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AIM AND SCOPES

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

Areas of particular interest are four topics. They are;

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

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

C- Interaction Between Oxidative Stress and Ion Channels in Neuroscience
(Effects of the oxidative stress on the activation of the voltage sensitive cation channels, effect of ADP-Ribose and NAD^+ on activation of the cation channels which are sensitive to voltage, effect of the oxidative stress on activation of the TRP channels, role of TRPM2 channels in neurodegenerative diseases such Parkinson's and Alzheimer's diseases)

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

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

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CONFERENCE

Conference No. 1

Brain, aging, and melatonin: Evidence of neuroprotection against experimental ischemic injury

Sergio D. Paredes, Lisa Rancan[1], Cruz García[1], Elena Vara[1], Jesús A. F. Tresguerres

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Stroke is a cerebrovascular accident or brain attack that represents a major cause of death and long-term disability throughout the world. Overproduction of free radicals during cerebral stroke, also referred to as ischemia and reperfusion (I/R) is known to contribute to neuronal functional disruption and death. In view of its actions, attention has been paid to melatonin as a neuroprotective drug against I/R brain injury. Melatonin functions in multiple ways to mitigate neural damage during the acute phase of neural hypoxia and reoxygenation. One of the major processes by which melatonin mediates protection of neurons and glial cells presumably relates to its ability to function as a direct free radical scavenger and indirect antioxidant. Other actions of melatonin that may aid in decreasingneural damage in I/R include its ability to limit apoptosis and reduce the inflammatory response. On the other hand, it is known that there is a gradual reduction in circulating melatonin concentrations with increasing age. This fact may be the reason why more marked ischemia-induced deleterious effects are seen in aged animals. Little is known, however, on the possible protective effects of melatonin in aged individuals affected by brain ischemia. Recent experimental studies suggest that treatment with melatonin may be able to decrease significantly the hippocampal and cortex expression of pro-inflammatory IL-1β and TNF-α, and pro-apoptotic BAX and BAD markers as well as reducing the mRNA levels of GFAP and elevating sirtuin 1 in aging rats subjected to ischemic lesion induced by blockade of the middle cerebral artery. Taken together, these results point to consider melatonin for protection of the brain from I/R injury.

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Conference No. 2

Physiology of cation channels and calcium ion in neurons: Focus on TRP Channels

Mustafa Nazıroğlu

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Sodium ion (Na^+) and calcium ion (Ca^{2+}) are high in outside of the neurons although potassium ion (K^+) is high in inside of the neurons. Cations have important role in physiology of neurons. For example, the Ca^{2+} is an important second messenger and plays a role in numerous signal transduction pathways including neuronal excitability, neurotransmitter release, cell proliferation, and cell death. It has long been known that Ca^{2+} is involved in the etiology of neuronal diseases such as epilepsy and peripheral pain. Different types of cation channels channels, such as voltage gated calcium channels (VGCC) and chemically gated calcium channels, likely play an important role in neurons. Apart from the VGCC and chemically gated channels, one family of Ca^{2+} channels comprises transient receptor potential (TRP) cation channels. The TRP channels were firstly expressed in photoreceptors carrying *trp* gene induced a transient voltage changes to continuous light mutations of *Drosophila* flyers. One subfamily of TRP channels is the vanilloid group containing 8 members, including TRPV1 channels. TRPV1 channels are non-selective

cation channels and they were first reported in sensory neurons such as dorsal root ganglion (DRG) and trigeminal ganglia neurons because the channels respond to various stimuli including oxidative stress, noxious heat (> 43 °C), protons and capsaicin. Another subfamily of TRP channels is the melastatin group containing 8 members, including TRPM2. TRPM2 channels were mostly expressed in phagocytic cells and brain. TRPM2 channels are activated by oxidative stress and ADP-ribose. Recently results of our group indicate that the TRPM2 and TRPV1 channels have important role on etiology of epilepsy and peripheral pain in hippocampus and DRG (Özdemir et al. 2015; Nazıroğlu and Övey, 2015).

In conclusion, ıt seems that Ca^{2+} entry through TRPM2 and TRPV1 channels has important role in physiological function of neurons and etiology of neuronal diseases such as epilepsy and pain.

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Keywords

Calcium ion; Neuron; Oxidative stress; TRPM2; TRPV1.

Conference No. 3

Translational approaches in neuroscience of sleep and wakefulness.

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Sleep is fundamental to our well-being and sleep disturbances produce wide-ranging morbidities in neurological, cardiopulmonary and metabolic systems. Combining animal models and clinical studies would be an the effort to pinpoint the molecular gears of neuropathophysiology of sleep disorders as well as better defining fundamental aspects of the biological clocks that govern daily rhythms of sleep and wakefulness. Interdisciplinary teams need to capitalize on these groundbreaking insights to discover how sleep contributes to healthy brain function, and when they go awry, possibly hasten the progression toward disorders like autism, depression, neurodegenerative diseases like Alzheimer's and Parkinson's disease.In today's competitive life,sleep deprivation is deemed as an alarming phenomenon affecting our neurological and mental health. In animal models, the experimentally-induced total sleep deprivation (TSD) and chronic partial sleeprestriction (CPSR) are knownto contribute to the emergence of cognitive impairments. This is hypothesized toresult from a consequent neuroinflammation which may also hasten the neurodegenerativeprocesses. Neuroinflammatory markers such as tumor necrosis factor-alpha (TNFα) arethought to be potential culprits in SD-induced neurodegeneration. Rats subjected to TSD and CPSR tend to show a pronounced impairment of memory, elevated serum corticosterone anddecreased brain-derived neurotrophic factor (BDNF) levels. On the other hand, CPSR rats which undergo delayed brain excision following behavioral testing, demonstrate deposition of the hyper phosphorylated tau (HPτ) and reveal the least numerical density in thehippocampal dentate gyrus (DG) neurons. Meanwhile, immunohistochemical study has revealed no amyloid beta (Aβ) deposition in the hippocampal DG in rats. Interestingly, treatment with a tumor necrosis factor alpha (TNF-a) neutralizing antibiody, is shown to abrogatesleep restriction-induced cognitive decline, biochemical changes and the immunohistopathology inthe hippocampal DG. The drastic effect of SD on neural dynamics in the hippocampus is shown to be via activation of the inflammatory mechanisms in part through TNFα-dependent pathways. Continued translational research in sleep neuroscience is expected to unlock mysteries about sleep, its function and basic and clinical pathophysiological aspects of sleep disorders.

Keywords: Translational Neuroscience, Sleep, Wakefulness, Circadian rhythms, sleep deprivation

Conference No. 4

Brain slice experiments in Patch-clamp

Ramazan Bal

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In this seminar, I will be summarizing how to do brain slices using vibratome, how to prepare patch micropipette and how to obtain giga seal on neurons in brain slices. Brain slices have been widely used for electrophysiological studies and the methods used to prepare brain slices are widely documented. We will explain the procedures that we use briefly, which seem to give good results with a variety of brain areas, different labs have their own way of doing it though.

After 16-18 day-old rats are decapitated under anesthesia and the brain is then dissected gently using fine slices and then removed and placed in physiological solution within 60 seconds of decapitation. Depending on the area of interest, the tissue is glued on to the stage of the tissue slicer, using cyano acrylate glue. The stage is placed in to the vibratome and immediately then physiological solution is poured over the tissue until it is submerged. The tissue is cut into slices of 100-300 µm thickness with vibrating slicer. Using a plastic Pasteur pipette, slices are transferred to the resting chamber which should be kept at room temperature with a good steady flow of O_2/CO_2 bubbling through the solution.

Under an upright microscope equipped with DIC and water-immersible lens, giga ohm seal is obtained. The major problem in obtaining giga seal on neuron is to have a clean Access for the patch electrode to the surface of neurons in brain slices. So different strategies to clean surfaces of neurons and tip of patch electrodes in brain slices experiments will be explained.

Using patch clamp technique, current and voltage clamp recordings were taken from cell bodies of neurons with one of four configurations, cell-attached, whole-cell, inside-out and outside-out.

Conference No. 5

Voltage gated sodium channels and epilepsy

Simon Hebeisen

B'SYS GmbH, The Ionchannel Company, Witterswil, Switzerland

Epilepsy is the fourth most common neurological disorder and affects people of all ages. Approx 3 out of 100 humans encounter at least a single episode of epilepsy during their life. About 50 million people worldwide suffer from recurrent seizures. Medication for epilepsy is often life-long and has a major impact on the quality of life - mostly being related to substantial adverse effects. Therefore, over 30% of people with epilepsy do not achieve sufficient seizure control whilst effective medication being available.

Ion channels are often the primary target of anticonvulsant drugs. They can either act as blockers for voltage gated sodium and calcium channels or as activators for potassium or chloride channels. Additionally, modulators of ligand gated ion channels like GABA or Glutamate receptors are frequently used to treat epilepsy.

Employing a panel of true functional patch-clamp assays on a broad range of voltage and ligand gated ion channels during the last 3 years we were able to successfully screen for drugs with a beneficial action profile. In successful leads we found drugs that selectively interacted with TTX sensitive, neuronal voltage gated sodium channels. Activation and fast inactivation were unchanged, while an increased affinity in the slow inactivated state was observed. This profile is in contrast to traditional anticonvulsant drugs like Carbamazepine or Oxcarbazepine which show their major effects on the fast inactivated state of voltage gated sodium channels.

Analysis of common denominators of these novel drugs made evident that this specific action on dedicated states of voltage gated sodium channels may be responsible for the improved tolerability. Novel anticonvulsant drugs that targeting partial seizures -representing the predominant seizure type- show improved efficacy and a beneficial tolerability profile over traditional

anticonvulsants.

Conference No. 6

Neuroanatomy and Dısorders of Goal-Dırected Behavıor

Serpil Demirci,

Süleyman Demirel University, Medical Faculty, Department of Neurology, Isparta, TURKEY

All actions can be classified simply as habitual and goal-directed. For an action to be signified as goal-directed, it requires the person to have a knowledge of causal efficacy of actions and their outcomes in the current status and context and to select and regulate the behavior using goal representations. Simply, goals directed and mediated by states of internal environment must be hold in working memory for online-evaluation, update and sustainment of the knowledge and then used for appropriate response production according to the specific states of the external world. Goal-directed behavior results from a well-structured and organized frontal network architecture in which goals are self-attributed on the basis of predefined needs, valence and multiple memory and planning, and self-control mechanisms. All these actions mainly implemented by two basic frontal networks. Medio-dorsal frontal pathways is strongly connected with basal ganglia, premotor frontal cortex, supplementary motor cortex and association areas and is responsible from motor control, performance monitoring, regulation of behavior and planning and conduct of action. The other basic pathways, orbito-ventral frontal pathway is responsible for integration of emotion, memory and information belonging to environmental stimulus. Orbito-frontal pathways also conduct recall, selection and processing of content of information that is processed in other systems according to goal. Orbito-ventral pathway's connections are with amygdala, hippocampus, temporal association areas and dorsolateral prefrontal cortex. Any compromise to the integrity of these structures may result in different clinical conditions.

Conference No. 7

Western-blot, Immuno-fluorescence and Genomic PCR

Denis Rousseau

Grenoble University France

Western-blot, Immuno-cytology and genomic PCR are rapid and informative technics, even like others submitted to artefacts and particularities.

Western-blot experiments allow to quantify cellular or extra-cellular protein level, as some of their status like their phosphorylation. It can be used on small biological samples, typically like humans biopsies.

Immuno-cytology can able to determine protein localization within the cell, like either DNA or ARN epitopes, and can be prolonged by flow cytometry measurements for semi-quantitative measurements.

Then, genomic PCR is a powerful tool to identify the presence or absence of genes, which is necessary in genotyping or for the search of DNA-mutation linked diseases.

All these technics are convenient to approach physiological or pathological cellular regulations.

Conference No. 8

Learning models in experimental animals and evaluation of learning

Arif Demirdaş

Department of Psychiatry, Suleyman Demirel University, Isparta, Turkey.

Learning is a quite long term change occurring in behaviors by an individual's experiences. Infact learning process is not only for human beings but also all organisms. That is why animal experiments and animal models are used to investigate learning process. There are behavioral, affective, cognitive and neurophysiological based learning rules which were put forward for learning. Behavioral

theoreticians admit that learning improves establishing a connection between stimulus and behavior and changing behaviors via reinforcement is essential. According to cognitive hypothesis, learning is attributing a meaning to whatever takes place around an individual. Affective concepts concern problems about learning rather than nature of it. Neurophysiological-based education principles emphasis that the brain is a parallel processor and learning should be evaluated as a psychological event. Experimental animal studies help us understand the physiological and psychological conditions of several diseases. Studies concerning learning and memory play a great role in explaining cognitive processes of neuropsychiatric diseases. When we have a look at the literature, experimental animal models about learning were described. These are morris water maze, T maze or radialarm maze, novel object recognition, fear conditioning, active avoid anceand passive avoidance. In this course 'Learning models in experimental animals and evaluation of learning'' will be presented.

Keywords: animal models, learning.

Poster Presentations

Poster No. 1

Raloxifene and tamoxifen reduces oxidative stress in brain of ovariectomized rats

Yener Yazğan[1], Betül Yazğan[2], İ. Suat Övey[1], Mustafa Nazıroğlu[1,3],

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Abstract

It has been well known that estrogen has antioxidant role on cardiovascular, gastrointestinal and neurological systems. Estrogen deficiency may active the channels although supplementation of estrogen may inhibit the channels via its antioxidant properties and support antioxidant levels in the neurons. Raloxifene and tamoxifen are selective estrogen receptor modulators (SERMs). They exert tissue-selective agonist–antagonist effects on estrogen receptors. The antioxidant properties of raloxifene and tamoxifen may also have a regulator role in the brain of ovariectomized rats. We aimed to investigate the effects of estradiol, raloxifene and tamoxifen on the oxidative stress in brain of ovariectomized (OVX) rats.

Forty female Wister Albino rats were divided into five groups: First group was used as control. Second group used as ovariectomized. Third, fourth and five groups received estrogen, tamoxifen and raloxifene, respectively. Estrogen, tamoxifen and raloxifene were subcutaneous given to these three groups for 14 days, after OVX.

We determined an increase in lipid peroxidation (as MDA) levels in brain of ovariectomized rats. While raloxifene, tamoxifen and estrogen treatment reduce levels of MDA versus to ovariectomized group. In addition, effect of tamoxifen is clearer than estradiol and raloxifene. Although reduced glutathione (GSH) and glutathione peroxidase (GSH-Px) levels were reduced by OVX they were increased by the three treatments.

In conclusion, we observed those estrogen, tamoxifen and raloxifene administrations are beneficial on oxidative stress level in the brain of OVX rats by modulating antioxidant system.

Keywords: Estrogen; Raloxifene; Tamoxifen; Ovariectomized; Oxidative Stress.

Poster No. 2

The investigation of the effect of some histone deacetylase inhibitors on angiogenesis in glioma

Taylan Turan, Aymelek Gönenç

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Angiogenesis process occurring during embryogenesis, wound healing and menstruation is a new vessel formation in a vasculature structure of pre-existing. This process takes on a significant role for tumor growth and progression in cancerous cells. Angiogenesis a marker of malignant cancers causes the most neovascularization in gliomas inside solid tumors. Recently, many new anti-angiogenic agents have been used for the treatment of various cancers including gliomas. But, there are limited number of studies examining the effects of histone deacetylase inhibitors (HDACI) on angiogenesis. Osuka et al. evaluated in vitro and in vivo anti-angiogenic potential of valproic acid (VPA), one of the HDACI, in malignant glioma (1). They suggested that VPA has an effect to decrease vascular endothelial growth factor (VEGF) secretion of glioma cells under both normoxic and hypoxic conditions, in a dose-dependent manner in vitro. Besides, they found that VPA inhibits endothelial cell proliferation in vivo. Similarly, Sawa et al. examined that the effects of HDACI on the expression of VEGF in diffrent cell line (2). They determined that HDACI diminish VEGF secretion and concluded that HDACI may inhibit angiogenesis. In one another study, it was found that the type II tumor suppressor Inhibitor of growth

1 (ING1), which is involved in DNA damage response and histone modification, is decreased in glioblastoma and related with glioma-induced angiogenesis (3). After that, they investigated the effect of HDACI on ING1 levels and the impression of modulating ING1 levels on HDACI-induced apoptosis in malignant glioma cells. Consequently, they demonstrated that ING1 contributes to HDACI-induced apoptosis in glioblastoma cells. There is no doubt that angiogenesis is a complex mechanism that are influenced by lots of parameters such as polymorphism, oxygen pressure etc. When these parameters taking part in this complex mechanism take into consideration, new studies may shed light on future.

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Poster No. 3

Serum vitronectin levels and antioxidant status in pancreatic and colorectal cancer

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Colorectal and pancreatic cancers rank third and fourth respectively, among the most common cause of cancer related deaths the world (1). Nowadays, researchers focused on effects of small adhesion molecules on the tumor malignancy. Vitronectin (VN) is a major plasma glycoprotein that exhibits many activities and functions as a cell adhesion molecule. Pirazzoli et al. demonstrated that vitronectin plays an important role in tumor growth (2). It is known that oxidative stress is one of the major predictive factors in carcinogenesis and serum total antioxidant capacity (TAC) indirectly reflects the level of oxidative stress. Decreasing serum TAC has been reported to have a strong association with various cancers (3). However, there are inadequate published data to verify the importance of serum TAC and VN concentrations in colorectal and pancreatic cancer in Turkish population. Therefore, the investigation of changes in VN and TAC expression and compare patients with healthy individuals was aimed for protective purposes in pancreatic and colorectal cancers. Serum VN were assessed in fifty three cancer patients (twenty three of them were pancreatic and the rest of the patients were colorectal cancer) and twenty nine healthy subjects as a controls by ELISA technique while TAC were assessed by a colorimetric method. In total patient group, both TAC and VN levels diminished as compared with healthy controls. But, a significant difference was found only TAC ($p<0.01$). On the other hand, no significant differences were found between serum levels of VN in two groups ($p>0.05$). TAC levels were decreased significantly in both colorectal and pancreatic cancers as compared to controls ($p<0.01$). VN levels in pancreatic and colorectal cancer groups were not different from controls ($p>0.05$). In conclusion, we think that these results can support the idea of decreased antioxidant defense mechanism is associated with carcinogenesis in these cancers.

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Poster No. 4

Epigenetics and the Brain

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Epigenetics is being recognized to take part in many biological processes as a general mechanism of altering and regulation of cellular and organismal responses and functioning that eventually creates intercellular differentiation. Realization of epigenetic events in the central nervous system, in association with the others, leads to the generation of several social, cognitive, healthy versus pathological, developmental, and adaptive states of the brain and the individual (1-3).The common approaches in studying epigenetics and the general means of epigenetic alterations are the DNA methylation, histone modification events, and non-coding RNAs. Thus the epigenetics in the brain and brain studies will be broadly overviewed here.

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Poster No. 5

Protective effect of tamoxifen and raloxifene on oxidative stress in liver, heart and kidney of ovariectomized rats

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Abstract

In the postmenopausal period, the decrease in ovarian hormones can influence inflammation and oxidative stress in many tissues. Estrogen has antioxidant role in all the physiological systems and its deficiency may active the channels via antioxidant properties of estrogen and support antioxidant levels in various tissues. Tamoxifen and raloksifen are selective estrogen receptor modulators (SERMs). They exert tissue-selective agonist–antagonist effects on estrogen receptors. The antioxidant properties of tamoxifen and raloxifene may also have a regulator role on oxidative stress in liver, heart and kidney tissues of ovariectomized rats. We aimed to investigate the effects of estradiol, raloxifene and tamoxifen on the oxidative stress in liver, heart and kidney of ovariectomized rats.

Two weeks after ovariectomy, female Wistar Albino rats (n = 40) were equally divided into five groups as control group, ovariectomized (OVX), OVX+Estrogen, OVX+Tamoxifen and OVX+Raloxifene groups. After OVX, The estrogen, tamoxifen and tamoxifen groups received subcutaneous 17-β estradiol (80µg/kg/day), tamoxifen (1mg/kg/day) and raloxifene (1mg/kg/day) subcutane for 14 days, respectively.

We determined an increase in lipid peroxidation (as MDA) levels in liver, heart and kidney of ovariectomized rats although its level was decreased by by raloxifene, tamoxifen and estrogen treatments. Reduced glutathione (GSH) level and glutathione peroxidase (GSH-Px) activity were decreased by OVX although they were increased by raloxifene, tamoxifen and estrogen in liver, heart and kidney. The effect tamoxifen was more significant than in estradiol and raloxifene in the three tissues.

In conclusion the current results indicated that estrogen, tamoxifen and raloxifene treatment have beneficial effects on OVX-induced oxidative stress in the liver, heart and kidney of rats by modulating antioxidant glutathionee system. However, effects of tamoxifen on the values seem more important than in estrogen and raloxifene.

Keywords: Estrogen; Raloxifene; Tamoxifen; Ovariectomized; oxidative stress; Liver; Heart; Kidney.

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