axotomy and cortical tension after axotomy were correlated each other significantly ($n=48$, $p<0.05$). Furthermore, absolute area changes which are derived from axotomy were correlated with lateral forces after axotomy, cortical forces after axotomy and changes in Caspase-3/7 intensity significantly ($n=21$, $p<0.05$). According to our statistical results, cortical tension increased and lateral tension decreased. Caspase-3/7 is responsible for increasing of cortical tension.

Conclusion: Results of our study can contribute to further studies on the mechanical changes underlying peripheral nerve injury. Ethics commitee approval gave from Istanbul Medipol University Experimental Animals Ethics Commitee (38828770-604.01.01-E23167).

Keywords: Dorsal root gangion neuron, membrane elasticity, axotomy, force measurement, Caspase-3/7.

P-009

Examination of the inwardly rectifier potassium channels in hippocampus in the experimental Alzheimer's model

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Objective: Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive deficit and memory loss. It is shown that the activity of N-methyl-D-aspartate receptors (NMDA) decreases in the brains of Alzheimer's patients, thus the cholinergic systems are downregulated. Also, in addition to increase of glutamate, the presence of acetylcholine is known to be the most decreasing neurotransmitter in Alzheimer's patients. In relation to these molecules, inward rectifying potassium channels (Kir), which are activated in the final phase of the action potential and carry potassium ions into the cell. Accordingly, the aim of the study is to investigate the role of protein and gene expressions of Kir6.2 buffering the glucose; acetylcholine-activated Kir3.1; and glutamate-buffering Kir2.1 channels.

Methods: An amyloid toxicity model similar to the AH phenotype was generated by infusing amyloid beta 1–42 peptide on Sprague Dawley rats (250–350 g, n=40) for 30 days with microsmatic pumps. Infiltrated amyloid plaques were identified by immunofluorescent staining. At 30th day, the animals were sacrificed and their brains frozen in dry ice, then the hippocampus separated for protein and gene expression. Quantitative real-time PCR method and Western blot method were used for gene and protein expressions, respectively.

Results: In the hippocampus of rats that applicated amyloid toxicity model, KCNJ11 gene encoding the Kir6.2 channel and KCNJ2 gene encoding the Kir2.1 channel were significantly reduced ($p=0.01$). KCNJ3 gene encoding the Kir3.1 channel, had a significant increase in expression was observed ($p=0.05$). It had been observed that significant decreases in the Kir2.1 and Kir6.2 channels protein ($p=0.01$) and a significant increase in Kir3.1 channel protein.

Conclusion: Significant changes in the hippocampus of Kir channels that buffer glutamate, acetylcholine and glucose molecules that are directly linked to target in drugs used to suppress AD indicate that these channels may play a role in the disease mechanism.

Keywords: Alzheimer's disease, inwardly rectifier potassium channels, molecular expression

P-010

The evaluation of MAPK signal pathway in young rats with L-thyroxine infusion to dentate gyrus

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Objective: The effects of thyroid hormones on hippocampal synaptic plasticity have been investigated for many years. There is, however, a limited number of studies in the literature on the effect of L-thyroxine on depotantation responses. In this study, the molecular mechanisms of the effect of acute L-thyroxine (T4) administration on learning are investigated in hippocampal dentate gyrus.

Methods: In this study, 18 Wistar albino male rats (n=6 per group) were used; Saline infused group (S), T4 infused group during HFS (HFS-T4)(High frequency stimulus) and T4 infused group during LFS (LFS-T4)(Low frequency stimulus). L-Thyroxine was infused to HFS-T4 group during HFS induction period and to LFS-T4 group during LFS induction period. In the S group, an equal volume of saline was infused during HFS. After taking the depotantiation responses, the hippocampus of the rats were isolated and the MAPK signaling pathway protein expression assay was performed by the western-blot.

Results: There was no difference in the expression of tau, p-tau, p-p44, p-p42 proteins between the S infusion group and the T4 infusion group during HFS period (P>0.5). When the SF infusion group and the T4 infusion group were compared during HFS period, the expression of the p44 (p=0.07, F=0.176), p42 (p=0.006, F=0.232), p38(p=0.015, F=0.072) proteins significantly increased in the HFS-T4 group.

Conclusion: Thyroid hormones increase the level of MAPK proteins in the hippocampus of the brain. The relationship with these proteins involved in cell regulation of learning disorders in hyperthyroidism should be examined in more detail.

Keywords: L-thyroxine, Western blot, learning, memory, hippocampus

P-011

Effect of alpha lipoic acid on ultrastructure and function of the vascular damaged sciatic nerve

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Objective: Several kinds of injury models, such as crush, cut and graft repair have been well studied. However, there are few studies on the effect of alpha lipoic acid on vascular damage of the sciatic nerve. In the present study, our aim was to study the effect of alpha lipoic acid on recovery of the sciatic nerve after vascular sciatic nerve injury.

Methods: A total number of 40 Wistar rats were used for this purpose and divided into four groups [Group 1: Control, Group 2: Sham-operated, Group 3: alpha lipoic acid (+), Group 4: alpha lipoic acid (+)]. Sciatic nerve regeneration was evaluated by walking track analysis, pinch test, light and electron microscopy and antioxidant effect of Alpha lipoic acid (+) was evaluated by biochemical analysis.

Results: In the preoperative day, SFI values of the experimental group showed no significant deference (p>0.05) compared with the control group. However there was statistical significance between Group 3 and 4 in 4th postoperative weeks (p<0.05). In control and sham-operated groups withdrawal responses to pinch were full response (Grade 3). The onset day of full withdrawal response (Grade 3 and 4) to pinch stimulation was in the 2nd and 4th postoperative week, respectively. At the same week, number of the animals of Group 3 showing withdrawal was higher than those in Group 4. Electron microscopic analysis revealed less phagocytic cells and lamellar separation of the myelin sheaths in animals administered alpha lipoic acid than those of sciatic nerve injury performed. Electrophysiologic ana biochemical analyses were also revealed beneficial effect of Alpha lipoic acid as shown other analyses done. Conclusion: We think that our study will add valuable knowledge to the literature on the understanding of the nerve regeneration. This study was supported by Çukurova Üniversitesi Research Projects management Unit (project number: TSA-2016-6518)

Keywords: sciatic nerve, alpha lipoic acid, vascular damage

P-012

Investigating of the role of some proteins after infusing T3 hormone on hippocampus during high frequency stimulation followed by low frequency stimulation to induce depotantiation

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Objective: Learning and memory at the cellular level occurs as a result of the strengthening or decreasing of the signal pathway in some neuronal synapses. It is known that thyroid hormones have effect on hippocampal synaptic plasticity. The aim of this study was to investigate the effect of infusing T3 during high frequency stimulation (HFS) hippocampal dentate gyrus on depotantation responses and expression of some proteins involved in MAPK signal pathway.

Methods: Fifteen 2-month-old male Wistar albino rats were divided to two groups. First group was infused with saline (n=6) and second group with T3 (n=9) during HFS. Depotantiation was induced by HFS (100 Hz,1 sec, 4 times), followed by low frequency stimulation (LFS; 900-pulse stimulation at 1 Hz) and the infusion of saline or T3 occured during HFS. After recording, hippocampii were extracted and some total MAPKs proteins (p42, p44, p38), phosphorylated (p-p42, p-p44, p-p38) proteins and total tau (p-tau) and phosphorylated tau proteins were measured using Western blotting technique. The results were analyzed with independent t-test method.

Results: The expression of p44 ($p<0.001$), p42 ($p<0.01$), p38 ($p<0.005$) and p-p38 ($p<0.005$) proteins were significantly higher in the infused T3 group compaired to infused saline group during HFS stimulation; however, there was no significant difference in the expression of p-tau, tau, p-p44, p-p42 proteins between these groups ($p\geq0.5$).

Conclusion: It is concluded that infusion of T3 through HFS to strenght synaptic effeciency via depotantiation has no effect, and also there were no differences in p-tau, tau, p-p44, p-p42 proteins levels. Taken together, the increasing in protein levels may be the result of the genomic effects of T3 hormone. (TYL-2015-6282,TYL-2016-6306)

Keywords: T3 hormone, depotantiation, hippocampus, MAPK proteins, tau protein

P-013

Analysis of Pea3 phosphorylation sites

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Objective: ETS domain transcription factors which include specific common purine rich DNA sequence in all family members regulate numbers of cellular genes being critical for cellular differentiation, proliferation or development etc. Pea3 (ETV4) transcription factor which is ETS family member known to be regulated by specific growth factors through MAPK/ERK pathway during neural development. In this study, our goal is to understand what kind of effect growth factors (especially bFGF) have on Pea3 and to identify Pea3 phosphorylation sites.

Method: To find out the effects of growth factors on Pea3, we created mimicking and silencing mutations on putative MAPK phosphorylation sites (Serine 90, Serine 458 etc.) on mouse Pea3 (mPea3) by Site-Directed Mutagenesis. Then the mutant mPea3 plasmids were transfected into the SH-SY5Y human neuroblastoma cell line. After the analysis of protein expression levels of mutant mPea3s by Western Blot Analysis, transfected cell lines were induced with growth factors by cell culture techniques and the protein expression levels were checked. Finally, morphology of growth factor induced cells that include mutant mPea3 were observed by Immunofluorescence technique.

Results: We know that Pea3 has a role in neurite extension in various model cells, as supported with our experiments. The protein expression pattern of mutant mPea3s with/without growth factors was diverse. Also we are investigating target promoters of Pea3 such as Neurofilament-L.

Conclusion: Pea3 is known to have a role in neurite outgrowth, and the elucidation of Pea3 activation could be contributed to understanding of neuroregeneration.

Keywords: neurite outgrowth, axonal branching, Pea3, phosphorylation, MAPK/ERK pathway

P-014

Adrenergic receptors in mesenchymal stem cells obtained from different sources

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Objective: Mesenchymal stem cells (MSCs) with different origin are used for immune-modulatory and regenerative purposes. These cells were frequently investigated with regard to adhesion molecules, hematopoietic markers and immunologic markers. Sufficient data are not available about adrenergic receptors expressions during production process and differentiation of these cells. In this study, we aimed to determine how MSCs from different sources express adrenergic receptors during production and differentiation stages.

Methods: In this study, mRNA expressions in α1A, α1B, α2A, α2B, β1, β2, β3 subgroups of adrenergic receptors were shown with RT-qPCR in 1st, 2nd, 3rd passages where they are frequently used for regenerative and immune-modulatory purposes in MSCs obtained from human placental fetal membrane (FM) and bone marrow (BM). Receptor mRNA expressions were also shown with the same method in osteogenic, adipogenic differentiation of these cells and in differentiation under the effect of blocker.

Results: In our study, maximum expression rate compared to control group, 1st passage was detected in MSCs of BM-derived α1A-AR, in adipogenic differentiation group under phenoxybenzamine effect (fold change: 4221 ± 1391; $p<0.05$). High expression was detected for no adrenergic receptor in FM groups, as in some of BM groups. Maximum expression increase in MSCs of FM-derived was seen in β1-AR in adipogenic differentiation group under propranolol effect (fold change: 2.97 ± 0.94; $p<0.05$).

Conclusion: This study indicates that MSCs obtained from different sources act dissimilar with regard to adrenergic receptor expressions and differentiation. Therefore it is important to investigate these cells from different sources and from different passages with regard to receptors and differentiation in in-vivo studies in order to use these cells more effectively for clinical purposes.

Keywords: adrenergic receptors, cell differentiation, mesenchymal stem cells, RT-Qpcr

P-015

Effects of Elk1 on cell viability of brain tumor cells

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Objective: Ets-like transcription factor 1 (Elk1) is a member of ETS oncogene superfamily, a well-known substrate of ERK,

JNK, and p38 kinases and it is involved in numerous biological functions. Elk-1 has been implicated in protecting cells from apoptosis and downregulating apoptosis-associated genes thereby mediating cell survival. Furthermore, Akt-dependent phosphorylation of Elk-1 has been shown to be important for proliferation of glioblastoma cells. The purpose of this study is to elucidate the effects of different potential phosphorylation sites of Elk1 on cell viability of various brain tumor cell lines.

Methods: The effects of potential phosphorylation sites on Elk1 protein sequence on cell viability were examine by site-directed mutagenesis, flow cytometry and XTT cell viability assays. Potential phosphorylation sites on Elk1 protein sequence were converted to alanine and glutamic acid amino acids by site-directed mutagenesis and these mutant proteins were transfected into various brain tumor cells. The effect of these mutations on cell viability was then analyzed by flow cytometry and XTT analyses.

Results: In this study, both the FACS analysis and the XTT test showed that cell viability of SH-SY5Y (human neuroblastoma) and U87 (human glioblastoma) cell lines decreased when some of these phospho-sites (Ser149 and Ser304) on Elk1 were transformed into alanine to inhibit phosphorylation of these sites by mutagenesis. Similarly, when these regions were converted to glutamic acid, to mimic phosphorylation, an increase in cell viability was observed. However, some of the phospho sites (such as Ser303 and Ser 326) have not been shown to have significant effects on the viability of brain tumor cells.

Conclusion: It has been observed that, some phosphorylation sites analyzed in this study has effects on cell viability of brain tumor cells. Activation of these regions by various protein kinases may be contributing to the formation or development of brain tumors.

Keywords: brain tumors, mitosis, mitotic kinases, phosphorylation, Elk1

P-016

Effects of brain-derived neurotrophic factor on early neuroinflammation

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Objective: Neuroinflammation has a role on the pathogenesis of central nervous system diseases such as stroke and depression. Neurotrophic factors are shown to have protective effects against the central nervous system diseases. There is evidence that the neurotrophin brain-derived neurotrophic factor (BDNF) has some effects on neuroinflammation. It is shown that cortical spreading depression (CSD) that is the underlying mechanism of migraine aura triggers some neuroinflammatory reactions. In this study, we aimed to investigate the changes in BDNF protein levels after CSD at different time points and to determine the relationship between BDNF and the neuroinflammatory markers.

Methods: In adult Swiss albino mice multiple CSD (n=9) is induced by applying 1 M KCl-absorbed cotton ball over the burrhole on the frontal bone for 1 hour and after recovery period for 1, 2 and 6 hours, BDNF protein levels of parietal cortices are determined by Western blotting. To investigate the effect of BDNF on inflammatory markers triggered by CSD, human recombinant BDNF protein is injected intracerebroventricularly. At the 2nd hour of injection, BDNF levels of cortical tissues are determined to assess the diffusion of the protein from lateral ventricle to cortical tissues. CSD is induced after BDNF injection and the effect of BDNF protein on inflammatory markers is investigated by immunohistochemical techniques.

Results: BDNF levels of ipsilateral cortices increased at the first hour after multiple CSDs compared to contralateral cortices, and it reached to a statistically significant level at the 6th hour. At the 2nd hour after BDNF injection, BDNF levels of cortical tissues reached almost 10 fold of the contralateral hemisphere. The assesment of the effects of increased BDNF on inflammatory markers triggered by CSD is ongoing.

Conclusion: BDNF protein level in the cortex increases after multiple CSDs and it is thought to have a role in neuroinflammation.

Keywords: neuroinflammation, brain-derived neurotrophic factor, cortical spreading depression

P-017

The effect of memantine on epileptic activity in WAG/Rij rats

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Objective: Absence epilepsy is a type of non-convulsive epilepsy and is characterized by spike wave discharges (SWDs) seen in electroencephalogram (EEG) synchronized with pauses in behavior. Memantine is a non-competitive NMDA receptor antagonist, has anticonvulsant effect in various experimental epilepsy models. In the present study, the effect of memantine on WAG / Rij rats, known as a genetic model of absence epilepsy, was investigated.

Methods: A total of 21 WAG / Rij type, 6–8 months aged male rats were used in the study. Animals were divided into three experimental groups; saline (control), memantine 1 mg/kg/day (i.p.) and

memantine 5 mg/kg/day (i.p.). Tripolar electrodes were placed on the rat's skulls. Animals were allowed to recovery after electrode implantation for a week. Basal electrocorticography (ECoG) recordings were taken for 3 hours starting at 10:00 in the morning and ECoG recording was taken for 3 more hours after injection.

Results: Memantine, at doses of 1mg/kg and 5 mg/kg, significantly decreased the total number of seizures, SWDs and duration ($p<0.05$) compared to baseline ECOG recordings. These doses of memantine did not change the amplitude of SWDs ($p>0.05$) in all experimental groups. There was no difference between 1mg and 5mg memantine groups in regarding total number of seizures, SWDs, duration and amplitude ($p>0.05$).

Conclusion: The effects of drugs and environmental factors are variable in the type of epilepsy models. There are differences in the pathophysiology, drug dose and effect of absence epilepsy with convulsive epilepsy. The present study shows that the effect of memantine on absent epilepsy is anticonvulsive, similar to the effect of convulsive experimental epilepsy models. The underlying reason for the low-dose of memantine effect, which is ineffective in other experimental epilepsy models, need to be enlighten by other analysis methods in the absence epilepsy model.

Keywords: absence, epilepsy, memantine, rat, WAG/Rij

P-018

Comparative and functional analysis of significantly changed genes in different brain regions for Parkinson's disease

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Objective: The aim of this study is to analyze transcriptome data that belong to Parkinson's Disease(PD) obtained from closely related brain regions Substantia Nigra Pars Compacta (SNPc), Putamen, Gloubus Pallidum Internal (GPI) and Gloubus Pallidum Externa (GPE) by bioinformatics methods and thereby compare the healthy and disease state with t-test to find commonly differentially expressed genes and the associated cellular processes.

Methods: The datasets were obtained from NCBI Gene Expression Omnibus (GEO), the public web-based transcriptome database.

Results: As a result of the t-test, the number of significantly changed genes was 2764 in SNPc, 616 in Putamen, 1486 in GPI and 2654 in GPE region ($p<0.05$). ITPR1, NPM1, REEP1, FLT1, RUFY3, CNPY2, TSSC1 and DCLK1 are found to be commonly changed in all considered brain regions. The cellular processes common between these genes were further investigated. ITPR1 and DCLK1 commonly function in calcium-mediated signaling process. ITPR1 is an inositol 1,4,5-trisphosphate receptor and it is located in the endoplasmic reticulum (ER). It controls the release of calcium from the ER. ITPR1 is also involved in processes such as autophagy, apoptosis, phosphatidylinositol signaling system, and glutamergic -dopaminergic-serotonergic synapses while DCLK1 is additionally involved in neurodevelopment and neural apoptosis associated processes. The literature report on the antagonist interaction between ITPR1 and caffeine overlaps with the recently reported therapeutic role of caffeine on Parkinson's Disease.

Conclusion: Taking into account the fact that the identified genes are commonly regulated in different brain regions and they haven't been specifically investigated in terms of their relationship with Parkinson's Disease, the focus of the following research will be the investigation of molecular interaction of the identified genes. This research was financially supported by TUBITAK (Grant Number: 315S302).

Keywords: Parkinson's disease, statistical analysis, functional analysis, transcriptome data

P-019

Effect of chronic glutamate toxicity on glia cell viability and membrane lipid composition

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Objective: Our main objective is to investigate the cell death and change of membrane lipid composition depending on the chronic glutamic acid application, and meanwhile to determine the potential effects of some anti-epileptics (phenytoin, levatiracetam) and oxytocin.

Methods: Glial cells prepared from cortex region of new-born Sprague-Dawley rat brains. Cells were incubated with media included 0–1000 µm glutamic acid for ten days. Followed by determination of the effective dose of glutamate, 3 agents known with their cytoprotective and antiepileptic properties; oxytocin (10–1000 nM), levatiracetam (10–300 µm) and phenytoin (10–500 µm), were added with glutamate and protective effects of these agents were tested. Cell viability test (MTT) was utilized to evaluate toxicity and effects of agents. Student-t test was used for statistically evaluation. To analyse membrane lipids; cells were homogenized, sucrose gradient was applied and extraction was performed by Bligh Dyer method. Composition of membrane lipids were determined by HPLC.

Results: It's seen that glutamate caused a dose-dependent decrease in cell viability on glial cultures, and the glutamate dose, which will be used at following assays, was determined as 100 µm. It's observed that glutamate-induced cell death is reduced when glutamate was added to the culture medium together with protective agents. The highest protective effects were observed at 100 nM ($p<0.0005$) for oxytocin, 100 µm ($p<0.0005$) for levatiracetam and 10 µm ($p<0.05$) for phenytoin. After the extraction of glial cells obtained from cortex, the percentages of sterol, phospholipid and sphingolipid groups were determined with HPLC. The changes of these percentages when protective agents applied will be determined by ongoing HPLC studies.

Conclusion: Results of our study showed that glutamate have negative effect on viability of glial cells and changed the membrane lipid composition. Additionally, it's seen that some antiepileptics and oxytocin could be helpful to eliminate these negative effects. Present study is supported by Tubitak project numbered 214S673.

Keywords: excitotoxicity, oxytocin, levatiracetam, phenytoin, chromatography, membrane lipids

P-020

Determination novel targets for Pea3 transcription factor related to its function in neurons

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Objective: In this present study, we aimed to identify novel neuronal targets of Pea3 as compared to the constitutively active fusion protein Pea3-VP16 in a combinatorial approach in different neuronal cell lines.

Methods: We have used two plasmid constructs of Pea3 (pCDNA3-Pea3VP16 and pCDNA3-Pea3) for overexpression in various neuronal cells and compared microarray data; genes of interest were later identified through bioinformatics analyses and validated through qPCR assays.

Results: Our results show that many of genes in cancer, cell cycle, immune system and MAPK pathways are regulated by Pea3. When neuron-related pathways were investigated in detail, some targets previously identified by our group -including semaphorins, ephrins and ephrin receptors - were confirmed in various neuronal cells.

Conclusion: Pea3, a member of ETS domain transcription factor superfamily, regulates various cellular processes such as proliferation, angiogenesis and metastasis. In our previous study, we have shown that Pea3 promotes neurite outgrowth (Kandemir et al, 2014), and have recently identified some of its novel transcriptional targets using a consititutively active version of Pea3 (Kandemir et al, 2017). In this study, we showed that Pea3 regulates thousands of genes by microarray analysis in neuronal cells and we verified some neuron related targets such as semaphorins and ephrins.

Keywords: Pea3, transcription factor, microarray, gene regulation

P-021

Effects of atropine and guanethidine on relaxations induced by ethanol in mouse gastric fundus

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Objective: The aim of this study is to investigate the effects of atropine and guanethidine on relaxations induced by ethanol in mouse gastric fundus.

Methods: In this study, 45 mice (Swiss albino) of either sex were used. After killing the mice by cervical dislocation gastric fundus was isolated and prepared longitudinally. And it was mounted under 0.5 g tension in an organ bath filled with Tyrode's solution. The bath medium was maintained at 37 °C and gassed with 95% O_2 and 5% CO_2. Experimental data were recorded by an isometric transducer. ANOVA (Post hoc: Bonferroni) test was used for statistical comparison.

Results: Ethanol caused reproducible relaxations in isolated mouse gastric fundal strips. These relaxations were significantly inhibited by guanethidine (10–6, 10–5, 10–4 M), a blocker of adrenergic neurons, and atropine (10–6, 10–5, 10–4 M), a blocker of cholinergic muscarinic receptors.

Conclusion: Experimental data show that adrenergic neurons and cholinergic muscarinic receptors may have a role on relaxations induced by ethanol.

Keywords: Ethanol, gastric fundus, adrenergic and cholinergic effect, relaxation

P-022

Effects of agomelatine on diabetic neuropathy and structural properties of the nociceptive neurons

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Objective: In this study, it was aimed to investigate the effect of agomelatine on diabetic neuropathy and the structural properties of the nociceptive neurons in a dose-dependent manner.

Methods: Adult male Sprague-Dawley rats were divided into four groups (n=8/group): Control, DM, DM + Agomelatine (40 mg / kg) and DM + Agomelatine (80 mg / kg). Agomelatine treatment was started 4 weeks after STZ injection and continued for 14 days (p.o). Activity and latency to mechanical and thermal noxious stimuli were assessed weekly. After intracardiac perfusion, the dorsal root ganglia were dissected from L4-L5 levels. Semi-thin sections embedded into araldite blocks were stained with toluidine blue. Mean soma and fiber diameters of neurons were measured by morphometric methods from the digital images of sections and differences were compared by using statistical methods.

Results: Response latencies in the tail flick and the tail clip tests showed significant decreases in diabetic rats. The mean soma and fiber diameters of diabetic rats were also lower than those of the control group. Following agomelatine treatment, significant differences were observed among groups according to the dose of the drug and the duration of treatment [F(3.28)=9.757,

p<0.0001]. Treatment was more effective at lower doses (40 mg/kg) compared to higher doses (80 mg/kg) in restoring the structural alterations (p<0.001). Mean fiber diameter values also showed significant differences in comparison to values of the control groups [F(3.12)=5.112, p=0.0166].

Conclusion: In diabetic neuropathic rats, low-dose (40 mg/kg) agomelatine administration is effective in preventing hyperalgesia response by causing structural changes in the sensory neurons of dorsal root ganglia. These results suggest that agomelatine can be used as a novel therapeutic approach in the treatment of neuropathic pain.

Keywords: diabetic neuropathy, agomelatine, morphometry, spinal ganglion, tail clip, tail flick

P-023

Effects of single dose ketamine injection on the level of c-fos activation in the nucleus accumbens of prenatally stressed rats

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Objective: Neuronal connections of nucleus accumbens, play an important role in the formation of stress response. An anesthetic agent ketamine N-methyl-D-aspartat (NMDA) receptor antagonist has been used recently due to its acute antidepressant activity. In this study, we examined the effects of single dose ketamine injection on the activation of neurons located in the nucleus accumbens of prenatally stressed juvenile rats.

Methods: Sprague-Dawley rats were exposed to immobilization stress at last week of pregnancy, for 3 hours. Male offspring were divided into two groups at postnatal day 40. While treatment group received a single dose of ketamine injection (10 mg/kg, i.p.), control group (n=4) received serum physiologic injections. Similar treatments protocols were applied to offspring (n=6) whose dams were not stressed during their pregnancy. Animals were stressed by forced swimming for 6 minutes right before perfusion and then neuronal activation level was evaluated via c-fos immunohistochemistry.

Results: Density of c-fos (+) cells in the shell region of the nucleus accumbens decreased significantly (p<0.05) in prenatally stressed rats. Ketamine administration reversed the neuronal activation level in this region (p<0.01) similar to the levels of control group. Prenatal stress exposure and ketamine did not significantly affect neuronal activation levels in the core region. However, in prenatally stressed animals density of c-fos(+) cells in the core region showed a significant increase (p<0.05) following ketamine administration.

Conclusion: Nucleus accumbens has reciprocal connections with ventral tegmental region, amygdala, medial prefrontal cortex, hippocampus and hypothalamus. Glutamatergic, dopaminergic and noradrenergic neurons in this nucleus are related to the limbic system; therefore it is an important center for emotional control. Since it is known that NMDA activation in neurons projected to nucleus accumbens enhances dopamine levels especially in the shell region; ketamine-induced neuronal activation observed in our study might be responsible from the antidepressive effects via dopaminergic system.

Keywords: nucleus accumbens, c-fos, prenatal stress, ketamine

P-024

Investigation of oxytocin neurons located in paraventricular nucleus by using transgenic mice

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Objective: Oxytocin is a promising neuropeptide due to its prosocial effects and potential use in psychiatric and neurodevelopmental disorders. In mammalian brain, oxytocin is produced by neurons in the supraoptic, paraventricular and accessory nuclei of hypothalamus, in neighborhood with third ventricle. It is secreted both in blood as a hormone and in brain and cerebrospinal fluid as a neuromodulator. There are many studies in literature, showing the projections of oxytocin neurons and oxytocin receptors throughout the brain by using different techniques such as in situ hybridization, immunohistochemistry. In this study, we used transgenic mice and Cre-dependent viral technology to target oxytocin neurons of paraventricular nucleus (PVN) specifically.

Methods: Oxytocin-Ires Cre mice, which are transgenic mice expressing Cre recombinase under the control of endogenous oxytocin promoter, were used in this study. Male mice (14-16 weeks old) were unilaterally injected with pAAV-CAG-Flex-rev-tdTomato into PVN. After three weeks for infection, brains were removed. Slices (70 µm) were examined under confocal microscope to reveal projections of oxytocin neurons located in paraventricular nucleus. For in vitro electrophysiological recording, 8-week-old male mice were bilaterally injected with pAAV-EF1a-DIO-hM3D(Gq)-mCherry into PVN. Coronal sections (250 µm) containing paraventricular nucleus were cut and patch clamp whole-cell recording were performed. Recordings were taken from PVN, both in artificial cerebrospinal fluid (aCSF)-perfused and clozapine-N-oxide (CNO)-applied bath, to activate oxytocin neurons expressing hM3D.

Results: Oxytocin neurons located in PVN were found to be projecting to the brain regions such as arcuate nucleus, medial amygdala, stria terminalis and brain stem. In electrophysiological recordings, there was significant increase in spontaneous firing rate after CNO application to bath.

Conclusion: Oxytocin neurons are important for their effects on social behavior. By using this method, oxytocin neurons, which are thought to be responsible for a certain behavior, can

be visualized and their electrophysiological properties can be investigated in future studies.

Keywords: electrophysiology, oxytocin, paraventricular nucleus, patch clamp, transgenic mice

P-025

Characterization of kisspeptin neurons by viral vectors in mouse brain

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Objective: Kisspeptin is known to be a key regulator peptide for the regulation of GnRH and LH release through GnRH neurons and puberty. It is encoded by the Kiss-1 gene and known to be expressed especially in the arcuate nucleus of hypothalamus (ARC) and anteroventralperiventricular nucleus (AVPe). New location(s) of kisspeptin in the brain is being investigated with modern methodological approach. In this study, it was aimed to identify localizations and possible neuronal projections of kisspeptin in the mouse brain and to determine the functional map of kisspeptin neurons for further studies.

Methods: In this study, adult Kiss1-CreGFP transgenic mice were used for visualizing KISS1 secreting neurons. Cre-dependent virus (AAV-Flex-SYP-GFP/ AAV-Flex-tdT, 1:1 mixture, 400 nL) injections were performed by stereotaxic targeting of specified brain areas such as ARC, AVPe for sensitive detection of kisspeptin neurons and their projections. Before viral injections, the mice were anesthetized with isoflurane inhalation. They were placed in a cage for recovery and AAV expression for two weeks. Mice were sacrificed with an overdose of isoflurane and then intracardiacly perfused with saline followed by paraformaldehyde (PFA) solution. Mouse brains were removed and coronal brain sections (50 µm) were cut with vibratome and examined by confocal microscopy.

Results: Soma and synapses of kisspeptin neurons were detected with combination AAV-Flex-SYP-GFP and AAV-Flex-td tomato viruses in Kiss1-CreGFP mouse. Dimorphic differences were observed between male and female groups after the confocal microscopic investigation. Beside arcuate nucleus and AVPe, kisspeptin neuron soma and projections were determined in cortex, hippocampus, medial amygdala, habenula and the PAG.

Conclusion: Presence of soma and projections in brain regions other than the hypothalamus suggests that kisspeptin neurons may have behavioral effects in addition to reproductive functions. Our data also suggest that there are similarities and differences in localizations of kisspeptin neurons in female and male groups.

Keywords: axonal projections, kisspeptin, Kiss1-CreGFP, neuronal mapping

P-026

The effects of ion channel antibodies on neuronal membrane protein exposure and survival

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Objective: Nervous system injuries usually cause irreversible degenerative processes. Lately it has been shown that excitatory amino acids and ion channels play a great role in neuronal injury. Also the effects of excitatory amino acid receptor antagonists on the neural tissue in many of the central nervous system disease models have been proven. In this context especially ion channel antibody associated autoimmune encephalopathies are indicated. Clinical and radiological findings conclude that the antibodies in autoimmune encephalitis syndromes not only play a part in pathogenesis by causing molecular changes, but also have toxic effects leading to neuronal death. In this study our aim was to show the lethal effects of autoimmune encephalitis antibodies on neurons.

Methods: In our study primary neuron cultures were isolated from hippocampal tissue. Neuron cultures at different days in vitro were treated with immunoglobulin G (IgG) that was isolated from serum samples positive for either leucine-rich glioma inactivated 1 (LGI1) or N-methyl-D-aspartate receptor (NMDAR) antibodies. Propidium iodide (PI) was used to detect neuronal cell death. To understand whether apoptosis was involved in the neurotoxic effects caused by IgG treatment, JC-1 dye and real time polymerase chain reaction (RT-PCR) to detect the apoptosis associated molecule expressions were both used. Lastly intracellular calcium flow levels were measured and compared between the groups in order to understand the reason for the apoptotic death related to autoimmune encephalitis antibodies.

Results: A lower calcium flow in neurons treated with LGI1 and NMDAR antibody positive IgG was observed compared to the calcium flow in neurons treated with healthy IgG.

Conclusion: Our results show that autoimmune encephalitis antibodies cause diseases not only by changing molecular expressions but also by causing apoptotic neuronal death. Also our results indicate that neuronal apoptosis in this case is not caused by calcium toxicity.

Keywords: hippocampus, autoimmune encephalitis, LGI-1, NMDAR, apoptozis

P-027

Neurotoxic effects of some marine algae on mouse neuroblastoma cell line

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Objective: Many studies have been carried out in vitro and in vivo to investigate the antiproliferative, apoptotic, cytotoxic, antiprotozoal, antitumoral and neurotoxic effects on different cell lines of extracts and purified substances from marine algae. This study aimed to investigate the neurotoxic effects of extracts from red, brown and green algae samples in vitro in mouse neuroblastoma (NA2B) cells and in vivo with clinical and histological methods.

Methods: The neurotoxic activities were studied of Petalonia fascia (O.F. Müller) Kuntze, Jania rubens (Linnaeus) J. V. Lamouroux, Codium fragile (Suringar) Hariot algae collected from the Aegean Sea coasts of Turkey. Cultured NA2B cells were examined for vitality and proliferation measured by MTT. NA2B cells were also investigated for oxidative stress by e-NOS and apoptosis by TUNEL. The in vivo toxic effect study was performed with total thirty mice. Clinical evaluation was done with Tarlov scoring. Routine histological evaluation were done and apoptosis was evaluated by TUNEL system kit.

Results: Petalonia fascia, Jania rubens and Codium fragile were found highly toxic to the NA2B cells according to their IC50 values. H-score for immunohistochemistry of e-NOS showed that all extracts increased oxidative stress. Apoptotic index were found similar to oxidative stress and all extracts caused apoptotic cell death at IC50 values. According to Tarlov scoring results, all extracts caused attack and tremor. Histological sections from brain showed that there was edama, bleeding and cell degeneration for all extract application. There was oxidative stress and apoptotic cell death for all extracts.

Conclusion: These results clearly showed that algae extracts produced important toxic effect to neurons in vitro and in vivo. These toxic effects were found related to increase of oxidative stress and apoptosis. Neurotoxic effects of these algae with these mechanisms are meaningful for human health and also significant for drug development.

Keywords: marine algae, neurotoxicity, mouse neuroblastoma cell line, oxidative stress, apoptosis

P-028

Examination of the effect of transcranial direct current stimulation on minimal consciousness by means of EEG

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Objective: Chronic disorder of consciousnesses (DOC) is a serious condition for the burden of patient care and family health, and the health of the community. In recent years transcranial direct current stimulation (tDCS) technique has been proposed in this area and few promising studies have been reported. However, the number of studies supported by neurophysiologic agents is limited. This situation presents difficulties both in showing the improvement within the framework of objective criteria and in understanding the underlying physiopathological mechanisms. In this case, we aimed to evaluate the effect of tDCS on clinical and QEEG in a case of minimal consciousness state.

Methods: Patient with hypoxic encephalopathy was admitted to the post-CPR day 41 at the Medipol Mega Hospital Intensive Care Unit. 10 sessions of spontaneous EEG were taken before and after anodal tDCS application on left dorsolateral prefrontal cortex (DLPFC) and EEG recorded with BrainAmp 21-Channel DC System. In the analysis, power spectra are looked at. Clinical status was assessed with JFK coma recovery scale-revised (CRS-R). Ethical consent was obtained.

Results: In the power spectrum; Delta (1–3.5 Hz), teta1 (4–6 Hz), teta2 (6–8 Hz), alpha (8–13 Hz), beta (16–24 Hz) and gamma (25–48 Hz) frequency ranges. As a result, there was a general increase in all measured frequencies after tDCS application (FFT values of F3 electrodes, on delta power spectrum pre/post; 0.85/2.25, theta 0.67/1.13, theta2 0.27/0.82, alpha 0.10/0.61 beta 0.03/0.11 gamma 0.05/0.21). JFK coma recovery scales (pre/post) showed an increase in auditory (1/3), visual (2/4), motor (2/4), oromotor / verbal (1/2) focus scores and arousal (2/3) scale scores.

Conclusion: In our study, the clinically positive effect of TDSC in minimal consciouss state supports the literature. Overall, increase after TdCs in the locations that we measured as a result of EEG analysis before and after TDCS can be correlated with an improvement in CRS-R score.

Keywords: tDCS, minimal conscious state, EEG, CRS-R, power spectrum

P-029

The role of CB1 cannabinoid receptors in absence-like seizures of WAG/Rij rat

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Objective: Cannabinoid system has an important role in epilepsy. Genetically epileptic WAG/Rij rats develop spontaneous absence-like seizures after 3 months of age. In this study, WAG/Rij rats were used to examine whether absence seizures are associated with the CB1 cannabinoid receptors agonist ACEA and antagonist AM-251.

Methods: Tripolar electrodes were placed on skull to perform ECoG evaluation. Following the recovery period, ECoGs were recorded at 09:00 am for 3 hours every day. In the first set of experiments; saline (1 µl), dimethyl sulfoxide (1 µl), ACEA (1. 25, 2.5, 7.5 µg), AM-251 (0.50 µg) were administered intracerebroventricularly. In the second set of experiments, AM-251, at a

dose of 0.50 µg (i.c.v.), was administered 15 min after ACEA (7.5 µg, i.c.v.) injection. After injection, ECoGs were recorded for another 3 hours. The total number, the total duration, the number of spikes per cluster and the amplitude of the spike-wave discharges (SWDs) were calculated offline in every ten minutes.

Results: The doses of ACEA (2.5 µg and 7.5 µg) reduced the total number, the total duration and the number of spikes per cluster of SWDs, while high dose of AM-251 (0.50 µg) significantly increased all parameters ($p<0.05$) without changing the amplitude. AM-251, at a dose of 0.50 µg, 15 min after ACEA (7.5 µg), blocked the proconvulsant action of AM-251 ($p<0.05$).

Conclusion: These results indicate that the endocannabinoid system plays a role in the formation of absent seizures. Further studies are required to elucidate the certain mechanism of these effects. This study was supported by TUBITAK (Project number: 215S808)

Keywords: absence epilepsy, WAG/Rij rat, SWDs, ACEA, AM-251

P-030

Investigation of *NOTCH3* and *HTRA1* gene mutations in CADASIL / CARASIL patients

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Objective: CADASIL and CARASIL describe the two different inherited forms of Cerebral Arteriopathy with Subcortical Infarcts and Leukoencephalopathy that are autosomal dominant and recessive, respectively. Both are characterized by recurrent ischemic attacks, stroke, and migraine with/without aura, cognitive decline, dementia, psychiatric manifestations. Clinical diagnosis is supported by MRI, hyperintensities occuring in periventricular and deep WM even in anterior temporal area and capsula externa. CADASIL is associated with mutations in *NOTCH3* (Notch homolog 3) with 33 exons, located at 19p13.12. Exons 2, 3, 4, 5, 6 and 11 harbor hotspot region for mutations. CARASIL, with great similarity with CADASIL in terms of clinical presentation, begins earlier in cognitive decline and characterized with gait disturbances, back pain and alopecia. Responsible gene is *HTRA1* (HtrA serine peptidase 1) with nine exons, located at 10q26.13. There are a few publications with *NOTCH3* and *HTRA1*, investigated in concert. Studies in Turkish population are mostly case reports, dealin with hotspot regions of *NOTCH3*, and emphasize the intra familial variability. In this study, we aim to reveal *NOTCH3* and *HTRA1* gene mutation frequencies in our country, investigate genotype-phenotype correlation in cases with CADASIL/CARASIL. We further expect to develope the most effective algorithm for molecular diagnosis.

Methods: In this study, 24 familial and 29 sporadic cases (n=53) with CADASIL at Neurology Clinic of Istanbul Medical Faculty between 2015–2017 are initially screened for hotspot regions of *NOTCH3*, by Sanger. Mutation negative cases are investigated for other regions of *NOTCH3* by Next Generation Sequencing (NGS) that includes 42 dementia-panel genes. Mutation unidentified cases are further screened for *HTRA1* gene by Sanger sequencing.

Results: Three familial and two sporadic cases are found to have known mutations in *NOTCH3* at the initial targeted screening test.

Conclusion: Our investigation is presently continuing and results will be presented at the poster session.

Keywords: CADASIL, CARASIL, mutation, next generation sequencing (NGS), Sanger sequencing

P-031

The expression of calcium binding proteins on somatosensorial cortex of genetic absence epileptic WAG/Rij rats

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Objective: Absence epilepsy is an idiopathic generalized epilepsy. WAG/Rij is an inbred genetic rat model that exhibits absence seizures and depressive like behaviour. Recent electrophysiological experiments showed hyperexcitable locus in the somatosensory cortex (SPo1) of these animals. This study investigates the changes of calcium binding proteins, which are the parvalbumin (PV), calretinin (CR) and calbindin (CB) proteins during epileptogenesis. There is only limited information regarding the roles of the calcium ion in the mechanism that result in the formation of absence seizures.

Methods: Male WAG/Rij and Wistar Albino rats were used in this western blot experiments. The rats were deeply anesthetized by intraperitonal injection ketamine (80 mg/kg) / xylazine (10 mg/kg) coctail and perfused transcardially with cold physiological saline. Proteins were separated by 12% SDS-PAGE and transferred to a PVDF membrane protein bands were visualized with and chemiluminescence detection system.

Results: Our results indicate that even though there are no significant changes in PV, CR and CB protein levels, the expression of PV tends to decreasement in the somatosensorial cortex of WAG/Rij rats.

Conclusion: Although our results do not show any significant results as statistically, we plan to extend our studies by using different molecular approaches to clarify relation between CaBPs and absence epilepsy.

Keywords: absence epilepsy, calcium binding proteins, parvalbumin, calretinin, calbindin, WAG/Rij

P-032

Effects of lipoxygenase inhibition on neuroinflammation induced by acute transient cerebral ischemia/ recanalization

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Objective: Lipoxygenases (LOX) has important roles in stroke, atherosclerosis, diabetes, cardiac failure and hypertension. 12/15-LOX inhibition reduces infarct size and brain edema in acute phase of experimental ischemia models; in the late phase it promotes functional recovery via axonal regeneration and revascularization. There is recent interest in the role of 12/15-LOX inhibition after stroke. However, the effect of 12/15-LOX on inflammation induced by ischemia is not clarified. The aim of this study was to investigate the effects of newly synthesized 12/15-LOX inhibitor on the neuroinflammation triggered by experimental acute ischemia/recanalization model.

Methods: In this study, ischemia/recanalization was performed by Middle Cerebral Artery (MCA) occlusion with intraluminal filament in adult Swiss albino mice and immunohistochemical analysis was performed. ML351, newly synthesized 12/15-LOX inhibitor by van Leyen et al., was used as it is more potent and selective compared to other LOX inhibitors. Filament was pulled back for recanalization 1hr after ischemia and ML351 (50 mg/kg) in treatment group and solvent of the inhibitor, DMSO, in control group was injected intraperitoneally. Mice were sacrificed by cardiac perfusion 24hrs after ischemia and brains were used for immunohistochemistry. Immunohistochemical staining protocols were performed for inflammatory cytokines; IL-6, TNF-alpha, IL-1beta, HMGB1 and a microglia marker Iba-1. Then sections were imaged under fluorescent microscope with appropriate filter sets.

Results: 12/15-LOX inhibition resulted in attenuated immunohistochemical staining of cytokines related to brain damage in infarct and periinfarct areas after 24hrs of ischemia/recanalization-applied mice compared to controls. Besides 12/15-LOX inhibition decreased Iba-1 staining suggesting diminished microglial activation.

Conclusion: These results suggest that one of the possible mechanisms of 12/15-LOX inhibition on reduced infarct size may be the suppression of neuroinflammation after acute cerebral ischemia. This finding further supports the studies that advice 12/15-LOX inhibitors as a drug itself or as an adjuvant drug with tPA in acute stroke treatment.

Keywords: cytokines, ıschemia, lipoxygenase, neuroinflammation, recanalization

P-033

Cuprizone as a suitable agent for multiple sclerosis model in mice

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Objective: Multiple sclerosis (MS) is a chronic, autoimmune, inflammatory, demyelinating disease that affects the central nervous system (CNS). Even though genetics and environmental risk factors are thought to play an important role in the MS pathophysiology, specific pathogenesis to MS is not well described. Therefore, the aim of this study was to investigate the systemic effects of cuprizone on demyelination and to get more information on MS pathogenesis. Also, we have allowed reaching a good knowledge of the pathogenesis of MS.

Methods: 40 male C57BL/6 mice were randomly divided into four groups. (i) Demyelination control group which was fed 6 weeks with normal chow and given cornoil via gavage daily; (ii) Experimental MS (demyelination) group was administered 0.2% cuprizone for 6 weeks via gavage daily; (iii) Remyelination group was administered 0.2% cuprizone for 6 weeks via gavage and then fed with normal chow for 6 weeks; (iv) Remyelination control group which was fed with normal chow for 12 weeks. Demyelination and remyelination groups were evaluated to open field and tail-flick behavioral tests and determined the myelin basic protein (MBP) and glial fibrially acidic protein (GFAP) expressions by using immunohistochemical techniques on brain tissues.

Results: It is observed that increased GFAP and also decreased MBP expressions in hippocampus of experimental MS group compared to the control and remyelination groups. The open field area test results of the experimental MS group showed that increased anxiety levels and decreased locomotor activity. Furthermore, tail flick test results indicated that demyelination group's latency was longer than remyelination group.

Conclusion: According to our results, cuprizone is a suitable agent for creating MS model in mice. In this context this study is novel and likely to produce new projects that associated with CNS inflammatory disorders.

Keywords: multiple sclerosis, animal model, cuprizone, demyelination

P-034

Body weight changes and hind limb splay formation in acrylamide-applied rats

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Objective: Investigation of effect of a neuropathic agent acrylamide on hind limb functions and body weight of rats during ten days with day-by-day analysis.

Methods: Fourteen Sprague Dawley rats were equally divided into 2 groups. While the first group was given only i.p. saline solution, the second group was injected with 40 mg/kg i.p. acrylamide for ten days. The general condition, body weight and hind limb function of the rats were recorded daily for ten days.

Results: The body weights of the acrylamide-injected rats showed a decrease every day ($p<0.05$), whereas the control group rats showed body weight increase ($p<0.05$). In the control group, body weight increased significantly between days 1 and 10 ($p<0.05$), while in the acrylamide group significant body weight decrease was observed in the same period ($p<0.05$). When the average body weights were compared between the two groups, it was found that there was a significant decrease in the acrylamide group compared to the control ($p<0.05$). In the acrylamide-injected rats, nervousness, weakness in the hind limb and hind leg splay were observed especially after the first five days of experiment. The rats were trying hard to step dragging of their feet and abdomen along the floor. This abnormal gait and walking pattern were further aggravated toward end of experiment.

Conclusion: Acrylamide leads to nervousness, body weight decrease, a conspicuous hind limb splay and weakness in rats. Further studies are needed to elucidate the mechanisms that lead to these alterations.

Note: This study is part of the master thesis of Sedat KACAR.

Keywords: acrylamide, hind leg splay, neuropathy

P-035

Demonstration of a sensory neuroprosthesis on behaving rats

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Objective: Patients with different neurological conditions, such as peripheral neuropathy, para/tetraplegia and amputees may partially compensate for sensorimotor loss by using neuroprostheses. This study demonstrates a tactile neuroprosthesis system based on inputs from strain-gauge sensors covering a boot. The artificial sensation was created real-time by microstimulation of the rat somatosensory cortex.

Methods: Two Wistar rats were implanted with microwire electrodes and conditioned for intracortical microstimulation (ICMS) with bipolar electrical current pulses (phase duration: 600 µs, interphase interval: 53 µs). Then, the animals wore a boot covered by strain-gauge sensors for measuring vibrotactile stimuli (frequency: 40, 80 Hz; amplitude: 3.5–200 µm). Based on psychophysical equivalence model, ICMS pulse amplitudes were computed by a real-time processor. Rats performed a yes/no detection task and sensitivity was determined by non-parametric index (A'). The performance of the neuroprosthesis system (boot+ICMS condition) was compared to both natural stimulation and boot-only condition in ANCOVA. The stimulus amplitude was set as covariate.

Results: The stimulation condition had a statistically significant effect for both rats and at both frequencies (40, 80 Hz: all p's<0.01). Post-hoc analyses showed that the boot+ICMS condition generated a significantly higher detectability than the boot-only condition in all cases tested. However, the detectability in the boot+ICMS condition was lower than that obtained by natural stimulation, except for 80 Hz in one rat ($p=0.07$) which suggests that the tactile neuroprosthesis mimicked the natural psychophysical performance in this case. The stimulus amplitude increased detectability significantly regardless of the stimulation condition, except for one animal at 40 Hz.

Conclusion: ICMS improved tactile detection significantly when the rats wore the sensor-covered boots. However, the artificial sensory feedback was not psychophysically equivalent to that obtained by inputs from the glabrous skin, probably because of contactor decoupling at the sensor surface. These results are the first demonstration of a tactile neuroprosthesis system used during movement.

Keywords: somatosensory, touch, cortex, vibrotactile, neuroprosthesis, ICMS

P-036

Phenotypic and genotypic analysis of hereditary neuropathy patients in Sakarya city

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Objective: Hereditary neuropathy is a progressive group of disease that causes demyelination and/or axon loss in the peripheral nervous system. Although hereditary neuropathies are frequently in familial form, sporadic cases can be seen rarely. This disease has a high rate of disability. In our study, demographic, clinical, electrophysiological and genetic characteristics of the hereditary neuropathy patients in the city of Sakarya were investigated.

Methods: From 2011 to 2016, 27 registered hereditary sensorimotor neuropathy (HSMN) patients of our clinic and 55 identified family members were recruited for this study. The cases were examined retrospectively in terms of age, gender, age at symptoms onset, referral complaints, consanguineous marriage, clinical, electrophysiological, familial and genetic characteristics of the patients.

Results: Thirteen of the 27 cases (48.1%) were male and 14 (51.9%) were female. The mean age was 40.9±12.05, while the mean age of the symptoms onset was 22.92±13.55. Electrophysiological examinations showed axonal in 9, demyelinating in 11,

and mixed degeneration in 4 cases. Genetic studies revealed heterozygous PMP 22 deletion in 3 cases with mixed type degeneration and PMP 22 duplication was detected in 4 cases with demyelinating type degeneration. Thus, 3 cases were evaluated as Charcot Marie Tooth (CMT)-1A, 4 cases were hereditary sensitive polyneuropathy (HNPP), 9 cases were diagnosed as axonal CMT and 11 cases were as non-typed hereditary neuropathy.

Conclusion: Recent developments in the field of molecular biology have provided important development in recognition of many unclassified types of hereditary neuropathies. In our study, the phenotypic characteristics of demyelinating type, which can be genotyped in Turkey in the city of Sakarya, have been evaluated and compared with the literature.

Keywords: Charchot Marrie Tooth, clinical, genetic, Sakarya

P-037

Effects of chronic agmatine administration on cisplatin induced neuropathy in rats

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Objective: Cisplatin is one of the most effective antineoplastic agents used for treatment of several cancer types. However peripheral neuropathy, one of the most important dose-limiting side effects of cisplatin may develop in many patients. It was also reported that agmatine has analgesic activity. The aim of this study was to investigate the analgesic effects of chronic administration of agmatine on cisplatin-induced neuropathy and involvement of nitric oxide (NO) in this effect.

Methods: Female Sprague Dawley rats (n=6 for each group) (160–220 g) were divided into four groups: control (saline), cisplatin, cisplatin+agmatine, cisplatin+agmatine+L-NAME. Five single intraperitoneal (ip) 3 mg/kg cisplatin injections were administered once weekly. Saline (2 ml) was also given to prevent cisplatin induced nephrotoxicity. Single dose of agmatine (100 mg/kg ip) or in combination with 10 mg/kg ip L-NAME was administrated 30 min before every cisplatin injection. Mechanical allodynia, thermal hyperalgesia, tail clip tests were performed on the 6th day of each drug injections and hind paw, tail withdrawal latencies were recorded. Data were analysed with Kruskal-wallis and Wilcoxon signed-rank tests. $p<0.05$ was considered as statistically significant.

Results: Cisplatin decreased hind paw withdrawal latency in mechanical allodynia test ($p<0.05$), but did not alter this latency in thermal hyperalgesia test. Agmatine did not change hind paw withdrawal latency in mechanical allodynia test, whereas increased it in thermal hyperalgesia. In L-NAME group, there were no significant differences in the effects of agmatine. No significant change was detected in tail clip test in groups.

Conclusion: Our results indicate that chronic agmatine treatment prominently prevented cisplatin induced neuropathy. However, NO seems not to involve in this effect.This study is supported by Eskisehir Osmangazi University Commission of Scientific Researches under the Project Number: 2016-984.

Keywords: agmatine, cisplatin, nitric oxide, peripheral neuropathy

P-038

Effects of chronic anandamide administration on cisplatin induced neuropathy in rats

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Objective: Cisplatin is one of the most effective antineoplastic agents used for treatment of several cancer types. However peripheral neuropathy, one of the most important dose-limiting side effects of cisplatin may develop in many patients. It is known that cannabinoids have analgesic activity. The aim of this study was to investigate the analgesic effects of chronic administration of anandamide, a cannabinoid receptor agonist, on cisplatin-induced neuropathy and involvement of nitric oxide (NO) in this effect.

Methods: Female Sprague Dawley rats (n=6 for each group) (160–220 g) were divided into four groups: control (0.9% NaCl), cisplatin, cisplatin+anandamide, cisplatin+anandamide+L-NAME. Five single intraperitoneal (ip) 3 mg/kg cisplatin injections were administered once weekly. Saline (2 ml) was also given to prevent cisplatin induced nephrotoxicity. Single dose of anandamide (1 mg/kg ip) or in combination with 10 mg/kg ip L-NAME was administrated 30 min before every cisplatin injection. Mechanical allodynia, thermal hyperalgesia using analgesia meter and tail clip tests were performed on the 6th day of each drug injections and hind paw, tail withdrawal latencies were recorded. Data were analysed with Kruskal-wallis and Wilcoxon signed-rank tests. $p<0.05$ was considered as statistically significant.

Results: Cisplatin decreased hind paw withdrawal latency in mechanical allodynia test ($p<0.05$), but did not alter this latency in thermal hyperalgesia test. Anandamide did not change hind paw withdrawal latency in mechanical allodynia test, whereas increased it in thermal hyperalgesia. In L-NAME group, there were no significant differences in the effects of anandamide. No significant change was detected in tail clip test in groups.

Conclusion: Our results indicate that chronic anandamide treatment prominently prevented cisplatin induced neuropathy. However, NO seems not to involve in this effect.This study is supported by Eskisehir Osmangazi University Commission of Scientific Researches under the Project Number: 2016-984.

Keywords: anandamide, cisplatin, nitric oxide, peripheral neuropathy

P-039

The effect of etodolac on depression-like behaviour in forced swimming test

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Objective: It is thought that inflammation is the underlying mechanism of psychiatric disorders such as depression. In the present study, the effect of acute administration of etodolac as anti-inflammatory, analgesic, and antipyretic and selective COX-2 inhibitorsgroup on antidepressant-like behavior was investigated in forced swimming test.

Methods: 10–12 weeks adult Wistar albino 24 female rats were adapted one week (ethical committee no: 2015–15–169). All subjects were fed ad libitum. Sucrose preference test (1% sucrose, 1week) and forced swimming test as depression test were applied. Etodolac was dissolved with dimethyl sulfoxide and administrated 50 mg/kg or 100 mg/kg (24 hours, 5 hours,1 hours). İn order to Show wrong antidepressant effect, locomotor activity and rearing numbers were calculated after drug administration. In order to measure stres, harderian gland secretion was recorded. One way anaova and tukey test were conducted as statistical analyze.

Results: Swimming behaviour (active behaviour) time increased significantly in 50 mg/kg 100 mg/kg groups ($p<0.05$), when compared with control and vehicle groups. There were no change among the groups in climbing behavior time (the other active bahviour) ($p>0.05$). Floating as depressive behaviour when compared with vehicle and control groups remarkably decreased in 100 mg/kg group ($p<0.001$). There were no change among the groups in locomotor activity and rearing numbers ($p>0.05$). In sucrose preference test; During the study sucrose solution intake ratio was above %65 in all groups. Only one subjects was determined harderian gland dysfunction (type1).

Conclusion: Present resulsts indicate that etodolac reduced depression like behaviour in forced swimming test. These effect of etodolac demonstrate by increasing swimming behaviour, thus etodolac can effect serotenerjic system. After drug treatment, Locomotor acticity and rearing numbers were investigated and so no change among the all groups. Under the this situation, etodolac is not wrong antidepressant effect. Consequently etodolac can administer shortterm for potential depression treatment in the future.

Keywords: depression, etodolac, forced swimming test

P-040

Lexical-semantic processing in Turkish: Preliminary results of an event-related brain potential (ERP) study

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Objective: The aim of this study is to investigate by using ERP measures how lexical-semantic processing occurs in brain.

Methods: The sample group of study was composed of 21 healthy Turkish native speakers and between age of 18–30. All the participants were right-handed. Each subject fulfilled a lexical decision task, which was comprised of 240 trials. Two words which the first one was prime and the second one was target, were presented successively to participants for semantic priming and participants were asked to decide whether the target was a real word or not. One hundred twenty of target words were real Turkish words which half were semantically related with prime, half were unrelated and 120 target words were pseudo words in paradigm. EEG was recorded from 30 scalp electrodes. EEG signal was filtered with a band-pass (0.5–30 Hz) filters and segmented into epochs of 1000 ms. Artifact-free epochs were averaged for each subject. Mean amplitude values of N400 component during the time window 350–500 ms were analyzed by using repeated measure of ANOVA which included tree within group factors CONDITION (related, unrelated, pseudo-word) X LATERALIZATION (left, midline, right) X REGION OF INTEREST (central, central-parietal, parietal).

Results: N400 amplitudes revealed a significant threshold between conditions [$F(2,40)=18.573$ $p<0001$] and interaction for condition X region of interest [$F(2.114,42.273)=3.841$, $p<0.05$]. N400 was more negative for pseudo-words than semantically unrelated targets or semantically related targets. N400 amplitudes of semantically unrelated targets were more negative than related ones. The amplitudes of N400 on central electrodes was most negative. The negativity was decreased from central to parietal sites.

Conclusion: To the best of our knowledge, this study is a pioneer study conducted with EEG recordings in in semantic priming and lexical decision task in Turkish words. “This research has been supported by Ankara University Scientific Research Projects Coordination Unit. Project Number: 16L02000001”

Keywords: semantic priming, lexical decision, event related potential, N400

P-041

Hand dominance and hemispheric asymmetry: visual-spatial perception of objects and shapes

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Objective: Previous studies on the relationships between hand dominance and the ability of visuo-spatial perception (VSP) have revealed inconsistent results (Hardie & Wright, 2014). The aim of this study is to examine the relationships between hand preferences and VSP of objects and shapes.

Methods: To this aim, three experiments were conducted. Two of these experiments were related to the recall of the familiar objects and unfamiliar shapes (abstract) and their places (presented on left or right). The third experiment was the Line

Orientation Test (JLO). The hand preferences were assessed using the Hand Preference Questionnaire (HPQ; Nalçacı, et al, 2002). 42 individuals (aged 18 to 25; 22 female) participated in this study, namely 15 right handed, 12 left handed and 15 ambidextrous.

Results: The results revealed that left-handed participants were significantly faster in detecting the places of the familiar objects, with having shorter response time for both true and false answers compared to right-handed participants. This difference between the two groups remained stable even the place of the stimuli was changed across learning and recall trials of the experiment. In the unfamiliar shapes task, left-handed participants gave significantly more false responses in recalling the place of the stimuli when it was presented in the left side of the screen. There were no significant differences between three groups on the JLO task.

Conclusion: A study by Spironelli, Tagliabue and Angrilli (2006) suggested that left hemisphere is specified in the motor response selection in right-handed individuals, therefore it is faster to process stimuli on the right by the left hemisphere, when compared to processing stimuli located on the left by the right hemisphere. Consistently in the present study, right-handed participants gave less false responses when recalling the stimuli located on the left than left-handed participants.

Keywords: hand dominance, hemispheric asymmetry, visual-spatial perception

P-042

Evaluation of learning performance in maternal and adult-onset hyperthyroid rats: possible mechanism for p38 MAPK signaling pathway

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Objective: Disorders of the thyroid hormone levels are known to cause impairment of cognitive functions such as learning and memory. However, there is a limited number of studies of the underlying molecular mechanisms related these disorders. The aim of this study is to investigate mitogen-activated protein kinase (MAPK) signaling pathway in the hippocampus of maternal-derived and adult-onset hyperthyroid rats.

Methods: The study was performed on three groups; the control (C) group, maternal hyperthyroid (MH) group, and adult-onset hyperthyroid (AOH) group (n=6/group). Hyperthyroidism was induced by administering intraperitoneally L-Tyroxine during 21 days in the gestation period to dam of MH group and in the AOH group when they were 39 days old. The control group was given an equal volume of vehicle. When the rats were 60 days old, learning and memory testing in the Morris water maze was performed, and then the hippocampus of each rat was isolated and MAPK protein was identified by western blotting.

Results: fT4 levels were significantly higher in AOH (2.91±0.18 ng/dL) and MH (3.15±0.11 ng/dL) group than C group (1.82±0.18 ng/dL). A one way ANOVA followed by LSD test revealed that the MH and AOH group swum longer time period to find the hidden platform than C group(Ps<0.005). This finding was accompanied with slower swimming speed in the MH rats (Ps<0.005). A one way ANOVA followed by post-hoc test revealed that the Tau and p38 MAPK protein expressions were significantly increased compared to the control group (Ps<0.01).

Conclusion: Findings of the present study show that maternal-derived and adult-onset hyperthyroidism have impaired learning function and these effects may be mediated by MAKP signaling pathway.

Keywords: hippocampus, hyperthyroid, MAPK

P-043

Effects of estrogen and androgen on seizure-induced oxidative brain damage in rats

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Objective: Seizure frequency varies in different phases of menstrual cycle. Contradictory studies indicate that estrogen may have pro-convulsant or anti-epileptic effects. Effects of sex hormones on seizure severity, memory function and oxidative brain damage were investigated following a seizure induction.

Methods: Male Wistar (n=60) rats were given 17β-estradiol (1 mg/kg), resveratrol (40 mg/kg), selective estrogen-receptor modulator tamoxifen (40 mg/kg), anti-androgen cyproterone acetate (40 mg/kg) in tap-water or only tap-water (PTZ-control) for 28 days. To evaluate memory, passive avoidance test was performed. Seizure was induced by a single-dose pentylenetetrazole (PTZ; 45 mg/kg; intraperitoneally). Another group receiving only tap-water was injected with saline (n=12; control) instead of PTZ. Seizures were video-taped for Racine's scoring. Treatments were continued for 3 days following PTZ. Passive avoidance test was repeated on 31st day, and rats were decapitated to determine brain levels of malondialdehyde (MDA) and glutathione (GSH) and myeloperoxidase activity (MPO). Statistical analyses were performed by ANOVA and Student's t test.

Results: In all groups, except resveratrol-treated group, ratio of rats with tonic-clonic seizures was 50–66% and memory dysfunction was observed. Compared to PTZ-control group, seizure ratio (33%) and maximal seizure scores decreased in resveratrol-treated rats, while memory performance was similar to control group. In other treatments, no differences in seizure rates or memory dysfunction were observed. GSH levels were increased in brain tissues of all PTZ groups with respect to control group ($p<0.001$), elevation was greater in estradiol-treated PTZ-rats ($p<0.01$). MDA levels were increased in all PTZ groups, but tamoxifen suppressed this increment ($p<0.001$). Compared to control group, MPO activity in PTZ-control group was higher ($p<0.01$), while levels in resveratrol, tamoxifen and cyproterone acetate groups were not different.

Conclusion: Resveratrol pretreatment improved PTZ-induced seizures and memory dysfunction, while estrogen increased antioxidant capacity, and resveratrol, anti-estrogen and anti-androgen treatments decreased seizure-induced oxidative damage via suppression of neutrophil infiltration.

Keywords: epileptic seizure, estrogen, memory dysfunction, oxidative stress, resveratrol

P-044

Enriched environment reverses depressive like behaviour and reduces oxidative stress in chronic cerebral hypoperfused rats

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Objective: Vascular dementia or its imitating model for chronic cerebral hypoperfusion (CCH) is characterized by progressive impairment of cognitive and behavioral functions induced by loss of blood flow in several parts of the brain. The brain tissue is sensitive to oxidative imbalance and previous studies have demonstrated that oxidative injury plays a key role in the pathogenesis of vascular dementia. CCH is also associated with depressive-like behaviors in rats. Environmental enrichment (EE) has been associated with cognitive improvement, motor function recovery, and anxiety relief with respect to various neurodegenerative diseases. The aim of the present study was to evaluate the neuroprotective effects of environmental enrichment in rats subjected to permanent bilateral occlusion of common carotid arteries (2VO).

Methods: 2VO was performed to induce CCH in male adult Wistar rats. The animals randomly assigned to three groups: sham surgery plus a standard environment (control + SE), 2VO surgery plus a standard environment (2VO + SE), and 2VO surgery plus an enriched environment (2VO + EE) for 4 weeks. Each group consisted of 8 animals. Forced swimming tests (FST) were performed to measure depressive-like behaviors. Oxidant and antioxidant status of rat brain was assessed by measuring the levels of malondialdehyde (MDA) and reduced glutathione (GSH). ANOVA was applied for statistical comparison of the groups, followed by analysis with Bonferoni test to determine differences between the groups.

Results: MDA production and GSH level were significantly increased both in the hippocampus and cerebral cortex in hypoperfused rats ($p<0.05$). EE decreased MDA level both in the hippocampus and cerebral cortex ($p<0.05$). GSH level increased in the EE group. EE also decreased immobility time in FST 54% as compared to 2VO+SE group.

Conclusion: This study demonstrated that the oxidative stress and depressive like behaviour were significantly alleviated by application of EE after 2VO.

Keywords: environmental enrichment, vascular dementia, oxidative stres

P-045

N-(p-Amylcinnamoyl) anthranilic acid effect on food consumption and body weight okadaic acid induced Alzheimer's model in rats

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Objective: Transient receptor potential melastatin 2 (TRPM2) is a member of the transient receptor potential (TRP) channel superfamily, a diverse group of cation-permeable channels expressed in many tissues. TRPM2 Ca^{2+} permeable cation channel was shown recently to regulate energy expenditure, inflammation, insulin resistance and protected mice from developing diet-induced obesity in TRPM2-deficient mice. We examined the role of TRPM2 inhibitor N-(p-Amylcinnamoyl) anthranilic Acid (ACA) on body weight and food consumption in Alzheimer's model induced by okadaic acid (OKA).

Methods: All applications made in this study were conducted in accordance with the experimental protocol with the approval of the Inonu University Medical Faculty Experimental Animal Research Ethics Committee. In the present study, fifty (n=50) male Sprague- dawley rats (320–380 g) were randomly divided into five group; i) control, ii) sham (rats were injected icv with artificial cerebrospinal fluid (aCSF) and treated vehicle for 13 day), iii) ACA (rats were treated with ACA intraperitoneally (ip) 25 mg/kg/day for 13 days), iv) OKA (OKA was dissolved in aCSF and injected icv (200 ng) in a volume of 10 µl bilaterally) and v) OKA+ACA (OKA was dissolved in aCSF and injected icv (200 ng) in a volume of 10 µl bilaterally and treated with ACA ip 25 mg/kg/day for 13 days. Daily food intake and body weight of animals was measured.

Results: The body weight and food consumption of ACA treated rats were decreased compared to the control group ($p<0.05$). While values of OKA treated groups were increased compared to all of others group ($p<0.05$), values of OKA+ACA treated group were returned to normal.

Conclusion: TRPM2 channel blockers, ACA, caused an decreasing appetite and body weight while they increased in animals with neurotoxicity induced by OKA.

Keywords: N-(p-Amylcinnamoyl) anthranilic Acid (ACA), okadaic acid, transient receptor potential melastatin 2 channels

P-046

The effect of agomelatine on pain threshold in depression model rats

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Objective: It is known that depression disease reduces pain threshold. In this study, it was aimed to investigate the effect of

agomelatine (Ago) on the pain threshold in the depression model created by Porsolt Forced swim test (FST) in male rats.

Methods: Forty male rats were used in the study (10–12 weeks). The rats were divided into 4 groups. ZYT was made on the first two days of the experiment and repeated on 15th day after the drug was administered. Ten male rats were applied FST as depression (Dep) group. This group was administered saline (SF) with gavage for 15 days. The other 10 rats undergoing FST were treated with 1 mg/ kg dose Agomelatine for 15 days and formed the depression+agomelatine (Dep+Ago) group. Ten male rats were divided as the control group. Ten male rats were also given Ago for 15 days before FST and a control agomelatine (Cont+Ago) group was formed. All groups were fasted for 24 hours at the end of drug treatment and then sucrose preference test was applied. Pain thresholds were measured using "hot plate" and "tail flick" methods.

Results: A one way ANOVA followed by LSD test; When the sucrose preference, hot plate test and tail flick test were evaluated, depression group was found to decrease significantly ($p<0.01$) compared to the control group. Furthermore, when comparing the depression and Dep+Ago group the sucrose preference, hot plate test and the tail flick test, Dep+Ago group rats significantly increased ($p<0.01$) compared to the depression group. In addition, there was no significant difference between control and Dep+Ago groups in terms of these parameters ($p>0.05$).

Conclusion: Findings of the present study shows that depression reduces the level of pain threshold, and agomelatine that is the melatonin analogue changes this effect of depression as an antidepressant supports. This study was supported by Erciyes University Research Fund (TTU-2016-6430).

Keywords: depression model, Porsolt forced swim test, pain threshold, agomelatine

P-047

Music and memory: effects of vocality and familiarity

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Objective: The aim of this pilot study was to examine the implications of the familiarity of music in addition to the inclusion of human voice in music on short term memory and working memory during a visuo-spatial delayed response task.

Methods: A spatial delayed response task (OpenSesame 3 software) was used during which participants were exposed to a screen on which a dot flashed in random locations for 150 milliseconds and asked to remember this location after a color differentiation distraction task. The response accuracy was calculated and used as a dependent variable in the statistical analysis. The participants were divided into four groups (n=8–9 for each group). Two groups were exposed to a familiar song; however, one group received the vocal version and the other group received the instrumental version of the same song. Similar division had been applied to the other two groups using an unfamiliar song.

Results: Although a statistical significance has not been reached, the results have indicated a tendency of a detrimental effect of the vocality of music on the task performance with an interaction effect of familiarity. When the music is familiar, the vocality resulted in less number of errors whereas when the music is unfamiliar, the vocality resulted in more number of errors.

Conclusion: In light of these pilot results, it seems that it is imperative to conduct the study on a larger sample in order to reach statistical significance as a future direction. The observed tendency implies that although instrumental music is often regarded as a performance enhancer in learning tasks, this enhancing effect on performance seems to vary based on the interaction effect of the familiarity and the vocality of music.

Keywords: delayed response test, familiarity, music, short term memory, vocality

P-048

Effects of red and blue lights on electrodermal activity and emotional state

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Objective: It has been known that colored lights affect people through visual and non-visual pathways. The effects on the non-visual pathways are mainly attributed to the changes on the sympathetic part of the autonomic nervous system. On the other hand, colored lights bring about a number of effects on emotional states. For this reason, in this study, it is aimed to investigate the effects of white, red and blue lights on EDA and emotional state.

Methods: In accordance with this purpose, 39 subjects (12 females (27±7), 27 males (29±8)) were included in this study. All subjects were randomly exposed to 300 lux white, red and blue lights for 5 minutes. EDA values of subjects were recorded during exposure. In addition to this, all subjects were asked to fill to the Self Assessment Manikin Test (SAM) after each colored light exposure, and hereby their instant mood states were determined. The calculations were made using one-way ANOVA and Multiple Regression Analysis (Stepwise).

Results: As to our results, it has been concluded that the EDA values measured during red and blue light exposure were significantly higher than the EDA values measured during white light ($p<0.01$). In addition to this, the reported pleasure level for red light was found to be significantly lower than white and blue light ($p<0.01$). Investigating the level of reported arousal, it was found that the level of arousal was significantly higher during red light exposure compared to white and blue light exposure ($p<0.01$). Reported dominance level during red light was also found significantly higher compared to white ($p<0.01$) and blue light ($p<0.05$).

Conclusion: Findings show that the red and blue light exposure increase the EDA significantly. In addition to this, the red light

exposure decreases the pleasure, and increases the arousal and dominance level significantly.

Keywords: electrodermal activity, emotional state, colored lights, sympathetic nervous system

P-049

The effect of colored lights on autonomic nervous system and the alteration of these effects in association with personality traits

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Objective: Due to the widespread use of LED ('Light Emitting Diode') light sources, there is a significant increase in exposure to monochromatic lights. Colored lights are known to have effects on autonomic nervous system (ANS). These effects have been investigated by many studies via blood pressure, body temperature, respiratory rhythm, hormonal activity, heart rate (HR) and heart rate variability (HRV) parameters. Moreover, it has been reported that there is a relation between personality traits and color preferences. Therefore, in this study, it is aimed to investigate the effects of white, red and blue lights on ANS activity and the alterations of these effects in association with personality traits and emotional intelligence level.

Methods: For this purpose, 48 subjects (15 females (29±8), 33 males (28±6)) were participated to the study. All subjects were randomly exposed to 300 lux white, red and blue lights for 5 minutes. During exposure, HR values were recorded and HRV values were calculated using HR values. On the other hand, all subjects were subjected to the Big Five Personality Traits Test (BFPT) prior to the experiments in order to determine their personality traits. The calculations were made using one-way ANOVA and Multiple Regression Analysis (Stepwise) using the SPSS software.

Results: As to our results, there is no significant difference between the mean values of HRV during each colored light exposure ($p>0.05$). The results indicates that the changes in a number of the HRV values measured during red and white light exposure can be explained by optimism among the dimensions of emotional intelligence and agreeableness among personality traits.

Conclusion: It has been found that the effects that occurs in OSS activity during different colored light exposure changes depending on personality traits.

Keywords: Colored light, autonomic nervous system, heart rate variability, personality traits

P-050

A semi-stochastic numerical model for the hippocampal adult neurogenesis in the mammalian brain

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Objective: Adult neurogenesis in dentate gyrus (DG) is thought to be a prominent contributor in the dynamics of hippocampal memory networks. This discrete model aims to estimate the temporal changes in the neural progenitor cell (NPC) populations in DG, together with the products of differentiation – neurons, astrocytes and oligodendrocytes.

Methods: The dynamics are described in an ideal environment, where there is no limit for the total volume and all required chemical and physical cues that direct neurogenesis are continuously available. The system works independently (free of interactions) on three levels. Each level is defined as the dynamics in a stage of neurogenesis with three types of NPCs: type I cell (radial glia), type II cell (transiently amplifying cells) and type III cell (neuroblasts). Cell fate was introduced as a semi-stochastic process (a "choice") with a population limit for each cell type.

Results: In a 30 day period, the mean growth curves of three cell types and their products mimic the expected logarithmic growth and positively correlate among themselves. From 10 repetitions of the simulation initiated by 10 type I cells, 214±12 type I cell, 52±14 type II cell, 62±11 type III cell, 22±4 astrocytes ve 6±1 oligodendrocytes on average were obtained in this 30 day period.

Conclusion: Although it is based on discrete processes and has a rather simplistic approach, the simulations successfully provide a numerical template for adult neurogenesis, which can be further modified and implemented in a hippocampal trisynaptic loop network.

Keywords: adult neurogenesis, computational modelling, dentate gyrus

P-051

Fitting of spiking neuron models to electrophysiological recordings

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Objective: Cracking the population code of the neural assemblies in the investigation of how neurons can learn, adapt, and function relies primarily on the building blocks of the nervous system, the single neuron. Neurons communicate through electrochemical processes which are generated in response to transmembrane currents evoked by the activation of presynaptic receptors. Following the work of Hodgkin and Huxley, researchers attempted to build detailed models of single neuron electrical activity. On one hand we have complex biophysical models and on the other hand, simpler integrate-and-fire type models. In order to bridge the gap to higher functionalities, it is not necessary to address all the spatio-temporal features of ionic interactions. This allows us to define a level of description that is simple, yet is still able to make the link with higher cognitive functions, and that is sufficiently complete to

permit robust modelling. In this study, we present a computational method that can be used as a reference in building models of electrophysiological recordings.

Methods: Our study aims to identify a method to build electrophysiologically realistic and simple point-neuron models. Parameters and frequency-current relations of the neural models are obtained from empirically validated models. First model is adjusted to capture primary firing characteristics of spiny neurons in the striatum whose activity is modulated by dopaminergic transmission. Method was also successfully applied to replicate the electrophysiological repertoire of CA3 pyramidal cells including, rebound bursting, low-frequency bursts, and high-frequency tonic spiking.

Results: Proposed model was shown to successfully mimic the electrophysiological recordings in modulating spikes through glutamatergic, GABAergic, or neurotransmitter transmission.

Conclusion: Proposed method can be a benchmark in building simple, but realistic models to understand the underlying mechanism of neural communication. Single cell models can later be integrated into large-scale models in the characterization of the brain rhythms observed in specific cognitive function.

Keywords: computational model, electrophysiology, model fitting, spiking

P-052

Effects of nicotine and synchronization in cultured neural networks

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Objective: We investigated the effects of nicotine on hippocampal neural networks grown on Multi Electrode Arrays (MEA), and analyzed network activity in terms of synchronization which is under nicotine influence.

Methods: We dissected hippocampi from newborn (P0/P1) male Balb-c mice, then dissociated the tissue and seeded cells onto MEA. Hippocampal neurons formed network connections in vitro and the network they form became mature approximately in 18 days. We monitored electrical activity and recorded spike data periodically from 64 channel electrode array, and investigated recorded data with various analysis techniques using custom Matlab scripts. To test the effects of nicotine we worked on mature networks, whose activity behaviour were characterized. We observed acute effects of nicotine, (Sigma N3876), applying a final concentration of 100 uM, where nicotine was dissolved in pure ethanol. Thus, as control stimulant we used pure ethanol of same concentration which did not contain nicotine. Then by recording electrical activity for 20 minutes after stimulation, we obtained the data to be processed and analysed offline. Processing time series data involved filtering of 64 channel time series, determination of spike times and calculation of firing rates. Finally, by utilizing phase differences between firing rates, we measured synchronization effect observed in network activity and obtained quantified results.

Results: Under stimulant effect, network activity entered a synchronized period and continued until the effect expired. We continue experimenting on optimization and modelling of the duration of the influence, and trying to determine underlying mechanisms.

Conclusion: We are planning to investigate usage of nicotine as a preventive on diseases like Alzheimer's and Parkinson's for future studies. This work is supported by Boğaziçi University Research Fund under the Project Code 8080D.

Keywords: cultured neural networks, MEA, nicotine, synchronization

P-053

Investigation of T3 hormone on depotentiation induced by high frequency stimulation followed by low frequency stimulation in the hippocampus

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Objective: Learning and memory at the cellular level occurs as a result of the strengthening or decreasing of the signal pathway in some neuronal synapses (may also be happen on the same neuron). Long term potentiation (LTP), long tem depression (LTD) and depotantiation (DP) which experimentally generated by appropriate stimulation patterns applied result in the formation of new synapses or deletion of existing ones. In this study, we aimed to learn the effect of infused T3 hormone on the depotantiatin magnitude and the relationship between T3 and synaptic plasticity.

Methods: Experiments were performed by infusing SF; T3 during HFS or LFS in anesthetized 2-month-old male rats (Wistar type (n=7/group). Before infusion, the baseline efficacy of perforant path - dentate gyrus synapses was examined using input - output relationships. Depotantiation was induced by a high-frequency stimulation (HFS; 100 Hz, 1 sec, 4 times), followed by low frequency stimulation (LFS; 900-pulse stimulation at 1 Hz). Thus, both synapse formation and deletion were electrically triggered and recorded in synapses of the stimulated neuron pool.

Results: The infusion of T3 hormone during HFS or LFS was found that the temporal changes in population spike (PS) amplitude and excitatory postsynaptic potential (EPSP) were the same compairing with that sf infusion's results.

Conclusion: According to these results, the application of T3 during HFS or LFS does not seem to have a significant effect on the DP response. This work was supported by the Scientific Research and Project Unit (TYL-2015-6282 / TYL-2016-6306).

Keywords: thyroid hormones, learning and memory, hippocampus, synaptic plasticity

P-054

The effect of T4 infusion on depotentiation induced by high frequency stimulation followed by low frequency stimulation in the rat hippocampus

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Objective: Normal thyroid hormone levels may play a role in the balance between long-term potentiation (LTP) and long-term depression (LTD) which induce synaptic plasticity. Decreased LTP responses and stable LTD responses were observed in hyperthyroidism and hypothyroidism, and these results were confirmed by the application of intrahipocampal thyroxine infusion. Depotantiation (DP), the third form of synaptic plasticity, has not been reported. In this study, we aimed to learn the effect of infused T4 hormone on the depotantiation magnitude and the relationship between T4 and synaptic plasticity.

Methods: Experiments were performed by infusing SF; T4 during HFS or SF, T4 during LFS in anesthetized 2-month-old male rats (Wistar type (n=7 / group). Before infusion, the baseline efficacy of perforant path - dentate gyrus synapses was examined using input - output relationships. Depotantiation was induced by a high-frequency stimulation (HFS; 100 Hz, 1 sec, 4 times), followed by low frequency stimulation (LFS; 900-pulse stimulation at 1 Hz). Thus, both synapse formation and deletion were electrically triggered and recorded in synapses of the stimulated neuron pool.

Results: The infusion of T4 hormone during HFS was not found to change in population spike (PS) amplitude but excitatory postsynaptic potential (EPSP) was the different compairing with that SF infusion's results. The administration of T4 hormone during LFS affected the population spike (PS) amplitude ($P \leq 0.05$), but excitatory postsynaptic potential (EPSP) was the same compairing with that SF infusion group findings ($p \geq 0.07$).

Conclusion: According to these results, the application of T4 during HFS does not seem to have a significant effect on the DP response but the application of T4 during LFS seems to have a significant effect on the DP response. This study was supported by BAP (TYL-2015-6282,TYL-2016-6549).

Keywords: rat, T4, depotantiatioan, hippocampus

P-055

Use of magnesium in treatment of refractory status epilepticus: a case report

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Objective: Refractory status epilepticus is defined as status epilepticus that continues despite treatment with benzodiazepines and one antiepileptic drug. We aim to present a case of refractory status epilepticus responding treatment with magnesium sulphate.

Methods: A 21-year-old male patient with cerebral palsy who had known history of epilepsy treated with phenytoin 300 mg/day, valproic acid 1500 mg/day, clonazepam 4 mg/day, levetiracetam 3000 mg/day 3 times a year, admitted to our hospital due to contraction of whole body.

Results: There was not any significant pathology in the initial laboratory examinations. Fever was 36.0 C, blood pressure was 120/65 mmHg, pulse was 95 / min. No acute pathology was observed on cranial CT. Seizure activity was ceased by IV diazepam treatment. After developing a generalized tonic-clonic seizure activity without an intervening period of neurological recovery, the patient was taken to intensive care unit and an intravenous midazolam 0.1 mg/kg/h infusion was started. Clonazepam was increased to a dose of 12 mg/day. Lacosamide 200 mg / day was initiated to the patient whose seizure activity recurred when the midazolam dose was reduced. Blood magnesium level was detected 1.6 mmol / L. IV magnesium sulfate was administered. Midazolam treatment was discontinued in patients' follow-up. Daily IV magnesium sulfate was administered until the serum magnesium level was reached to 3.5 mmol / L. After 24 hours of magnesium administration, patient regained consciousness and seizure activity did not recurred. The patient were extubated and taken to neurology service.

Conclusion: Refractory status epilepticus is a life-threatening condition with high mortality despite aggressive treatment. It should be taken into consideration that magnesium sulfate treatment may be an important alternative in cases with resistant status epilepticus.

Keywords: refractory status epilepticus, magnesium, cerebral palsy

P-056

Obtaining the frequency-input characteristics of the medium spiny neurons with a computational model

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Objective: In this study, the role of input current on the frequency and number of spikes of the striatal medium spiny cells (MSN) will be investigated. Striatum, input structure of the basal ganglia circuits, has an important function in cognitive and motor tasks.

Methods: Izhikevich model given in the literature is modified to obtain a realistic computational model of MSNs. The data obtained using the Python-BRIAN software using the model are compared with the experimental measurement results given in the literature.

Results: MSNs are the most common cells in the striatum and connect to other nuclei of the striatum. Although MSNs have five different types of dopamine (DA) receptors linked to G-proteins on the membranes, MSN cells are classified as D1 and D2

according to the functions of their receptors. The D1 family, formed by D1 and D5 type receptors, has an enhancing effect on the cortex connections, whereas the D2 family containing D2, D3 and D4 receptors has a reducing effect on the cortex connections. The MSNs have GABA neurotransmitter and inhibit the Globus Pallidus. In the striatum, there are also large middle and small interneurons that use GABA, acetylcholine and somatostatin neurotransmitters. Behavior of MSNs according to experimental measurement results is bursting consisting of up-state and down-state levels. The up-state levels of membrane potential of the MSNs in dorsal striatum are about -67mV and the down-state levels are -88 mV, while the up-state levels of the MSNs in the ventral striatum are -63 mV and the down-state levels are -77 mV. The relation between the spike frequency of MSN cells and input current are also determined in relation to the presence of D1 and D2 type receptors.

Conclusion: The behavior of membrane potentials, up-state, down-state values, and stimulus-spike frequency relationship of the MSNs with the model are obtained in accordance with the experimental measurement results.

Keywords: medium spiny neuron, ventral striatum, dorsal striatum, burst, frequency

P-057

Effects of synaptic strength, stimuli and background noise on a central pattern generator which consists of a neuron population

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Objective: Central pattern generators are biological neural networks that produce rhythmic patterned outputs without sensory feedback. In our work, a central pattern generator based on Wilson-Cowan neural oscillator is presented and our aim is to determine the behaviour of this neural oscillator according to varying parameters such as synaptic strength, stimuli and background noise.

Methods: The dynamical model of the Wilson-Cowan oscillator is analyzed using simulations via Simulink software and the bifurcation diagrams are constructed using XPPAUT software.

Results: Wilson-Cowan neural oscillator represents the activity of large populations of neurons and synapses. However, although motivated by neurobiological considerations the mass models cannot hope to recreate some of the rich repertoire of responses seen in real neuronal tissue. When the behaviour of a nonlinear system such as Wilson-Cowan neural oscillator is examined using state-space diagrams, it is seen that the qualitative behaviour vastly differs from linear systems in a way that a parameter change can alter the dynamics of a nonlinear system completely. Such changes in the topology of the system are called as bifurcations. In our case, this alternation of parameters can be interpreted as connection strenghts of synapses or amplitudes of injected currents or frequency and magnitude components of the background activity. Essentially, we are trying to answer the questions like: What happens if the synapses make more connections or what happens if there is more stimuli? To answer these questions, bifurcation diagrams of the Wilson-Cowan neural oscillator are constructed. The dynamical behaviour of the oscillator for certain parameters and certain initial values are presented.

Conclusion: The synaptic strength, stimuli and background noise determine the dynamical behaviour of a neuron population. There are some criticial qualitative changes on critical parameter values called bifurcations. It is crucial to consider bifurcations when examining neuron population.

Keywords: central pattern generator, bifurcation, Wilson - Cowan neural oscillator

P-058

Action potential generation and propogation on a model of CA1 pyramidal neuron with axon-carrying dendrite

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Objective: Pyramidal neurons are the main cell types in cortex and hippocampal neural network. In glutamatergic neurons the main morphology consists of pyramidal cell body, apical dendrite extends between layers that have dendritic branches, basal dendrites connecting in the layer that have cell body and axon extending from the cell body. However the recent studies show that the axon in the cornu ammonis of healthy mammalian brain does not extend from the cell body, instead placed on the basal dendrite in many instances. This phenomena happens in 50% in CA1 and 30% in CA3 and in these cell types the stimulus is privileged on the axon carrying dendrite. The effect on information processes in these cell types on hippocampal trisynaptic circuits are not clarified yet.

Methods: In our study a conductance based CA1 pyramidal neuron with axon carrying dendrite (CA1-Acd) is modeled with NEURON simulation program and its action potential generation and propogation patterns are examined. The patterns are compared with a classical CA1 pyramidal neuron model and the effect of synapse location and density and axon initial segment morphology on CA1-Acd model on this process is examined elaborately.

Results: The obtained results verifies the priority of synapses on Acd and AIS length and its biophysical characteristics results in significant changes on anterograd and retrograd propogation with AP generation.

Conclusion: This model with complete characterization may help examinations on changes of neural network information processes when integrated with a trisynaptic circuit model.

Keywords: axon carrying dendrite, pyramidal neurons, CA1, CA3, action potential generation, action potential propogation

P-059

Implicit associative learning deficit in spinocerebellar ataxia: a functional MR imaging study

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Objective: Cerebellum's role in non-motor implicit associative learning (IAL) is not well known. To investigate cerebellar involvement in IAL, this study utilized fMRI in patients with spinocerebellar ataxia (SCA) and healthy controls (HC) during triplet learning task (TLT) including predictive cues, random cues and target stimulus. Since implicit learning is characterized by reduced activity within related areas, we expected regional decrease in fMRI activation by implicit learning of the associations between predictive cue and target.

Methods:15 patients with SCA (8 females, age: 36.67±13.22, education: 9.4±3.7) and age-gender-education level matched 15 HC (age: 35.67±12.74, education: 10.07±3.6) performed TLT in 3T MR scanner (Phillips-Achieva). The experiment, which included cued (CC) and control conditions each consisting of three blocks was repeated twice. fMRI data analysis was performed in SPM8. Significant clusters with cluster-forming threshold uncorrected $p<0.001$ combined with cluster-level FWE-correction were reported. Clusters showing significantly decreased activity were defined as ROI. Correlation analysis was performed between mean activation and behavioral values.

Results: Accuracy was identical between groups ($U=72.5$, $p=0.098$). SCAs were slower than HCs throughout the experiment ($t=5.54$ $p<0.001$). In CC, HC responded faster with training ($t=2.89$, $p=0.012$), while no significant difference was found in SCA. For HC>SCA, significant reduced activation in CC was found in a cluster including bilateral cerebellum VIII-VIIb (100 voxels, tmax=5.51, $p=0.023$, MNI peak coordinates: 15-73-49). Activation in this area was positively correlated with mean reaction time (RT) in CC ($r=0.589$, $p<0.001$).

Conclusion: Our findings showed that increased activity of bilateral cerebellum lobule VIII-VIIb in early IAL was reduced with training correlating with RT. More importantly, this learning effect was weak in SCAs compared to HCs. In conclusion, when the hidden associations were learned, task could be performed with less cognitive effort and cerebellar circuits play an important role for this achievement. Supported by TÜBİTAK project #115S437 and IU-BAP project #42362.

Keywords: non-motor implicit associative learning, implicit memory, cerebellum, spinocerebellar ataxia, functional magnetic resonance imaging

P-060

The impact of stimulus design on a Jansen rit temporal lobe epilepsy model

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Objective: Large-scale brain networks provide essential information about how functional dynamics arise from structural connectivity. Simulations on these networks can be a key to understand cognition and neurological diseases. In recent years, stimulation-based non-invasive techniques that affect large-scale brain dynamics are becoming a promising research area. The exact mechanism of how externally applied inputs into neural mass models influence the activity of neural population is still to be described. The purpose of this study is to present a systematic characterization of stimulus-driven activity on the large scale brain dynamics of temporal lobe epilepsy using Jansen Rit neural mass model in The Virtual Brain (TVB), which is a neuroinformatics platform for large-scale brain network modelling.

Methods: Using two different neural mass models in TVB, the impact of the spatial extent of stimulation (regional vs. surface) was analyzed, specifically on the temporal lobe regions. Moreover, the effect of the temporal parameters of the stimulation and the modulation of wave patterns are further investigated on the model.

Results: A parameter sweep has been performed on the Jansen Rit and Epileptor neural mass models and parameters that are more critical in in delaying or suppressing the temporal lobe epileptic activity have been founded. Also the impact of pulse parameter intervals on temporal lobe epileptic waves has been determined by changing stimulus onset, frequency, amplitude, pulse width and duration. It has been observed that amplitude is the most critical parameter among them when comparing all the parameters.

Conclusion: The impact of different stimulus models and different parameter intervals has been discussed. The presented findings about the effect of stimulation patterns on the temporal lobe epilepsy model can be further used to provide insight on the possible therapeutic uses of electromagnetic stimulation.

Keywords: neural mass models, stimulus-driven activity, temporal lobe epilepsy

P-061

Comparison of memory performance of late onset depression patient and healthy controls: fMRI study

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Objective: Recent studies have shown that, regional dysfunctions play important role in disease onset. In late onset depres-

sion (LOD), after age of 45, it is thought that genetic effects are less pronounced and neurodegenerative process is more dominant in contrast to early onset depression. Thereby, it is thought that acquired pathophysiology, starts shortly before syndrome, causes clinical symptoms. This study aimed to present comparing block design functional Magnetic Resonance Imaging (fMRI) activations of LOD and healthy individuals during a memory task. Determination of brain regions in which disruption occurs during LOD can lead us to understand the pathology of the disease, develop treatment and predict the treatment prognosis.

Methods: 15 LOD-patients and 14 healthy controls (HC) are included in this study. Following psychiatric assessment, SCID-1, Mini Mental State Examination (MMSE), Hamilton Rating Scale-Depression (HRSD), Clinical Dementia Rating (CDR) is performed. Participants underwent structural and functional MRI scanning. Brain activities of patients and HC were evaluated during a memory task. Encoding of face-name pairs, face recognition and name recognition after five minutes of encoding were used to test memory performance. fMRI images were analyzed by using Matlab-SPM12 software. Comparison of activations were evaluated with ANOVA.

Results: Findings showed that LOD patients had less activation in hippocampal region compared to HC. In contrats, LOD patients showed higher activation in parietal lobe for face recognition and higher activation in posterior precuneus, parietal and limbic regions for face-name recognition interaction.

Conclusion: In this study, hippocampus activity is decreased in LOD patients for encoding; however, the activity of cognitine network is increased for more complex cognitive task like face recognition. Although LOD patients and HC showed similar memory performances, LOD patients also used emotional areas. Especially, LOD patients showed increased activity in limbic regions in addition to cognitive network for face-name association. Conclusively, patients used related brain regions more active to continue their memory performances.

Keywords: Functional Magnetic Resonance Imaging (fMRI), late onset depression (LOD), major depression, memory

P-062

Anatomical connectivity changes in bipolar disorder and schizophrenia investigated using whole brain tract-based spatial statistics

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Objective: Previous studies showed common reduced white matter density in the fronto-temporal and fronto-thalamic pathways in patients with both bipolar disorder (BD) and schizophrenia (SCH). Actually, most of these studies used manually prespecified regions of interest (ROI) and had methodological limitations. However, tract-based spatial statistics (TBSS) is a whole brain analyses method without prespecified ROIs, which uses nonlinear image transformation and permutation tests with a correction for multiple comparisons. The main aim of the current study is investigating possibly common anatomic connectivity changes in BD and SCH using whole brain TBSS.

Methods: The study consisted of 39 SCH patients, 41 individuals with BD and 23 controls. Whole brain images were acquired using a 1.5 Tesla Philips Achieva MRI scanner. The 4D projected FA data were calculated for each individual using TBSS and were fed into GLM modeling to conduct group comparisons. The threshold-free cluster-enhancement was used for multiple comparisons.

Results: As compared to SCH group, BD group showed significant FA reductions in the following white matter tracts in the right hemisphere: anterior thalamic radiation, inferior fronto-occipital fasciculus, and uncinate fasciculus. Compared to controls, SCH and BD groups showed FA reductions in similar white matter tracts.

Conclusion: Although individuals with BD and SCH patients showed similar anatomical connectivity changes compared to healthy subjects, the connectivity reductions in the right hemisphere of BD can differentiate two groups from each other. The findings of the current study can contribute to a better understanding of the etiology and pathogenesis of the BD and SCH.

Keywords: bipolar disorder, diffusion tensor imaging, schizophrenia, tract-based spatial statistics, tractography

P-063

Investigation of sella turcica morphometry in empty sella syndrome

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Objective: Sella turcica (ST) is a bone structure closely related to the pituitary gland and have anatomically and clinically importance. Empty sella syndrome (ESS) is the large or deformed radiologic image of ST filled with cerebrospinal fluid partially or completely. The purpose of our research is to investigate the sella turcica morphometry in ESS and to reach a conclusion in the light of the literature on the reasons and consequences of the change in ST.

Methods: In our study, datas of 33 adult patients with ESS in cranial CT and 30 adults objects with normal findings as control group were used. Sella length, sella height, sella anterior-median-posterior heights, sella area, sella depth and sella anterior-posterior diameter were evaluated in both groups.

Results: In all parameters, the values determined in the group of patients with ESS were higher than those in the control group, but it was seen that sella depth and anterior-median-posterior heights were statistically significant.

Conclusion: It is a literary knowledge that ST of ESS patients is expanding. But we could not find a morphometric study that showed this. We believe that the morphometric measurements we detected in patients with ESS will be referred to physicians working on pituitary and sella surgeries. In a study of patients with Sheehan's syndrome, it was found that the volume of the ST decreased as a result of the pituitary gland which contracted due to infarction and necrosis. We observed that any local or systemic clinical problem affecting the pituitary gland can change the size of the sella in our study and in the literature. We think that big or small formation of an organ or a tissue may be either variant or pathologic, as well as physical interaction with neighboring tissues or organs and is especially important for radiologists and surgeons.

Keywords: empty sella syndrome, sella turcica, CT, morphometry

P-064

Age differences in recognition of facial expressions: analysis of EEG delta responses

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Objective: Face perception/recognition is one of the most basic activities of a person's life and one of the most fundamental aspects of social communication. There are limited studies investigating the effect of age on the perception of facial expressions. In the present study, we aimed to investigate effect of age on facial expression perception by analysis of EEG Event related Oscillations.

Methods: 9 healthy young subjects and 8 healthy elderly subjects with no neurological and psychiatric diseases were included in the study. EEG was recorded with BrainAmp 32-Channel DC System. Nine photographs were selected from Ekman and Friesen (1976) series. These photographs consist of 3 different facial expressions (angry, happy, neutral) with 3 different faces. After each EEG recording session, subjects were asked to identify the each facial expression that was presented. In EEG recording Delta (0.5–3.5) frequency band were analyzed. Repeated measures of ANOVA was used for statistical analysis ($p \leq 0.05$).

Results: There were statistically significant results for group Xhemisfer ($F=6.43$, $p=.023$) and group Xlocation ($F=3.18$, $p=.053$) comparisons. Elderly subjects had reduced delta responses in comparison to younger adults over occipital (o1, o2) and parietal (p7, p8) locations. In addition, the face recognition success score of the young participant group was 8.6/9, while that of the old participant group was decreased to 6.8/9.

Conclusion: In the literature facial expression recognition were reported be related with increased delta responses over occipital areas. In the present study, elderly adults showed decreased delta responses during identification of facial expression. This decline was especially evident for the parietal and occipital lobes. Decrease of delta responses in the elderly subjects could be due to their impairment in recognizing facial expressions.

Keywords: delta, EEG, emotion, event related oscillation, facial expression

P-065

Cognitive impairment in Parkinson's disease and brain's functional connectivity networks

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Objective: Mild cognitive impairment in Parkinson's disease (PD-MCI) representing a potantial prodromal state of Parkinson's disease dementia (PDD) is the biggest risk factor for PDD. There is no reliable MRI-based biomarker available for PD-MCI and PDD which are currently diagnosed by clinical evaluation and neuropsychological tests. In order to develop a discriminative biomarker for progressive cognitive decline in PD, the resting-state functional connectivity networks (RSNs) of the PD-MCI, PDD and healthy controls (HC) were compared in this study.

Methods: 43 PD (31 PD-MCI, 12 PDD) patients diagnosed according to UK-PD Society Brain-Bank Criteria at the Behavioral Neurology and Movement Disorders Unit of Istanbul Medical Faculty and 13 HC were included in this study. MR imaging was performed on 3T Phillips MRI scanner (Achieva, Philips, The Netherlands). RSNs were obtained by using independent component analysis (ICA) in Group-ICA fMRI Toolbox (GIFT). Significant clusters with a cluster-level FWE-corrected $p<0.05$ were reported.

Results: Compared to HC, PD-MCI group displayed significantly decreased functional connectivity in the precuneus cluster of the default mode network (DMN) ($p=0.007$), while PDD patients showed significant decreases in two DMN clusters covering the precuneus ($p<0.001$) and the angular gyrus ($p=0.021$), Compared to both HC ($p=0.018$) and PD-MCI ($p=0.027$), PDD showed significantly decreased functional connectivity in middle and inferior frontal gyriof the left frontoparietal network (FPN).

Conclusion: Both PD-MCI and PDD showed decreased functional connectivity in the posterior DMN, while PDD showed decreased connectivity in the executive function related FPN compared to both HC and PD-MCI. Our results show that hypoconnectivity in DMN may represent the progressive cognitive decline in PD, while the addional FPN hypoconnectivity may be an indicator for PDD. Supported by TUBITAK #115S219 and IU-BAP #21336.

Keywords: cognitive impairment, fMRI, resting-state networks, Parkinson's disease

P-066

Effect of vitamin B12 on pentylenetetrazol-induced seizures in rats

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Objective: Epilepsy is defined as a short-lived paroxysmal disorder of the brain functions observed in seizures by sudden, abnormal and hypersynchronous discharges of a group of neurons in the central nervous system. Vitamin B12 derivatives are complex organometallic cofactors used by a limited number of enzymes. B12 vitamins are involved in many cellular functions, both glial and neuronal, in the central and peripheral nervous system.

Methods: In our study, 18 240–280 gr male Wistar albino rats were used. Animals were divided into three groups: control (n=6), 50 µg/kg /day vitamin B12 (n=6) and 100 µg/kg /day vitamin B12 (n=6). Serum physiologic to control group and other two groups were administered for seven days at the indicated doses of vitamin B12 intraperitoneally. On the seventh day, pentylenetetrazole (PTZ) was intraperitoneally injected at 70 mg/kg 45 minutes after drug administration. The animals were observed for 30 min. Stages were determined according to the racine seizure scale and the first myoclonic jerk time (FMJ) was recorded in seconds. After the procedure, animals were sacrificed of brain tissues. After routine histological follow-up, serial sections from brain tissues were stained with toluidine blue. The hippocampal CA1, CA2, and dentate gyrus regions were evaluated histopathologically.

Results: The results of epileptic behavior were evaluated according to Racine convulsion scale, the difference between the control and 50 µg vitamin B12 group was statistically significant ($p<0.01$). The first myoclonic jerk time was considered, the difference between the control and 50 µg vitamin B12 was statistically significant ($p<0.05$). When the groups were evaluated histopathologically, it was statistically significant that 50 µg B12 treatment reduced neuronal damage in CA1, CA2 and dentate gyrus regions ($p<0.05$).

Conclusion: This study suggests that vitamin B12 therapy may reduce epileptic seizures and post-seizure neuronal damage.

Keywords: epilepsy, pentylenetetrazole, vitamin B12

P-067

Antidepressant and anxiolytic effects of *Euterpe olerecea* in rats and comparison with reference antidepressant and anxiolytic drugs

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Objective: Depression and anxiety are commonly seen disorders. In addition, these disorders accompany and negatively affect prognosis and treatment response of some other medical diseases. Inflammatory processess are suggested to be involved in the pathogenesis of depression. We aimed to investigate the antidepressant and anxiolytic effects of a flavonoid compound *Euterpe olerecea* (EO) considering its antiinflammatory effects.

Methods: Wistar rats (300–350g) were randomly divided into 8 groups as below (n=8 in each group): Control (saline), EO 30, 100, 300 mg/kg, amitriptyline 20 mg/kg, fluoxetine 20 mg/kg, diazepam 5 mg/kg and ketamine 5 mg/kg. Forced swimming and elevated plus maze tests were used to investigate antidepressant activity and anxiolytic activity respectively In addition, locomotor activity of the rats were assessed with activitymeter. All the drugs were administered via oral route and tests were performed 1.5 hours later the drug administration. The results were statistically analyzed with Kruskal-Wallis test and $p<0.05$ was accepted as statistically significant.

Results: All doses of *Euterpe olerecea* significantly reduced immobility time in forced swimming and time spent in closed arms in elevated plus maze tests compared to control. The effects of 100 mg/kg *Euterpe olerecea* on these tests were similar to amitriptyline 20 mg/kg, ketamine 5 mg/kg and diazepam 5 mg/kg. On the other hand fluoxetine 20 mg/kg did not reduce immobility time and time spent in closed arms compared to all groups including control group.

Conclusion: We suggest that *Euterpe olerecea*, especially at a dose of 100 mg/kg exerts antidepressant and anxiolytic effects comparable to reference antidepressant and anxiolytic drugs used in this study. However, the ineffectivenes of the reference drug fluoxetine on depression and anxiety tests may be attributed its acute administration.

Keywords: Euterpe olerecea, antidepressant effect, anxiolytic effect

P-068

Evaluation of the P300 and N100 components of the event related potentials of Parkinson's disease with and without cognitive impairment

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Objective: Mild cognitive impairment or dementia is a cognitive disorder that is associated with Parkinson's disease and highly prevalent in the elderly. In the present study we aimed to evaluate P300 and N100 components of event related potentials (ERP) of Parkinson's disease (PD) patients with and without dementia during visual oddball paradigm.

Methods: 11 PD patients without cognitive impairment, 12 PD patients with mild cognitive impairment (MCI) or dementia and 13 healthy volunteer controls (HC) were included in the study. Visual oddball paradigm was used as cognitive stimulation. Visual Oddball Paradigm included total 120 stimuli (40 targets – rare and 80 non-targets – frequent). Electroencephalography (EEG) recording was recorded in the dimly illuminated, soundproof chamber. EEG recording was amplified with BrainAmp 32-Channel DC System. ERP responses were filtered between 0.5–30 Hz with Brain Vision Analyzer 2.0 program. Epochs with artifact were removed, then P300 and N100 amplitudes were measured from baseline to peak for each electrode and for each subject. Repeated measures of ANOVA was used for statistical analysis ($p \leq .05$).

Results: N100 peak to baseline amplitude of HC were significantly higher than PD patients without cognitive impairment and PD patients with MCI/dementia in frontal locations ($p<.05$). Furthermore, P300 peak to baseline amplitude of HC were also significantly higher than PD patients without cognitive impairment and PD patients with MCI/dementia in frontal locations. ($p<.05$). There were statistically significant results for group Xlocation Xhemisphere comparison for N100 component of vERP. ($F=2.714$; $p=.038$). There were statistically significant results for groupXlocation comparison for P300 component of vERP ($F=3.637$; $p=.037$).

Conclusion: Results of our study state that cognitive impairment of PD could be represented with ERP components and this cognitive impairment was represented with decreased N100 and P300 component of ERP. This work was supported by TÜBİTAK (grant number 214S111)

Keywords: EEG, event related potentials, N100, Parkinson's disease, P300

P-069

Haloperidol treatment causes pathological changes in striatal neurons of guinea pigs: a light and electron microscopical study

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Objective: Antipsychotic medications have been available since the mid-1950s. The older antipsychotics such as haloperidol, in the treatment of acute psychotic states may even produce side effects that resemble the more difficult-to-treat symptoms. The toxic effects of haloperidol on different brain sides are well known. But, dose levels provoking these side effects and mechanisms of neuronal injury are not clear. In the present work, we investigated whether there would be any change in histological structure of striatal neurons after haloperidol applications at different doses.

Methods: For this purpose, adult male guinea pigs were treated once a day with saline (Group 4, control) or haloperidol during 6 weeks and dose was 1, 2 or 3 mg/kg (Group1, 2 and 3; respectively). After treatment, all animals were anaesthetized and striata were dissected. Following embedding in Araldite CY 212, obtained semi-thin sections were stained with toluidine blue. Then, cut ultra-thin sections were contrasted with uranyl acetate and lead citrate, and examined in an electron microscope.

Results: When striata were evaluated histologically, dark neurons and some degenerating striatal neurons had distinctive morphological changes consistent with cell death, including reduced neuronal size with nuclear and cytoplasmic shrinkage. Also, in sections of striata in groups 1 and 2 but not in group 3, more glial cells were observed than in those of the control group. In all treated groups, fibrous content of intersititium was paralelly increased by increasing dose. Ultrastructural investigation of striatal neurons in haloperidol terated animals showed notched nuclei and many lysosomes. Moreover, degeneration of myelin, scarce microglial macrophages, expansion of nuclear intermembranous space, degenerated mitochondria, and vacuoles were found. Also cytoplasmic swelling, seconder lysosomes, and apoptotic bodies were present.

Conclusion: These results suggest that haloperidol treatment may lead to several damages in neurons via necrotic process in both low- and high-dose applications.

Keywords: haloperidol, corpus striatum, histopathology, electron microscopy, guinea pig

P-070

Important to the apparent diffusion coefficient and the diffusion value to determine the grade in the glial tumors

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Objective: Diffusion-weighted imaging (DWI) is one of the fast developing techniques in the field of MRI. This method uses the random diffusion motion of water molecule depending on physiological and anatomical characteristics of living organisms. DWI has been used to study brain tumors and response to treatment and its diagnostic potential and usefulness for obtaining apparent diffusion coefficient (ADC) have been reported. The aim of this study was to calculate the ADC and diffusion value to determine the degree in the glial tumors.

Methods: In this study, cases were selected who diagnosis of intracranial mass from cases admitted Department of Neurosurgery, Faculty of Medical, Erciyes University. A total of 20 patients were participated 7 of glioblastoma multiforme (GBM), 4 of anaplastic astrocytoma (AA), 4 of diffuse astrocytoma, 3 of ependymoma, 2 of low-grade astrocytoma in study. The ADC images were acquired from echo-planar diffusion-weighted images. ADC map images were transfered to DTI studio for postprocessing. The mean ADC values and the mean diffusion value of each tumor were measured through tumor and from normal brain which is tumor symmetry by defined large region of interest (ROI).

Results: We were calculated the mean ADC values (2.91x10–3 mm^2/s, 0.69×10–3 mm^2/s, 3.13×10–3 mm^2/s, 2.41×10–3 mm^2/s and 0.70×10–3 mm^2/s) and the mean diffusion values (0.11×10–3 mm^2/s, 1.6×10–3 mm^2/s, 0.10×10–3 mm^2/s, 1.58×10–3 mm^2/s, 0.90×10–3 mm^2/s) respectively GBM, AA, diffuse astrocytomas, low degree astrocytoma and ependymoma. The diffusion value of normal brain was calculated 0.85×10–3 mm^2/s. A P-value <0.05 was considered statistically significant. We were estimated to statistically a significant different between GBM and AA (p=0.001). Although there were a statistically significant difference between diffuse astrocytomas, low degree astrocytoma and ependymoma, not evulated statistically.

Conclusion: The mean ADC value and diffusion value, intensity region may be provide additional information in determining the grade of malignant glial tumors.

Keywords: brain tumor, ADC, DWI, ROI

P-071

Expression of TRPM2 ion channels activated by oxidative stress in medial vestibular nucleus

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Objective: Melastatin-like transient receptor potential 2 (TRPM2) ion channels are non-specific cation channels that are mostly permeable to calcium and activated by reactive oxygen and nitrogen species and ADP-ribose produced during oxidative stress. They play role in pathologies of the nervous system like Alzheimer disease and ischemic damage. Vestibular nuclei receive their input from peripheral semicircular canals and otoliths and mediate balance reflexes like vestibuloocular and vestibulocollic reflexes. Balance problems, falls, dizziness, vertigo, drug toxicity and loss of vestibular functions in aged people occur very often and most of them involve oxidative stress. This study investigated the expression of TRPM2 ion channels in the medial vestibular nucleus neurons, which are the central nervous system structures of the vestibular system.

Methods: Brainstems containing medial vestibular nucleus were surgically dissected from 4–6 week-old young adult Sprauge Dawley rats after decapitation. Brainstem slices (30 µm thick) were incubated with primary antibodies of TRPM2 channels and secondary florescent antibodies. The expression of TRPM2 ion channels in the medial vestibular nucleus was analyzed using florescent microscopes.

Results: There was strong staining with florescent antibodies against TRPM2 channel proteins especially in the soma of the neurons as well as proximal dendrites and axons. Florescent staining was also observed in the rostral and caudal parts of the nucleus, which are functionally different parts.

Conclusion: This study shows that TRPM2 ion channels are strongly expressed in the medial vestibular neurons. These ion channels can effect cytosolic calcium levels and depolarization state of the neurons and hence determine their excitability and survival rate. TRPM2 ion channels and changes in their expression level with aging may be involved in the pathological mechanism of especially aging related vestibular disorders. This study was supported by OMÜ BAP with a project number PYO.TIP.1901.12.026.

Keywords: immunohistochemistry, oxidative stress, TRPM, vestibular nucleus

P-072

Investigation of the resting state networks in adolescents with obsessive-compulsive disorder

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Objective: In the literature, there is limited number of functional magnetic resonance imaging (fMRI) studies that examine obsessive compulsive disorder (OCD) in children and adolescents. In this study, it was aimed to investigate the neural mechanisms underlying childhood or adolescent-onset OCD using fMRI. For this purpose, the resting state networks (RSNs) were compared between adolescent OCD patients and healthy controls and RSN changes associated with OCD were revealed.

Methods: 15 medication-naïve adolescents with OCD [mean age 15.27 (±1.49)] and age, gender and education level matched healthy controls participated in the study. Resting state fMRI data were collected with 3 Tesla MRI device from all participants with their eyes closed. After preprocessing, RSNs were obtained with independent component analysis (ICA) using GIFT toolbox and were compared with voxel-wise two sample t-tests between two groups.

Results: OCD group showed significant functional connectivity increase in the precuneus including a cluster of 115 voxels (MNI peak coordinate x=4, y=-68, z=38) compared to healthy controls (cluster forming threshold p <0.001 uncorrected, cluster-level FWE corrected p=0.03). No statistically significant difference was found between the two groups in other RSNs.

Conclusion: From the clinical aspect, functional connectivity increase observed in the precuneus may be related to the effort to shift attention from obsessive thoughts in OCD. Our findings indicate that parietal brain regions such as precuneus also play a role in the pathophysiology of OCD beside the orbito-fronto-striatal regions.This study was supported by the Scientific Research Projects Unit of Istanbul University (project # 42362 and project # TTU-2017-21107).

Keywords: resting state networks, obsessive-compulsive disorder in adolescents, functional magnetic resonance imaging

P-073

Effects of NO and Na+/K+-ATPase on ethanol-induced relaxations

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Objective: In this study we aimed to clarify the role of nitric oxide (NO) and sodium-potassium pump (Na+/K+-ATPase) on relaxations induced by ethanol in isolated mouse gastric fundus.

Methods: In this study, 42 mice (Swiss albino) of either sex were used (There is no prediction for either sex. It can be used either sex in gastric fundal experiments). After killing the mice by cervical dislocation, gastric fundus was isolated and prepared longitudinally. And it was mounted under 0.5 g tension in an organ bath filled with Tyrode's solution. The bath medium was maintained at 37 °C and gassed with %95O_2 and 5%CO_2. Experimental data were recorded by an isometric transducer. ANOVA (Post hoc: Bonferroni) test was used for statistical comparison.

Results: Ethanol (164 mM) caused reproducible relaxations in isolated mouse gastric fundal strips. These relaxations were significantly inhibited by Nω-Nitro-L-arginine (L-NOARG; 10–5–5×10–4 M), a potent inhibitor of nitric oxide synthase, in a concentration dependent manner. On the other hand ouabain (10–5–10–4 M), a potent and specific inhibitor of Na+/K+-ATPase, failed to affect the relaxations induced by ethanol (164 mM) in the mouse gastric fundus.

Conclusion: The results of experimental data suggest that NO may play a role on relaxations induced by ethanol in the isolated mouse gastric fundal smooth muscle. The results also suggest that Na+/K+-ATPase may not have a role on relaxations induced by ethanol in the related tissue.

Keywords: nitric oxide, Na+/K+-ATPase, ethanol, relaxation

P-074

The effect of changing sleep patterns on locomotor activity and anxiety in pregnant mice

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Objective: Circadian rhythm is the most important mechanism that expresses psychological, physiological, biological changes that occur in many mammal species in 24-hour period and determines the sleep awake cycle. This rhythm has importance in physiological processes such as tissue growth, blood pressure control, heartbeat and blood sugar regulation. It is known that rhythm disturbance resulting from change of sleeping hours during pregnancy is caused by preterm birth, miscarriage and low birth weight. For this reason, locomotor activity and anxiety levels were assessed in mice whose sleep mode was changed during pregnancy.

Methods: In this study, sixteen 6-week-old female Balb C mice weighing 25–30 g were used. The initial sleeping rhythms of all animals were set to 12-hour light: 12-hour dark. The appearance of vaginal plaque was accepted as the first day of pregnancy. On the second day of pregnancy, Open Field Test was performed on rats and all rats were separated as control and experiment. The control group continued to sleep at 12-hour light: 12-hour dark for 15 days, while the sleeping time of experiment group was taken forward 6 hour every 5 days and it was changed 3 times. At the end of the 15th day, Open Field Tests of both groups were repeated.

Results: In both groups, it was determined that distance, frequency, speed, duration in external/internal quadrant and number of defecation decreased compared to the initial values, but the decrease was only significant in the experimental group. Locomotor activity was deteriorated and anxiety was increased depending on the circadian rhythm in both groups.

Conclusion: According this data, it was determined that the change of circadian rhythm during pregnancy affects emotional state and locomotor activity adversely. It has come to the conclusion that shift workers may have adverse effects of changing sleep patterns during pregnancy.

Keywords: circadian rhythm, sleeping, open field test, locomotor activity, anxiety

P-075

MLPA and next generation sequencing for molecular diagnosis of Parkinson's disease

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Objective: Parkinson's disease (PD), which affects 1–2% of the >65-year-old population, is result of dopaminergic neuron loss at the substantia nigra. Primary findings include rigidity, bradykinesia, impaired balance, and tremor at rest. Genetic studies have underlined the importance of three different cellular pathways; synaptic communication, mitochondrial dysfunction, and lysosomal autophagy. Mutations in LRRK2 (PARK8), SNCA (PARK1), VPS35 (PARK17) genes are associated with autosomal-dominant PD forms. LRRK2 mutations usually linked to late-onset and sporadic forms, with frequency of 2–40% in different populations, while SNCA and VPS35 mutations are sparse. Recessively inherited PD has been linked to many more genes; PARK2, PARK1, DJ-1, PARK5 and ATP13A2 are the most frequently reported ones. PARK2 is associated with 10–20% of early-onset, and 77% of familial cases. DJ-1 mutations reported in 1–9% and PINK1 in 1–2% of patients. Other genes are infrequently reported. The fact that the environmental factors are the important contributors of the disease complicates the diagnoses. Our aim is to screen the known PD associated genes for mutations, reveal the frequencies, and acknowledge if genotype-phenotype relationship exists. We expect that our designed panel-gene test can contribute to the clinical diagnosis of the patients and enable families with genetic counseling.

Methods: Study group is composed of 23 familial and 22 sporadic PD patients (n=45) with the age range of 21–81 years, diagnosed by Neurology Clinic of Istanbul Medical Faculty, between the years of 2015–2017. MLPA analysis for screening of gross deletion/duplication is followed by screening of 42 genes by next generation sequencing (NGS) platform.

Results: Analysis is actively continuing and comprehensive results will be presented at the poster session. Presently, three cases with total SNCA gene duplication and two cases with PARK2 exon deletion by MLPA are identified.

Conclusion: Screening with NGS presented that three cases have missense mutations in different genes.

Keywords: Parkinson's disease, MLPA, NGS

P-076

Genome-wide target identification of neurogenic transcription factor NEUROD2 reveals novel regulatory mechanisms of the REELIN signaling pathway

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Objective: During development, cortical neurons differentiate from neural stem cells. Once born, they migrate to superficial cortical layers. This process is controlled by several transcription factors and signaling pathways. Neurogenic differentiation factor 2 (NEUROD2) is a basic helix-loop-helix (bHLH) transcription factor which controls the differentiation and physiology of neurons during cortical development. Although previous studies indicate the importance of NEUROD2 during cortex development, its target genes and biological processes controlled by NEUROD2 are unknown. Aim of this study was to explore a potential role for NEUROD2 in the neuronal migration process by identifying its genetic targets during the peak of neuronal migration.

Methods: To identify the genes bound by NEUROD2 during cortical development, we performed chromatin-immunoprecipitation followed by high-throughput sequencing (ChIP-Seq) from the developing cortices of mice. To identify those genes whose expression is dependent on NEUROD2, we carried out RNA sequencing (RNA-Seq) from neurons whose Neurod2 expression had been silenced.

Results: Computational analyses of ChIP-Seq and RNA-Seq data revealed several genes that were both bound and transcriptionally regulated by NEUROD2. Among them we selected the Reln gene for further analyses given its essential role in neuronal migration. The Reln gene encodes REELIN, which is a glycoprotein secreted by Cajal-Retzius cells, but not by the migrating neurons of the developing cortex. REELIN regulates the cellular adhesiveness of the migrating neurons. We verified that NEUROD2 binds to multiple intragenic locations in the Reln gene and silencing Neurod2 expression in primary cortical neurons causes a significant upregulation of Reln expression.

Conclusion: Our results suggest that NEUROD2 suppresses the Reln expression in migrating neurons and thereby offer a novel transcriptional regulatory mechanism for Reln expression. Given that mutations in the Reln gene both in mice and in humans cause severe defects in neuronal migration, cortical and cerebellar development; we believe that our results will have important implications for early human brain development.

Keywords: NEUROD2, Reln, neuronal migration, cortical development

P-077

A low-cost microelectrode fabrication method for multichannel extracellular neural recordings

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Objective: Microelectrode arrays (MEAs) are common multichannel interfaces for in vivo recording of neuronal activity in the central and the peripheral nervous system. According to the anatomical structure of the targeted neural tissue, MEAs can have various geometry and number of recording sites. In this work, we propose a low-cost fabrication method for microwire-based MEAs which are chronically implantable and can be used for extracellular recordings from the layer V of the rat primary motor cortex.

Methods: In the present fabrication method, polyimide insulated tungsten microwires with a diameter of 35 micron are used to form recording channels of the MEA. A stainless steel template prepared using laser milling is utilized to align the microwires parallelly with

a precise interelectrode distance of 250 micron. Using this template we prepared, the microwires can be aligned in order to form a multi-column and multi-row structure. After the alignment process, microwires are soldered onto the pins of an Omnetics connector. The positions of the microwires are fixed using an epoxy adhesive and the MEA fabrication process is completed.

Results: Using the present method, we produced a 16-channel MEA whose microwires are aligned within two rows and eight columns (2×8). The impedance range of the present MEA was measured as 0.2–1.4 MΩ (@1000 Hz) after chronic implantation into the rat brain. It is observed that the fabricated microwire array enables recordings from rat motor cortex up to two-three months after implantation surgery.

Conclusion: The present fabrication method enables an experienced researcher to prepare a 16-channel tungsten MEA within 6–8 hours. The cost of a 16-channel MEA produced using the present method is approximately $30.

Support: The Scientific and Technological Research Council of Turkey (TÜBİTAK), Grant No: EEEAG-115E257

Our research was approved by the Istanbul Medipol University Ethics Committee on Animal Research.

Keywords: extracellular recording, microelectrode array, electrophysiology

P-078

Optogenetic analysis of AGRP neurons' synaptic pharmacologies

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Objective: Feeding behavior is regulated by hypothalamic nuclei which are connected to each other with dense neural networks. In particular, AGRP (Agouti-related peptide) neurons in the arcuate nucleus play an extremely important role in appetite control. It has been shown that these neurons are necessary and sufficient to increase or decrease food intake. The link between the AGRP neurons of the hypothalamus and the other paraventricular hypothalamic nucleus (PVH) is the key to control appetite. It is possible to control the appetite modulating this connection. In this study we aimed to test the pharmacological properties of AGRP-> PVH synaptic connectivity.

Methods: We have tested the effect of strong synaptic connectivity of AGRP-> PVH on serotonin and other major neuromodulators using viral injection, optogenetic and electrophysiological methods in acute brain slices from transgenic mice.

Results: We observed that AGRP-> PVH synaptic connectivity is strongly and significantly inhibited by serotonin agonists and resulted in a change in the strength of this connection.

Conclusion: This study presented synaptic pharmavcologies of AGRP neurons.

Keywords: AGRP, arcuate, electrophysiology, feeding, hypothalamus, PVH

P-079

Serum prolidase enzyme activity in patients with acute ischemic stroke

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Objective: The aim of this study was to investigate whether serum prolidase activity (SPA) levels in acute ischemic stroke (AIS) patients can be used as a potential diagnostic and prognostic marker.

Methods: SPA levels were prospectively evaluated in 37 patients aged between 20 and 85 years who were admitted within 24 hours of the onset of AIS. The control group consisted of 37 healthy volunteers of similar age without any disease. The demographic data, biochemical parameters, neuroimaging, and cardiac evaluation were done. Stroke severity was assessed by the National Institutes of Health Stroke Scale (NIHSS) score on admission and at 24 hours, 48 hours, and 28 days after stroke. The Oxfordshire Community Stroke Project (OCSP) classification was used to determine the anatomical subtype of stroke.

Results: There was a significant difference between AIS patients and healthy controls with respect to prolidase activity ($p=0.035$). In the ischemic stroke group, prolidase activity on admission averaged 1331.39±399.007 pg/mL. Prolidase activity in the controls was 1169.14±221.729 pg/mL. SPA was not correlated with age, gender, hypertension, diabetes, triglycerides, total cholesterol, low-density lipoprotein, high-density lipoprotein, hemoglobin, c-reactive protein, or hemoglobin A1c levels ($p>0.05$). However, it was positively correlated with the presence of atrial fibrillation ($p=0.032$). SPA levels were also uncorrelated with NIHSS, infarct volume, Trial of Org 10172 (TOAST) and OCSP classifications, and duration of hospitalization ($p>0.05$).

Conclusion: Increased levels of serum prolidase enzyme activity may contribute to ischemic stroke pathophysiology and may be an independent predictor of the disease. However, further studies in larger patient groups are needed to explain the role of SPA in AIS.

Keywords: acute ischemic stroke, prognosis, prolidase activity

P-080

Evaluation of side effects in the use of fingolimod: one-center experience

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Objective: The aim of this study is to evaluate our clinical observation regarding the side effects of the fingolimod.

Methods: Forty-three patients who were diagnosed with relapsing-remitting multiple sclerosis (RRMS) and treated with fingolimod were included in our study. Patient records were retrospectively reviewed and the fingolimod-associated side effects were assessed. Patient demographics, duration of MS, Expanded Disability Status Scale scores, previous MS specific therapies, and duration of fingolimod use were recorded. The time of appearance and termination of these side effects, necessity for symptomatic treatment or the necessity to stop fingolimod treatment were noted.

Results: All patients were in the clinical form of RRMS and the duration of fingolimod use ranged from 1 to 41 months. One patient developed grade 1 AV block and symptomatic bradycardia. During treatment, 4 cases of hair loss, 5 cases of menstrual disorders, 1 case of over cancer, 3 cases of skin lesions (1 patient developed severe shingles) were observed. We needed to stop fingolimod in one patient out of 5 patients who developed drug-related lymphopenia. Liver enzymes were mildly elevated in 1 patient. In 1 patient, central retinal artery occlusion accompanying macular edema was observed and we stopped fingolimod. During the follow-up period, 85% of the patients continued to use fingolimod, 15% needed a change in MS treatment due to ineffectiveness or side effects of fingolimod.

Conclusion: We evaluated the side effect profile of fingolimod in MS patients. Most of the side effects were mild. However, 4 severe side effects leading to drug withdrawal were observed.

Keywords: multiple sclerosis, fingolimod, side effect

P-081

A Turkish family with X-linked hereditary spastic paraparesis

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Objective: Hereditary spastic paraparesis (HSP) are rare progressive neurodegenerative diseases that are symptomatic with weakness and spastisity of low limbs muscles. Inheritance may be of different forms including autosomal dominant (AD), autosomal recessive (AR), X-dependent or mitochondrial.

Methods: In this article we submit a 41 year-old-patient presented with progressive gait disturbance that diagnosed by HSP and five family members.

Results: The index case was refered with thinning of the right leg after the second decade, leg pain and progressive gait disturbance since the last six months. There was no feature in his history. Parents was the same village but there was no relationship. In the family history, it was learned that father, three uncles and grandfather had a gait disturbance. On neurological examination; high palate, paraparesis (4/5 motor force present), bilateral low limbs significantly atrophic and spastic, spastic-gait, deep tendon reflexes were increased on all four extremities, and bilateral extensor plantar response were found. Cranial and spinal imaging was normal, and CK level was 63 (Normal value: 20–200 mU/L). Neurological examination of her mother, 96-year-old grand-mother, a 72- and 68-year-old hawk were normal, in the screening of family members. There were similar findings in the case of 61-year-old uncle, 54-year-old father, 60-year-old uncle, and 51-year-old uncle. Inheritance was evaluated by mitochondrial transition with pedigree study and directed to genetic analysis, the exome analysis is ongoing.

Conclusion: HSP is diagnosed with spasticity and muscle weakness predominantly in the lower extremity following normal neurological development, hyperreflexia and the presence of extensor plantar response, exclusion of acquired causes, and / or the presence of family narratives. Having such a wide range of genetic pool prolongs the genetic analysis process of the disease and makes inadequate the available opportunities.

Keywords: Hereditary spastic paraplegia, X linked, genetic

P-082

The analysis of visuo-spatial orientation ability in Parkinson's patients

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Objective: Parkinson's is a chronic and progressive disease, which is mostly prevalent after the age of 60 and characterized with tremors, muscle stiffness and postural problems. In addition to motor disabilities, psychopathological problems such as depression and anxiety as well as cognitive problems such as visuo-spatial disabilities are also seen in Parkinson's disease. The aim of this study is to measure route finding skills, a dimension of visuo-spatial ability, in Parkinson's patients of different stages. We also tried to understand the possible relation between the degree of this disability and gender, education level, caregiving type they obtain, heredity and the duration of the disease. We believe our results could lead the way to a better understanding of the cognitive decline in Parkinson's disease and novel neuropsychological therapies.

Methods: Parkinson's patients of different age (50–95), gender, education level, caregiving type (family, care home, etc.) participated in the study. The patients and the control group completed a clock-drawing test and route finding test (4 mazes from easy to hard). The results and the completion times were evaluated for each participant.

Results: In the clock-drawing test, we frequently observed hemi-neglect, numbering and spacing problems. In the route finding test, mostly repetitive drawing mistakes were observed and rarely lines crossing the maze were detected. We found that clock-drawing and maze test results, indicators of two different cognitive abilities, did not correlate with each other. On the drawings, skewed lines were observed as a result of hand tremor. Some patients had more excessive hand tremor, while

others were better at drawing but had lower scores in cognitive tasks.

Conclusion: We found that loss of motor and cognitive abilities showed a variety in Parkinson's patients of different age, gender, family history, caregiving, and the loss of these abilities were not correlated with each other.

Keywords: Parkinson's disease, visuo-spatial orientation, motor control, neuropsychology

P-083

Establishment of an organotypical spinal cord slice culture

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Objective: Organotypic spinal cord slice culture represents an ideal experimental approach to investigate neuronal maturation and physiology of the spinal cord. In this respect, the behavior of cells in different physiological processes such as proliferation, differentiation, cell migration can be examined in *ex-vivo* conditions. The aim of this study is to establish the spinal cord cross-sections cultivated in our laboratory, allowing for analysis of the traumatic spinal cord injury in *ex-vivo* conditions.

Methods: Spinal cord isolated from four-week-old C57BL/6 mice was housed in artificial cerebrospinal fluid at +4°C. Tissue sections with a thickness of 500 µm were taken using spinal cord matrix. The medium used for spinal cord/neuron culture consisted of 50%-MEM, 25mM-HEPES, 25%-horse-serum, 25.5 mg/ml-D-glucose, 25%-HBSS-without glutamine, 1%-glutamax, 1%-antibiotic solution. Spinal cord slices were placed on culture inserts placed in petri dishes. The slices maintain their viability up to 24 days in these conditions. Cell morphology and typing was done after the structural integrity of the slices was determined. For this procedure, neural cells in the spinal cord were marked by immunofluorescence and visualized on a fluorescent microscope. In addition, viability analysis was performed with MTT. Also, spinal cord injury culture was achieved by weight reduction and the assessment of injury was evaluated with the same methods.

Results: Spinal cord slices acquired structural integrity on the 4th day of culture. The cross-sectional thickness and age of the experimental animal were effective at the rate of culture success. Our research team created a spinal cord cross-sectional culture and *ex-vivo* conditions for nerve injury models.

Conclusion: In this respect, the activity of the compounds synthesized for therapeutic purposes can easily be determined in the organotypic culture medium under physiological conditions. It also allows the damaged tissue to be analyzed and the newly developed support materials to be examined for repair processes.

Keywords: organotypic culture, spinal cord culture, trauma

P-084

The role of nicotinic receptors in the effects of galangin in the Morris water maze

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Objective: Enhancing cholinergic transmission is suggested to improve cognitive functions. We aimed to investigate the effects of galangin, a flavonoid compound that is reported to inhibit acethylcholinesterase enzyme,on mecamylamine-induced spatial memory impairments in rats.

Methods: Morris water maze test was used to investigate the spatial memory. Galangin 50 and 100 mg/kg was administered acutely 30 minutes before the impairment of spatial memory by a nicotinic receptor antagonist mecamylamine injection. Donepezil 1 mg/kg used as a reference drug. Distance to platform and time spent in escape platform quadrant were recorded and analyzed with Ethovision XT version 9.0 (Noldus, Wageningen, Netherlands). Results were statistically analyzed with one-way ANOVA.

Results: Mecamylamine significantly increased the distance to platform and decreased the time spent in the escape platform quadrant compared to control group. Galangin 100 mg/kg significantly decreased the distance to platform and increased the time spent in the escape platform quadrant compared to mecamylamine group comparable to donepezil 1 mg/kg.

Conclusion: Galangin 100 mg/kg may improve memory comparable to donepezil and nicotinic receptors may be involved in this effect. This study is a part of an ongoing project which is supported by the Commission of Scientific Researches Projects in Eskisehir Osmangazi University (Project number: 2013-93).

Keywords: galangin, mecamylamine, memory

P-085

Developing an easy portable alternative method for diagnosis and follow-up of movement disorders

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Objective: The motor system is an important element that provides the ability to synthesize relocation and synthesis of various complex movements in humans. Therefore, it is essential, for the human health, to monitor its development both quantitatively and qualitatively. The conventional methods serving the purpose, are bound by limitations such as immobility, cost of the devices and difficulty in evaluation of the results. In the present thesis work, the primary aim was to develop an intuitive, portable and cost-effective method capable of assessing the motor system quantitatively by using a prototype accelerometer device.

Methods: The device, used in the study, has been designed and prototyped in our unit (Ilhan and Puralı, 2010). It is essentially, a miniature three dimensonal acceleration measurement system. It has up to 6 channels, and has been downsized to easily fulfil its medical use. Its sensors can be attached to individual fingers and all extremities easily. The device is consisted of 4 main components: sensors, central processor, USB connection module and computer interface. In the present study, the effectiveness of the device was firstly tested on a peripheral neuropathy patient group. The acceleration responses in response to hand gesture protocols designed based on established anatomical and functional information, were recorded. The recordings were compared to those recorded in the control group and post-operation records of the patients.

Results: Analyses indicated that there is a statistically significant difference between the patient and control groups (p=0.0398), especially when Protocol2 responses were compared.

Conclusion: This is a ground-breaking study. The results indicated that the novel method is capable identifying the differences between the patients and the healthy individuals in peripheral neuropathy cases though the number of patients was limited. The next steps will be improving the operation range and resolution power of the device, and using it on the other movement disorders and disease groups.

Keywords: accelerometer, peripheral neuropathy, biophysics

P-086

Analysis of neurotoxic effects of acrylamide regarding immunohistochemical markers

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Objective: Analysis of the immunohistochemical markers in acrylamide studies and comparison of their use in the nervous and other systems.

Methods: By using keywords of "acrylamide" and "immunohistochemistry", the articles were scanned in the Pubmed, Web of Science, Sciencedirect and Google Scholar. Of 432 articles, relevant articles were selected and split into two categories as the studies of nervous system and other systems. The studies of cerebral cortex, striatum, hippocampus, midbrain, ganglion, nerves, spinal cord, cerebellum, cell culture, pineal gland and eye were included in the nervous system, other studies were included in other systems.

Results: The number of immunohistochemical studies of acrylamide was 31 in the nervous system and 17 in other systems. Acrylamide was most commonly studied in the cerebellum and cerebral cortex in the nervous system. In other systems, reproductive system, especially the testis is the leading organ to be studied; it is followed by the tumor formation, digestive system and cell culture studies. The most commonly used markers in the nervous system include GFAP, synaptophysin, NeUN, neurofilament markers, GABA, reelin, PCNA, α-tubulin, BrdU, and GAD65. In other systems, Ki-67, kaspaz 3, 8-OHdG, γH2AX, iNOS, PCNA were mostly utilized markers. CRGP, α-tubulin, kaspaz3, iNOS and 8-OHdG were used both in nervous and other systems.

Conclusion: The number of immunohistochemical studies of the nervous system are more than the other systems since the effect of acrylamide on the nervous system are known for a long time. Cerebellum and cerebral cortex are the most studied organs, and GFAP, Neun, GABA and neurofilament markers are the most commonly used markers. Immunohistochemical studies of acrylamide in reproductive system (particularly testis) are the second leading studies and different markers were used. We consider the markers we mentioned in this study will facilitate the researchers in the immunohistochemical studies of acrylamide.

Keywords: acrylamide, markers, immunohistochemistry, nervous system

P-087

Customizable and low-cost voltammetry system for in vivo measurement of dopamine concentration

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Objective: Fast-scan cyclic voltammetry (FSCV) is an electrochemical technique which enables measurement of fluctuations in dopamine concentration with high temporal resolution. For application of FSCV in neuroscience research, there are commercially available systems. However, these systems are generally expensive and their customizability according to the research requirements is limited. In this work, our aim was to develop a customizable and low-cost FSCV system for enabling measurements of dopamine transients in the rats chronically implanted with carbon fiber microsensors.

Methods:A microcontroller is at the centerpiece of the present system and controls the shape and amplitude of the voltammetric signal applied to a carbon fiber microsensor. The amplitude and waveform of the applied signal are specifically selected in order to lead to selective oxidation and reduction of dopamine. Current values appearing as a result of these redox reactions are recorded using a PC and dopamine concentration in the medium is calculated using these current values.

Results: One rat was chronically implanted with a microsensor in the dorsal striatum for measurement of dopamine concentration and a bipolar stimulating electrode in the nigrostriatal pathway for stimulating the dopaminergic neurons during the measurements. One month after implantation surgery, voltametric recordings were realized using the present system. During the recordings, square shaped 4-ms-long biphasic current pulses with amplitude of 400 μA were applied to the stimulating electrode for stimulation of the dopaminergic neurons. Based on the recordings, we measured that the dopamine concentration at the tip of

the microsensor increased approximately 150 nM as a result of the stimulation of the dopaminergic neurons.

Conclusion: Our results with in vivo experiments indicate that the present system provides a low cost solution for measurement of dopamine concentration with subsecond temporal resolution. The cost of the present system is approximately $1200.Approved by IMU Ethics Committee on Animal Research.

Keywords: voltammetry, dopamine, electrical stimulation

P-088

Effect of subchronic vitamin D treatment on pentilentetrazol-induced epileptic seizures in mice

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Objective: Epilepsy refers to heterogeneous group of neurological disorders characterized by seizures caused by recurrent, triggered electrical discharges in the cortical and subcortical regions of the brain. Epilepsy is serious disease affecting about 1% of the world population and has high mortality rates. Derived from skin and activated in the kidney, vitamin D has an important role in calcium and phosphate homeostasis. However, current studies have shown vitamin D is locally activeted in the brain and has important neuronal functions. Although vitamin D and acute administration of icv have been shown to reduce epileptic seizures, no studies have been published on systemic and subchronic administration. In this study, we aimed to investigate the effects of supchronic vitamin D treatment on pentilentetrazol-induced epileptic seizures.

Methods: In our study, 21 35–40 gr BALB-c albino male mice were used. Animals were divided into three groups: control (n=7), 1 µg/kg/day vitamin D (n=7) and 2 µg/kg/day vitamin D (n=7). Serum physiologic to control group and other two groups were administered for seven days at the indicated doses of vitamin D intraperitoneally. On the seventh day, pentylenetetrazole (PTZ) was intraperitoneally injected (60 mg/kg) 45 minutes after drug administration. The animals were observed for 30 min. Stages were determined according to the modified Racine seizure scale and the first myoclonic jerk time (FMJ) was recorded in seconds.One-Way ANOVA was used for statistical analysis and Post Hoc analysis was performed with the Tukey test.

Results: The results of epileptic behavior were evaluated according to modified Racine convulsion scale, the difference between the control and 1 µg vitamin D group-2 µg vitamin D was statistically significant ($p<0.05$). The first myoclonic jerk time was considered, the difference between the control and 1 µg vitamin D group was statistically significant ($p<0.05$).

Conclusion: We think the appropriate dose of supchronic vitamin D therapy may reduce epileptic seizures.

Keywords: epilepsy, pentylentetrazole, vitamin D

P-089

Effect of vitamin E on the epileptiform activity induced by penicillin in rats with short-term maternal deprivation

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Objective: Epilepsy is a neurological disorder characterized by spontaneous, recurrent seizures caused by abnormal and excessive discharges of cortical neurons. Maternal deprivation can be defined as loss of the care of the mother to the infant, which may have intense or long continued even irreversible effects on the baby's brain activity. Vitamin E is an essential fat-soluble antioxidant. This study investigates the effect of vitamin E on the penicillin-induced epileptiform activity in rats that are deprived of mothers for short-term.

Methods: In this study, 20 male Wistar rat puppies aging 2 months were used. The puppies in the first group (n=10) were separated from their mother and received vitamin E injection, while the puppies in the second group (n=10) were not separated from their mother and received vitamin E injection. The left cerebral cortex was exposed by craniotomy under urethane anesthesia (1.25 g/kg). Silver ball electrodes were placed over the cortex and connected to a data acquisition system for electrocorticography recording. The epileptiform activity was induced by intracerebroventricular injection of penicillin G (500 IU/2.5 µl). Vitamin E (200 mg/kg) was given intraperitoneally 30 minutes before penicillin G injection.

Results: Vitamin E did not cause any significant difference in the frequency and amplitude of the epileptiform activity between puppies exposed to maternal derivation and those with no maternal deprivation ($p>0.05$). The mean frequencies of epileptiform activity between 60th and 65th minutes after injection in the first and second groups were 42±5 (spikes/min) and 39±6 (spikes/min), respectively. The mean amplitudes of epileptiform activity between 60th and 65th minutes after injection in the first and second groups were 457±44 µV and 544±194 µV, respectively.

Conclusion: Vitamin E has no effect on the epileptiform activity in puppies with short-term maternal deprivation.

Keywords: epilepsy, maternal deprivation, rat, vitamin E

P-090

Could thymoquinone be used as a rescuer in the amyloid beta-induced neurotoxicity?

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Objective: Alzheimer's Disease, characterized by synaptic deficiency, neuronal damage and loss in the brain is the most common neurodegenerative disease in aged population. Nowadays, investigations of neuroprotective substances preventing Alzheimer's Disease-induced neuronal damage gain importance. One of these candidate molecules is Tymoquinone (TQ), an aromatic hydrocarbon found in Nigella Sativa. The aim of the present study was to examine the molecular principles of the neuroprotective effects of TQ in the hippocampus of rats which have infused with Aβ peptide.

Methods: An infusion canula linked to a micro-osmotic pump including aggregated Aβ was placed into the hippocampus of 6 month-old rats. During 15 days, TQ at a dosage of 20 mg/kg/day were intubated intragastrically. After behavioral tests, the rat brain sections were analysed by anti-Aβ antibody and Congo red staining for the formation of amyloid plaques. Also, molecular effects of TQ in the treatment of Alzheimer pathology were evaluated by RT-PCR and Western blot, in order to propose a potential mechanism, several elements of survival and growth.

Results: TQ increased the cell survival and decreased Aβ plaques in the hippocampus. The mRNA expression level of APP, BACE-1, PSN1 and PSN2 genes did not change in both Aβ induced and TQ treated animals. TQ administration decreased the expression levels of synaptophysin and NGF in the Aβ infused animals. Expression levels of phosphorylated AKT, ERK and JNK decrease while GSK increases. Pro-survival protein Bcl-2 levels increase after amyloid beta induction and further increase after TQ treatment. Pro-apoptotic Bax protein shows an opposite pattern of expression.

Conclusion: According to present results, TQ is effective in the clearance of Aβ plaques to increase the survival rate of neurons. On the protein level activity of TQ seems to be regulated by complex mechanisms. However, these molecular effects did not reflect to behavioural results.

Keywords: thymoquinone, Alzheimer's disease, amyloid beta, RT-PCR, Western blot

P-091

Antiinflammatory effect of levetiracetam in the HET-CAM assay

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Objective: Several lines of evidence have indicated that inflammatory processes within the brain contribute to epilepsy. And also, there is a linkage between epilepsy and vascular network in the brain. Evidences also have indicated antiinflammatory effect of the antiepileptic drug levetiracetam in various conditions. In this study, we aimed to investigate antiinflammatory effect of levetiracetam on small blood vessels via HET-CAM assay as an in vivo model for chronic inflammation.

Methods: The antiinflammatory activity of levetiracetam (50 µg/pellet) was evaluated in vivo using the HET-CAM assay in fertilized hens' eggs. Chronic inflammation was induced by sodium dodecyl sulfate (SDS). For the evaluation of the effect, a scoring system was used: formation of granuloma, degree of vascularization of granuloma and grade of starlike network of capilaries around granuloma were evaluated and scored at values between 1–4. Scoring was followed by the conversion of the score index in the proportional inhibition of inflammation. 17 eggs were utilized for the experiment. As control, SDS 50 µg/pellet were also tested (n=6).

Results: In the HET-CAM assay, SDS induced a characteristic strongly vascularized granuloma with starlike capillaries surrounding the pellet. SDS applied with levetiracetam showed variable effects including normalization of the irritation to barely inhibitory effects on SDS induced irritation. Percent inhibition of inflammation by levetiracetam was found to be 62.5, which is described as weak antiinflammatory effect in the inhibition scale.

Conclusion: The HET-CAM assay exposes the chorionic membrane of embryonated chicken eggs to test compound and evaluates acute effects on the small blood vessels. Antiinflammatory effect observed in this study points out to the possibility that levetiracetam may be effective in epileptic conditions via an antiinflammatory effect on blood vessels and proteins of this soft tissue membrane.

Keywords: chorioallantoic membrane, levetiracetam, antiinflammatory agents

P-092

The attenuation of oxidative stress of retina by environmental enrichment in ischemia induced by bilateral carotid artery occlusion

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Objective: Numerous studies have proven that enriched environment could reduce lesions induced by toxic, ischemic, and traumatic injuries. The model of bilateral carotid occlusion (2VO) is a well-established model for chronic brain hypoperfusion leading to brain capillary pathology in general, retina and optic nerve degeneration in particular. Retinal ischemia leads to the degeneration of all retinal layers through a complex mechanism involving oxidative damage as well. Therefore, we investigated the effects of environmental enrichment on oxidative stress in rat retina exposed to 2VO.

Methods: Male Wistar rats weighing 250–300 g were subjected to permanent carotid artery occlusion bilaterally. Animals were randomly divided into groups of sham control, 2VO +Standart environment (SE), and 2VO+enriched environment (EE) for 4 weeks. Each group consisted of 8 animals. Oxidant and antioxidant status of rat retina was assessed by measuring the levels of malondialdehyde (MDA), reduced glutathione (GSH) and ascorbic acid. One-way analysis of variance

(ANOVA) was applied for statistical comparison of the groups, followed by analysis with Bonferoni test to determine differences between the groups.

Results: Environmental enrichment significantly reduced malondialdehyde and GSH levels in EE group compared to SE group ($p<0.05$). Ascorbic acid level was found decreased in 2VO groups compared to control group ($p<0.05$). EE has no efffect on ascorbic acid levels in retina ($p>0.05$).

Conclusion: The present study suggests that EE might be effective for reducing retinal oxidative stress induced by 2VO in rat.

Keywords: environmental enrichment, oxidative stress, retina

P-093

Investigation of *NOTCH3* and *HTRA1* gene mutations in CADASIL / CARASIL patients

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Objective: CADASIL and CARASIL describe the two different inherited forms of Cerebral Arteriopathy with Subcortical Infarcts and Leukoencephalopathy that are autosomal dominant and recessive, respectively. Both are characterized by recurrent ischemic attacks, stroke, and migraine with/without aura, cognitive decline, dementia, psychiatric manifestations. Clinical diagnosis is supported by MRI, hyperintensities occuring in periventricular and deep WM even in anterior temporal area and capsula externa. CADASIL is associated with mutations in *NOTCH3 (Notch homolog 3)* with 33 exons, located at 19p13.12. Exons 2, 3, 4, 5, 6 and 11 harbor hotspot region for mutations. CARASIL, with great similarity with CADASIL in terms of clinical presentation, begins earlier in cognitive decline and characterized with gait disturbances, back pain and alopecia. Responsible gene is *HTRA1 (HtrA serine peptidase 1)* with nine exons, located at 10q26.13. There are a few publications with *NOTCH3* and *HTRA1*, investigated in concert. Studies in Turkish population are mostly case reports, dealin with hotspot regions of *NOTCH3*, and emphasize the intra familial variability. In this study, we aim to reveal *NOTCH3* and *HTRA1* gene mutation frequencies in our country, investigate genotype-phenotype correlation in cases with CADASIL/CARASIL. We further expect to develope the most effective algorithm for molecular diagnosis.

Methods: In this study, 24 familial and 29 isolated cases (n=53) with CADASIL at Neurology Clinic of Istanbul Medical Faculty between 2015–2017 are initially screened for hotspot regions of NOTCH3, by Sanger. Mutation negative cases are investigated for other regions of *NOTCH3* by Next Generation Sequencing (NGS) that includes 42 dementia-panel genes. Mutation unidentified cases are further screened foe-r *HTRA1* gene by Sanger sequencing.

Results: Three familial and two isolated cases are found to have known mutations in *NOTCH3* at the initial targeted screening test.

Conclusion: Our investigation is presently continuing and results will be presented at the poster session.

Keywords: neurogenetics, CADASIL, CARASIL, mutation, next generation sequencing (NGS), Sanger sequencing

P-094

The opening of sinus rectus to right sinus transversus: a case report

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Objective: The veins which drain venous blood of brain into internal jugular vein are dural venous sinuses, locate between periosteal and meningeal sheets of dura mater. Dural sinuses are classified in supero-posterior and infero-lateral groups according to their localizations.Straight sinus belonging to supero-posterior group, traces posteriorly and inferiorly along the margin which cerebellar tentorium and cerebral falx merge. Straight sinus, also incorporates great cerebral vein (Galen vein) and superior cerebral veins at proximal. Straight sinus continues as left transverse sinus along infero-posterior line by external occipital protuberance.

Methods: A pediatric patient, born in 2011, was referred to our clinic by the reason of cranial venous thrombosis diagnose from department of neurology in February of 2015.

Results: No obvious thrombus was detected in sinuses after contrast enhanced venous MR angiography examination. Maximum intensity projection images were created. It was noticed, superior sagittal sinus continues as transverse sinus along left side nearby internal occipital protuberance. Nevertheless, it was detected that straight sinus merges with transverse sinus by turning right at the point of 15.8 mm superior and 9.7 mm lateral of internal occipital protuberance.

Conclusion: Superior sagittal sinus is generally drained into right transverse sinus. In current case it was detected that superior sagittal sinus opened into transverse sinus. There isn't much information in literature about non-anatomical trace of transverse sinus. It is a rare variation of left transverse sinus in the form which it traces nearby internal occipital protuberance without having relationship with it, after getting along the margin which cerebral falx andcerebellar tentorium merge. During the occipital transtentorial approach, getting in via merging point of falx and tentorium from occipital pole without damaging straight sinus, superior sagittal sinus or transverse sinus. The information of that uncommon trace as important as to be taken into consideration during interventional procedures.

Keywords: sinus rectus, sinus transversus, variation, anatomy

P-095

Importance of relationships between surrounding structures of hypoglossal canal

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Objective: The aim of this study was to evaluate the hypoglossal canal morphologically and emphasize the importance of relation to adjacent anatomical structures, which may affect transcondylear surgical approach.

Methods: In the study, 25 adult human skulls belonging to Uludağ University Anatomy Laboratories were evaluated. Totally 21 parametres includingthe measurements of hypoglossal canal and the distances between the canal and surrounding structures were evaluated.

Results: The distance between the hypoglossal canal and anterior border of the occipital condyle was found 15.8±2.8 mm on left side and 15.8±3.1 mm on right side. The external distance between the hypoglossal canal and the anterior border of the occipital condyle was found 11.5±2 mm on left, 11.6±2 mm on right. The distance between the hypoglossal canal and the posterior margin of occipital condyle was 11.7±1.8 mm on left and 12.2±2.1 mm on right. Furthermore, the distance measured externally between same anatomical structures was found as 14.04±1.89 mm and 14.4±1.6 mm on the left and rights respectively. We also found a positive correlation between the length of foramen magnum and occipital condyle. In addition, the diameter of foramen magnum and the distance between hypoglossal canal and anterior margin of the occipital condyle was positively correlated.

Discussion: In transcondylar approach, the locations of important anatomical structures must be well known to perform safe occipital condyle resection without harming the neural tissue. Especially, surgeons should pay attention to the distances between hypoglossal canal and anterior and posterior margins of the occipital condyle in order to prevent from the hypoglossal nerve injury. The length of occipital condyles may affect the success of the operation during condylectomy. A detailed anatomical assessment is needed before the similar procedures in order to aviod surgical injuries.

Keywords: hypoglossal canal, transcondylear approach, occipital condyle

P-096

The surface and intracranial localization of asterion

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Objective: Asterion is identified as the connection point of parietomastoid suture, occipitomastoid suture and lambdoid suture. Asterion, the intersections of these sutures, is primarily preferred as a landmark during posterolateral surgical approach for intracranial procedures. Assignment the localization of asterion, which varies considerably, is important for surgeons because of proximity with transverse sinus. The aim of this study was to evaluate distances between asterion and intracranial anatomical structures.

Methods: At this current study, 11 half dry skulls situated at the laboratory of Uludağ University, Anatomy Department were used. According to the proximity of asterion with transverse sinus, three different groups were classified. Nineteen parameters were measured by Image J software on digital photographs. The thickness of cranium where the asterion located was measured by using mechanical caliper. The obtained data were analyzed in SPSS 22.0 the statistical software.

Results: The distance between asterion and sigmoid sinus, internal auditory meatus, confluence of sinuses, sigmoid-transverse sinus junction, jugular foramen were measured as 26.1±4.56 mm, 43.23±6.26 mm, 56.32±8.72 mm, 14.43±4.61 mm, 42.62±5.75 mm respectively. It was identified that, 15 asterion points were at the surface, 5 of them were aproximately 3.42±2.52 mm above the projection of transverse sinus and 2 ones were aproximately 3.21±2.26 mm below of the projection of transverse sinus. There was no significant difference between the measurements in left and right sides.

Conclusion: Asterion is an important landmark for retrosigmoid surgical approaches. The surface and intracranial relationships and distances of asterion and proximity with dural sinuses are important for surgeons especially during the "Burr Hole" technic of neurochirurgy.

Keywords: asterion, retrosigmoid approach, Burr hole

P-097

Compening of learning memory performance in aged rats with hypothyroidism and hyperthyroidism

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Objective: In the projects supported by TUBITAK and BAP, our research group has been investigating how learning and memory are influenced in thyroid hormone disorders occurring at adulthood over 8 years by using behavioral, electrophysiological and molecular methods. The results indicated impaired learning and memory in Morris water maze task, decreased LTP and enhanced LTD in adult rats with thyroid hormone imbalance. In aging, thyroid hormone is closely associated with learning and memory disorders, especially with hippocampal-depended spatial memory. The aim of this study was to assess learning and memory dysfunction in thyroid hormone disorders during aging.

Methods: In 10-months old male Wistar rats (n=13), hypothyroidism was induced by giving PTU for 2 months whereas

hyperthyroidism by giving intraperitoneal thyroxin for another group at the same duration. Learning and memory performances were assessed in Morris water maze task and behaviors during this task were recorded by NOLDUS monitoring system. In all rats, plasma T3 and T4 levels were measured.

Results: Plasma T3 and T4 levels were found to differ significantly in aged rats with hyperthyroidism and hypothyroidism. It was found that mean path length spent to find platform in 4 trials showed significant decrease from day 1 to day 4 ($F3.126=41.077$; $p<0.001$), and significant day-group and trial-group interaction ($F2.44=6.529$; $p<0.001$) during learning period. A significant difference was found regarding percent time in target quadrant.

Conclusion: It was also detected that there were significant differences in learning and memory performances among euthyroid, hypothyroid and hyperthyroid groups and that thyroid status significantly decreased learning and memory performances. This study was supported by Erciyes University Research Foundation (TYL-2016-6549, TCD-2016-6262).

Keywords: learning, memory, Morris water maze, aging, hypo- and hyperthyroidism

P-098

The effect of post-learning REM sleep deprivation on hipocampal REST and miR-9 expression in mice

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Objective: Making lasting memories in the brain is thought to occur during the memory consolidation process of sleep. It has been shown that REM sleep deprivation leads to impairment of spatial memory function at a specific time after learning. The gene silencing transcription factor REST represses neuron-specific genes important to the synaptic plasticity and targeted by miR-9,a brain-specific microRNA. The aim of study is to determine specific time period of sleep, using a spatial memory test and to investigate the effect of REM sleep deprivation on REST and miR-9 expression in hippocampus.

Methods: 50 male BALB/c mice aged 2 month and separate groups of mice (n=10) were sleep deprived (SD) in one of the two after the last training session the mice were deprived of sleep for 3 h (SD1), in the second group after the last training session and a waiting period of 3 h the mice were deprived of sleep for 3 h (SD2) and the last group was Non-sleep deprivation (NSD) as control group. REM sleep was eliminated by using the modified multiple platform method.Quantitative RT-PCR was used to measure changes in RNAs. Repeated-measures ANOVA was used to analyze the changes in Distance Moved (DM) and Escape Latancy (EL). Probe trial (PT) were analyzed using oneway ANOVA. For analyses of RNAs were used ANOVA and Kruskal–Wallis H test.

Results: We found that DM and EL reduced in NSD and SD1 ($p>0.05$), but these parameters were higher in SD2 ($p<0.05$). In SD2,PT was found lower than NSD. REST mRNA was lower in sleep deprivation groups ($p>0.05$),decrease in miR-9 in SD1 was significant ($p<0.05$).

Conclusion: Our findings indicate a fundemental period, extending from 3 to 6 h after last training, during which sleep deprivation impairs spatial function. SD may enabling a consequence that REST down regulated in hippocampus. Also SD may cause dramatic changes in hippocampal miR-9 levels.

Keywords: memory, REM sleep deprivation, REST, miR-9

P-099

The effect of low calorie diet administered during adolescence on spatial learning/memory and hippocampal asymmetry; evaluation in terms of neurogenesis

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Objective: Hippocampus, which plays an important role in spatial memory, is a dynamic brain region in which production of new neurons (neurogenesis) continues through lifetime. It is known that hippocampi have both anatomical and functional asymmetry. Although neurological basis of the differences between cognitive functions (including spatial cognition) in children and adults are being associated with hippocampal asymmetry, this issue is not yet clear. Hippocampal asymmetry formation starts in intrauterine period, develops during adolescence and is retained in adulthood. It is accepted that hippocampal asymmetry is an indicator of a mature and an active brain. We earlier showed that low calorie diet (LCD) application during adolescent period increases hippocampal neurogenesis and improves memory performance in adulthood. This present work is the first study that investigates the effect of LCD on hippocampal asymmetry formation.

Methods: Four different diets were applied to female Sprague Dawley rats starting from 28th day after birth: 4 weeks standard diet (SD) (n=6), 4 weeks LCD (n=8), 8 weeks SD (n=6), 4 weeks LCD+4 weeks SD (n=8). Groups were taken to Morris Water Maze (MWM) test, which investigates spatial memory, after related diet. Proliferated cells in dentate gyrus where hippocampal neurogenesis occurs marked with PCNA (proliferating cell nuclear antigen). In each group, data of right and left hippocampus were compared with Mann Whitney test.

Results: According to the results obtained, right hippocampus had more proliferated cells than left hippocampus in each group,

the difference was statistically significant in the group 4 weeks LCD+4 weeks SD ($p<0.001$). In this group, spatial memory performance was better.

Conclusion: This result suggested that LCD strengthens age-related hippocampal asymmetry and improved spatial memory depending on asymmetry. These original results draw attention to importance of neurogenesis for formation of hippocampal asymmetry and may be useful for future studies investigating treatment approaches to neurocognitive diseases.

Keywords: hippocampus, neurogenesis, asymmetry, adolescence, low calorie diet

P-100

Dependency of the dynamics of L-type calcium current on the time constant

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Objective: To calculate the changes in the dynamics of the calcium concentration with respect to the time constant of a neuron with L-type calcium current.

Methods: In the model, Goldman-Hodgkin-Katz equation is used for the L-type calcium current. Calcium concentration is dependent both on this current and on the calcium concentration difference between inside and outside of the cell. Time constant determines the speed of the calcium concentration change. For each specific time constant value, bifurcation diagram is drawn so that the dynamics of the calcium concentration with respect to the applied current can be seen. The value of the time constant is changed to investigate the change in the behavior of the system.

Results: In the beginning the value of the time constant is chosen 400.0 ms so that the system goes only to a stable point. Hopf bifurcation occurs at 1.3517 mA/cm^2 when the value of the time constant is 386.9 ms. If the time consant is decreased further two Hopf bifurcation points are created at two distinct current values. Between these current values there are both unstable fixed points and stable periodic points. Unstable periodic solutions emerge when the time constant is 308.8 ms and 163.2 ms, the applied current is 1.2456 mA/cm^2 and 1.2312 mA/cm^2 for the first and second Hopf bifurcation points, respectively. If the time constant is less than 91.1 ms stable periodic solutions disappear. Only stable and unstable fixed points exist if the time constant is less than 40.0 ms.

Conclusion: The dynamics of the calcium concentration in an L-type calcium current model with respect to the applied current is investigated by varying the time constant between 20–400 ms. Stable and unstable fixed points and stable and unstable periodic solutions are possible depending on the parameter values.

Keywords: bifurcation, calcium current, time constant

P-101

Effect of caffeine on the neuronal survival in rats exposed to electromagnetic field

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Objective: Advances in technology increased the use of devices that produce electromagnetic field, particularly mobile phones, because of which people are exposed to higher levels of EMF than present naturally. Several studies showed that electromagnetic field emitted by cell phones causes a decrease in the neuron number in some areas of brain. In this study, the protective effect of caffeine on the neuronal loss in the animals exposed to electromagnetic field was investigated.

Methods: This study included 30 rats in 5 groups each with 6 animals. The first group animals were exposed to 900 MHz EMF for 60 minutes per day, for 28 days and fed with water containing 1 mg/L caffeine. The second group received EMF but fed with tap water. The third group was not exposed to EMF and fed with caffeine containing water. The fourth group served as sham and placed in the EMF device without exposing to EMF. The fifth group was control and was not subjected to any procedure. At the end of 28 days, after undergoing cardiac perfusion, the brain and cerebellar tissues of the rats were removed for histological processes. The effects of caffeine on the neuron numbers in hippocampus and brain were investigated with stereological methods.

Results: There was a significant reduction in the number of Purkinje neurons of the cerebellum and pyramidal neurons of the hippocampus in rats exposed to EMF. In rats fed with caffeine in drinking water, exposure to EMF did not cause a significant decrease in neuron number in both hippocampus and cerebellum.

Conclusion: The results show that caffeine can reduce neuronal loss in the cerebellum and hippocampus in rats exposed to electromagnetic field. This study was supported by TÜBİTAK with a project number 214S642.

Keywords: electromagnetic field, Purkinje cell, pyramidal neuron, stereology

P-102

Cavum velum interpositum (subarachnoid cyst of velum interpositum): a case report

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Objective: Velum interpositum is a membrane which includes a small cavity and located superior and anterior to the pineal

gland. In case that membrane expands cavum velum interpositum (CVI) emerges. Velum interpositum emerges as the result of invagination of pia mater in the form of triangular membrane which apex locates anteriorly. That variation varies considerably.

Methods: A male patient, born in 2004, was referred to our clinic for MRG with diagnosis of intracranial mass from department of neurology in February of 2015.

Results: Intracranial mass was not detected in the contrast enhanced cranial MR examination. However, cavum velum interpositum (CVI) variation was detected. The close relationship with columna fornicis supero-anteriorly and commissura hippocampalis supero-posteriorly was also observed. It was also adjacent to the tela choroidea of ventriculus tertius and vena cerebri interna anteriorly and with thalamus postero-laterally. The narrow base of the triangle was contiguous with splenium corporis callosi. The distance between columna fornicis and splenium corporis callosi anteroposterior was measured as 22.2 mm at the sagittal plane which was close to the section. The lateral and supero-inferior distances were measured as 18.5 mm and 15.6 mm respectively, on coronal section.

Conclusion: CVI usually closes at the sixth month after birth. However, this structure can be found incidentally in 3% of children younger than 2 years. Therefore, it is a rare variation found in imaging studies. Although current case was 11 years old, no cystic dilatation and subsequent ventricular blockage caused by CVI was observed. There are reports that CVI cases are associated with delayed mental retardation, epilepsy and motor development. To classify the types of this variation in similar studies and give the incidence in our population may guide the treatment of symptomatic cases.

Keywords: cavum velum interpositum, CVI, variations, anatomy

P-103

The effects of the duration of hypertension on the cognitive functions in the hypertensive patients: an association with trace element zinc

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Objective: Cognitive impairment is considered as a form of dementia. Hypertension (HT) is one of the risk factors associated with dementia. The role of hypertension in determining the loss of cognitive function is not entirely defined. Various studies in animal models have shown the effects of zinc deficiency on decreased cognitive development activities. Our aim was to research effects of the duration of hypertension on the cognitive function and its relationship to relationship to zinc levels in serum.

Methods: This study examined 60 patients who were over 60 years of age, had at least five years of education, and had an essential HT diagnosis. We also had 30 healthy control subjects. The Mini Mental State Examination (MMSE) was applied to the patients. Scores of 24 and lower from the MMSE indicated a cognitive disorder. Serum levels of zinc were determined by an atomic absorption spectrophotometer.

Results: The MMSE scores of the patients with HT >10 years (n=19) were significantly lower than those of the patients with HT<5 years (n=17) ($p<0.05$). Zinc levels in the HT patients >10 years (152.95±40.53 µg/dL) compared to the HT patients <5 years (179.04±47.94 µg/dL) and were significantly lower ($p<0.05$).

Conclusion: It was found that long-term hypertension is associated with decrease in cognitive functions and their zinc levels decreases in relation to the duration of hypertension. Underlying mechanisms of these findings should be researched in further studies.

Keywords: hypertension, mini mental state examination, zinc

P-104

The effect of lithium therapy on thyroid dysfunction, hemorheological parameters and trace element levels in patients with bipolar disorder

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Objective: Although lithium is the most commonly used drug in the treatment of bipolar disorder, it causes functional impairment in various tissues depending on dose and duration. These functional disorders as shown in clinical and experimental studies are related with thyroid disorders ranging from mild to severe disturbances in TSH response. Studies of thyroid dysfunction, trace element balance and concomitant changes in the rheological properties of blood with lithium therapy have not been fully explained, and some results seem to be contradictory. In our study, we aimed to investigate the relationship between hemorheological parameters, serum iron, copper and zinc levels with thyroid dysfunctions occurring at different stages of lithium treatment.

Methods: In a group consisting of 22 individuals with no lithium treatment with bipolar disorder and no thyroid dysfunction (Group 1), 7 patients with hyperthyroidism (Group 2) and 15 patients with normal thyroid function (Group 3), Group of 7

patients with hypothyroidism (Group 4) and group of 4 patients with hyperthyroidism (Group 5) and 13 patients with normal thyroid function (Group 6), and serum lithium,iron,copper and zinc levels, thyroid panel and plasma-whole blood viscosity were measured by using atomic absorption spectrophotometer, electrochemiluminescence method and Harkness capillary viscometer method, respectively.

Results: Serum iron, hematocrit and erythrocyte counts were not significantly different between all groups. In Group 4 and 6, plasma and whole blood viscosity were higher than group 1, Group 2 and Group 3. Hemoglobin levels in Group 4 were decreased compared to those in Group1, Group 2, Group 5 and Group 6. Fibrinogen levels were higher in Group 4 and Group 5 than fibrinogen levels in Group 1. Zinc levels in Group 1 were lower than in Group 2, Group 3 and Group 6. Similarly, copper levels in group1 were lower than in Group 2, Group 4 and Group 6.

Conclusion: According to our findings, it can be suggested that changes of hemorheological parameters and trace element levels in lithium treatment of bipolar disorders were related to alterations in thyroid functions. This mechanism needs to be elucidated in more detailed studies.

Keywords: bipolar disorder, thyroid dysfunction, hemorheology, trace element

P-105

Celebrating Brain Awareness Week 2017 in Turkey – Neuroscience Society of Turkey (NST)

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Objective: The Brain Awareness Week (BAW) is a global campaign to let people know about the progress in brain research as well as diagnosis, treatment and prevention of disorders of the brain, such as Alzheimer's, Parkinson's, stroke, schizophrenia and depression. Every March, on the third week, BAW partner organizations world wide makes a celebration of the brain for people of all ages, from nursery school to the elderly.

Methods: Neuroscience Society of Turkey (NST) and Society for Neuroscience (SfN) Turkey Chapter performed a large number of events throughout Turkey in nine cities with a high number of collaborators. The BAW team was as follows: Selim Kutlu in Konya, Esat Adıgüzel in Denizli, Necip Kutlu and Ertuğrul Tatlısumak in Manisa, Ferhan Esen in Eskişehir, Ahmet Ayar in Trabzon, Piraye Kervancıoğlu and Mustafa Orhan in Gaziantep, Işıl Aksan Kurnaz, Esin Öztürk Işık, Kemal Türker, Yasemin Gürsoy Özdemir and Meral Yüksel in Istanbul, Hilmi Uysal in Antalya, Leyla Şahin and Nailcan Öztürk in Mersin, and Gülgün Şengül, Burcu Balkan, Aysegül Keser and Lokman Öztürk in Izmir.

Results: We performed a high variety of activities including public conferences, school conferences, nursery school activities, visits to house for the elderly, activities in shopping malls, neuroanatomy laboratory tours to high school students, movie evenings, television and radio programs. We estimate we have reached a total of one thousand five hundred people in person, and many more with radio and television programs, and newspaper articles.

Conslusion: As of thisyear as the BAW celebrates its 22nd anniversary and its 20nd anniversary in Turkey. NST and SfN Turkey Chapter aims to cover more activities in many more cities every coming year and also increase public outreach by media to have an impact on understanding the brain and brain research in our community. Grant support: BAW Turkey activities were supported by FENS (Federation of European Neuroscience Societies) and Society for Neuroscience (SfN).

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