ISSN: 1308-416X

Cell Membranes and Free Radical Research

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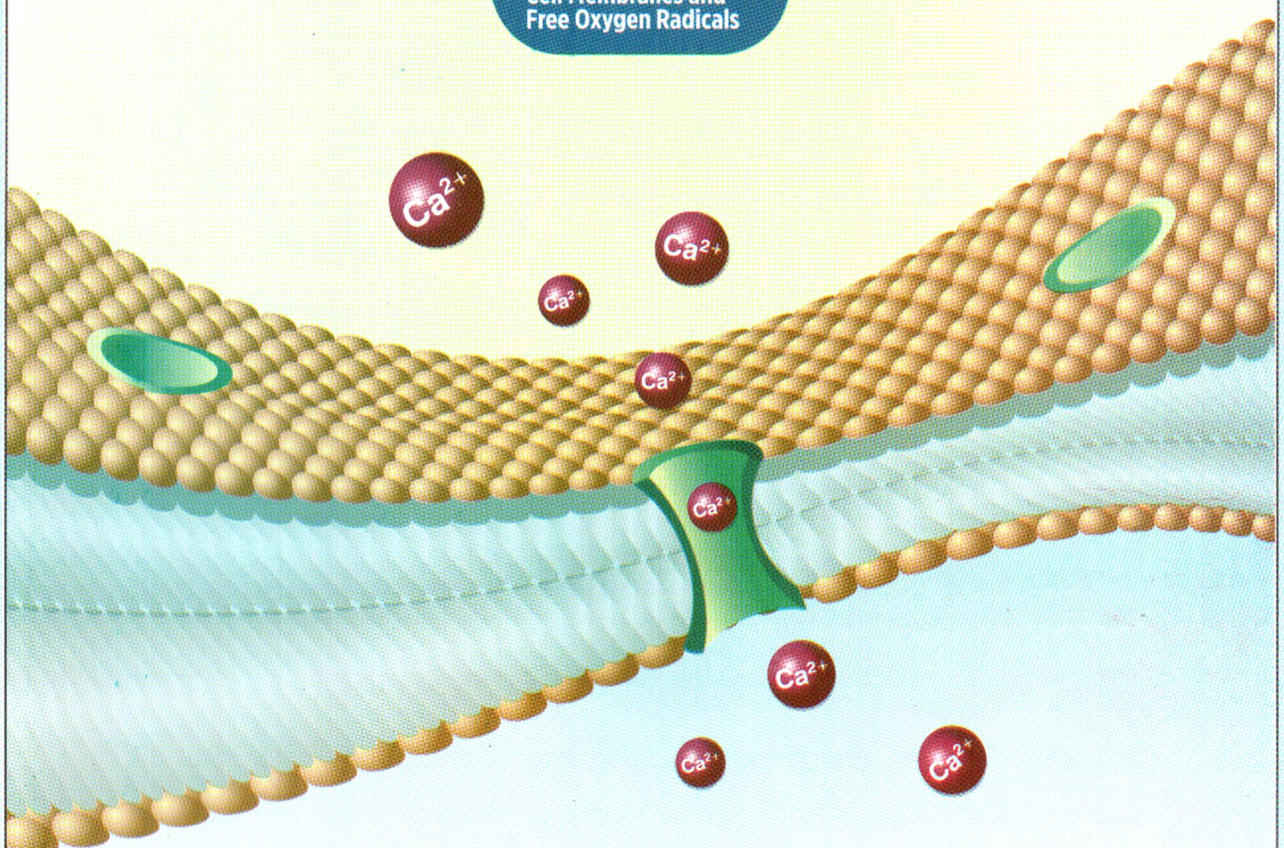

SÜLEYMAN DEMİREL ÜNİVERSİTESİ
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Volume 5, Number 2, 2013

Society of
Cell Membranes and
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Advanced Cellular Neuroscience
Research Group

Neuroscience Research
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5th Cellular Neuroscience Days

Neuroscience Research Center, Suleyman Demirel University

1-2 June 2013 Isparta / Turkey
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Cell Membranes and Free Radical Research

Volume5, Number2, 2013
ISSN Numbers: 1308-4178 (On-line), 1308-416X
Indexing: Google Scholar, Index Copernicus, Chemical Abstracts, Scopus (Elsevier), EBSCOhost Research Database

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AIM AND SCOPES

Cell Membranes and Free Radical Research is a print and 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

(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)

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.

5th Cellular Neuroscience Days

Neuroscience Research Center, Süleyman Demirel University

[Contents]

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The Effect of Antiepileptic Zonisamide on Ca^{+2} Signaling, Apoptosis and Oxidative Stress in PC12 Cells

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Introduction

Parkinson's is a incurable progressive neurological condition caused by a degeneration of dopamine-producing cells charecterized by motor and non-motor symptoms. In fact, zonisamide is used as an antiepileptic molecule in the world and benzisoxole (1,2-benzisoxazole-3-methaesulfonamide) is a moleculer structure of zonisamide which has a long half-life (63 hours) and a well tolerated drug. ZNS revealed evidence of an effect on improves motor function in with Parkinson's patient that on oxidative stress and neuroprotective in clinical and experimental research.

Groups

PC12 cells were divided into 4 groups :
Group 1 (control group),
Group 2 (ZNS group),
Group 3 (MPP^+),
Group 4 (ZNS + MPP^+ group).

The dose and duration of ZNS and MPP^+, were determined according to MTT analysis which used to assessed the cell viability. The cells were incubated to for 5 hours with 100 μM ZNS, 10 hours with 100 μM MPP^+ and 10 hours with ZNS and MPP^+.

Results

Lipid peroxidation levels were significantly higher in the MPP^+ group, but they were significantly lower in ZNS and ZNS+MPP^+ groups($p < 0.05$). GSH and GSH-Px levels were significantly lower in the MPP^+ group ($p < 0.05$) but in ZNS and ZNS+MPP^+ groups, GSH and GSH-Px levels were significantly higher($p < 0.01$). Cytosolic Ca^{2+} release was found to be significantly increased in the MPP^+ ($p < 0.001$) and ZNS+MPP^+ ($p < 0.01$) groups than the ZNS ($p < 0.01$) group. And also according to the control

group cytosolic Ca^{2+} release was found to be significantly decreased in ZNS ($p < 0.01$) group. Caspase-3 activity was found to be significantly lower in the ZNS ($p < 0.001$) group than the MPP^+ ($p < 0.001$) group.

In conclusion, zonisamide has a neuroprotective effect against MPP^+ neurotoxicity in experimental model of PD in PC12 cells.

Key words

Zonisamide; MPP^+; Neuronal PC12 cells; Oxidative stress; Ca^{2+} signaling; Apoptosis

Role of Melatonin on the Inactivation of TRPM2 Cation Channels Activated by Oxidative Stress

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Introduction

Transient Receptor Potential Melastatin-Like 2 (TRPM2) is a non-selective Ca^{2+} permeable cation channel and is known to be activated by H_2O_2 which is one of the most important indicators of intracellular oxidative stress. Recent reports exhibited that TRPM2 currents may be blocked by aminoethoxydiphenyl borate (2-APB) in a manner rapid and reversible. Melatonin is a neurohormone and has a very high antioxidant effect, which released from pineal gland. In this study we investigated effects of melatonin on whole cell currents and Ca^{2+} influx arising from TRPM2 channels activated by H_2O_2 in transfected Chinese Hamster Ovary Cells (CHO).

In whole-cell patch clamp experiments, TRPM2 currents in the cells were consistently induced by H_2O_2. However, the current were inhibited by both extracellular (0.3 mM and 1 mM for 2 hours incubation) and intracellular (0.2 mM) melatonin. Cytosolic Ca^{2+} release was measured by Fura- 2 and the cytosolic free Ca^{2+} content of the cells were higher in H_2O_2 groups than in control. When intracellular melatonin is introduced by pipette TRPM2 channel currents were not activated by H_2O_2 although H_2O_2-induced Ca^{2+} gates and release were not blocked by the 2-APB. Melatonin inhibited also apoptosis dose dependent.

In conclusion, we observed the modulator role of intracellular melatonin on Ca^{2+} influx and apoptosis through a TRPM2 channel in transfected CHO cells. Since cytosolic melatonin depletion due to Ca^{2+} influx is a common feature of oxidative stress-induced diseases, our findings are relevant to the etiology of pathology in oxidative stress-induced diseases.

Keywords

Melatonin; Oxidative Stress; Ca^{2+} Signaling; TRPM2 Channel Antagonist; Apoptosis

Effects of Electromagnetic Radiation (900 MHz) on TRPV1 Channels, Apoptosis, Caspase Activities and Oxidative Stress in Hippocampus of PTZ-Induced Epileptic Rats

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Abstract

Many of toxic effects elicited by exposure to environmental stressors including electromagnetic radiation (EMR) are reported to be mediated the regulation of apoptosis and oxidative stress through Ca^{2+} influx. Changes in the apoptotic process induce in most of the neurodegenerative diseases including epilepsy. However, there is no report of cell phone exposure on Ca^{2+} influx, apoptosis and oxidative stress in epileptic rats through TRPV1 cation channels. Therefore, we tested the effects of cell phone frequency (900 MHz) exposure on Ca^{2+} influx, apoptosis, oxidative stress and TRPV1 channel activations in the hippocampus of pentylentetrazol (PTZ)-induced epileptic rats.

Twenty-one rats were used in study within three groups namely control, PTZ and PTZ+EMR. Epilepsy was induced intraperitoneal administrations of PTZ. After 1 hour, the hippocampal neurons were freshly isolated from the rats. The neurons in 900 MHz and PTZ+EMR groups were exposed to the 900 MHz EMR for one hour. The apoptosis levels, caspase-3 and -9 activities, mitochondrial depolarization rate and cytosolic reactive oxygen species (ROS) levels were higher in PTZ and PTZ+EMR groups than in control. Cytosolic free Ca^{2+} $[Ca^{2+}]$ i concentration was also higher in epilepsy+capsaicine group than in control although its concentration was decreased by TRPV1 channel blocker, capsazepine. There was no statistical significance on the values between PTZ and PTZ+EMR groups.

In conclusion, in our experimental model, epilepsy but not cell phone-induced EMR (900 MHz) exposure induced Ca^{2+} influx (via TRPV1 channel activation), apoptosis and oxidative stress in the hippocampus neurons. We were not able to see risk on use of cell phone in the epileptic hippocampus.

Keywords

Hippocampus; Electromagnetic Radiation; Caspase Activity; Apoptosis; TRPV1 Channels; Epilepsy

Acknowledgement

The study was partially supported by The Scientific and Technological Research Council of Turkey (TÜBİTAK)

Apoptosis in Nervous Tissue and its Markers

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Abstract

Apoptosis, Programmed cell death (PCD), essential events in maintaining of organisms' live as well as process of embryological development. In the neurogenenesis, about half of the neurons were eliminated through a process of apoptotic selection. Then, at the postnatal period this process will be continuing during physiological aging. But its rate cannot reach at renewal tissues, in which about at mitotic rate. Apoptotic events were increased seriously at neurodegenerative diseases (e.g. Alzheimer's, MS, ALS), certain infections (such as HIV encephalitis), cellular stress and excitotoxicity, traumatic events in contrast decreased pathologically in neuroglial cancer events.

Although, observations of this kind of cellular death is almost a century, identification and nomenclature based on the 1970s. Via the specific morphological and biochemical characteristics, energy-dependent an active process features, it is distinguishable events from necrosis, passive and an uncontrolled death. All of these processes, activation and inactivation of the apoptotic protein, especially caspases, in question. However, all of these proteins, molecular mechanisms of how the function is and how can activated or inactivated are not fully understood. The identification and interpretation of apoptosis is based on the different morphological and biochemical characteristics.

Roughly, apoptotic processes begins extracellular (extrinsic) and cellular (intrinsic) mechanisms. At this process, biochemical properties such as protein degradation or cross-links, DNA fragmentation and the determination of the phagocytic process, as well as cytomorphological changes such as nuclear, cytoplasmic and cell membrane changes were made constitute the main logic tests. However, a very rapid progression (within minutes), and low rates of apoptosis especially in healthy tissues, as well as the be nonspecific and false positive results of these tests were the main handicap. In apoptotic process, expressing of the some proteins for a temporary period is the second limitation.

Cytomorphological changes were interpreted in routine histological sections. But not show in the early phases and the rapid development and the rapid phagocytosis of waste, as well as the realization of a limited number of cells makes it difficult to detect. Dynamic imaging and ultra structural investigations, to be preferred to static imaging studies with conventional light microscope.

DNA laddering technique used to detect DNA fragmentation. Because of the requirement of a large number of cells, TUNEL test was more popular, showing in situ fractures of the DNA chain (terminal dUTP nick end labbelling). TUNEL give results in a short time, and with fewer cell. However, for the positive results, amount of DNA fracture is not clear. As well as the high cost of false (+) results in necrosis, require verification is disadvantages of this test.

Determining caspases and proteolytic activity is widely used. Appropriate to immunohistochemistry, western blotting, immun-precipitation many types and kinds of mono and polyclonal antibodies (fluorescent conjugated) are available. As apoptosis initiators caspase: 2, 8, 9 and 10, as executor: 3, 6, 7 and as inflammatory: 1, 4 and 5, and specific to embryonic tissues caspase 14 were reported. In addition, for extrinsic pathway: caspase 8, for the intrinsic pathway: caspase 9, and for common pathway: Caspase-3 is more specific.

Annexin V test based on the principle in detecting changes phosphatidyl serin, an inner cell membrane molecule, to the outer membrane (externalization) is commonly used particularly in caspase confirmation. Also propidium iodide and trypan blue tests are used to show disintegration of necrotic cell membrane.

Acridine orange, neutral red and Nile blue dyes are used in the evaluation of whole embryo and other tissues. Because these dyes were acidophilic, lysosomic and phagocytic activity can demonstrate quick and inexpensively. But they are nonspecific and highly toxic and should be confirmed by other tests.

Determining the detection of changes in mitochondrial cytochrome C is important to show the early phase. For demonstrating the mitochondrial membrane potential, internal membrane depolarization, redox potential and reactive oxygen species, as well as the calcium entry confocal microscopic imaging is preferred. Healthy cells accumulate fluorescent lipophilic dyes; apoptosis mitochondrial outer membrane (MOM) disintegrated and dye dispersed in the cytoplasm. MOM dyes used to measure the redox potential and metabolic activity. However, tests should be confirmed with caspase.

Also antiapoptotic BCL 2, Bax and Bid can be measured with the fluorescent and confocal microscopy.

The identification and interpretation of apoptotic processes, in particular a better understanding physiopathology of neurodegenerative processes and thus to contribute to new therapeutic approaches and is likely to continue to be a popular and important area.

Keywords

Apoptosis; Cell; DNA; Caspases

Analytical Fluorescence Microscopy Techniques

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Abstract

Fluorescence microscopy techniques provides an indispensible and efficient approach for studying live and fixed cells with their high sensitivity and specificity. Fluorescence microscopes do not only visualize fluorescence emitted from labelled molecules, but also collect data about the intensity and spectrum of the emitted light. By exploiting the properties of fluorescence physics, it has become possible to visualize and analize various dynamic events in the cell, organels or even at smaller scales. In this presentation, basic characteristics and applications of multiphoton microscopy, fluorescence recovery after photobleaching (FRAP), fluorescence loss in photobleaching (FLIP), fluorescence localization after photobleaching (FLAP), fluorescence resonance energy transfer (FRET) and fluorescence lifetime imaging microscopy (FLIM) techniqes will be discussed.

Keywords

Fluorescence microscopy techniques; FRAP; FLIP; FRET

A Visceral Pain Model Induced By Coorectal Distension

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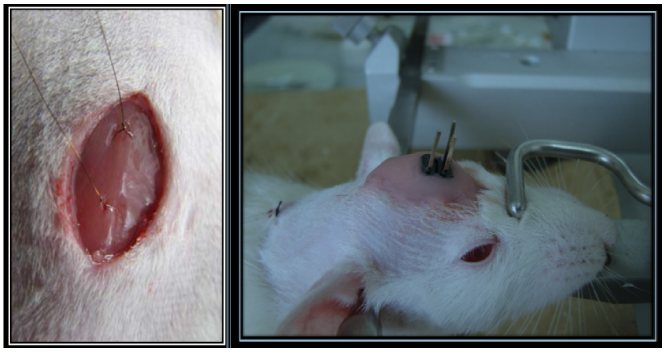
Abstract

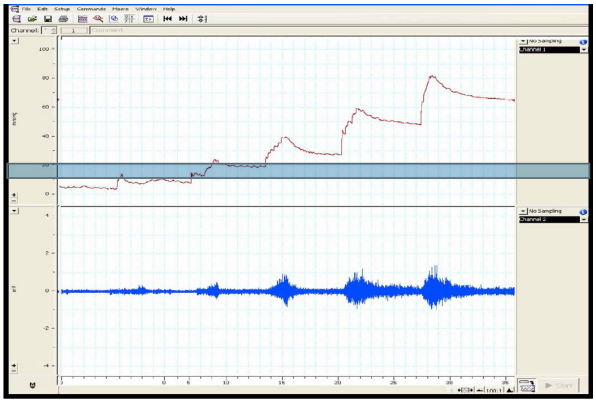
Colorectal distension (CRD) is a reliable and usuable behavioral pain model which was founded by Ness and Gebhart first in the awake rats. Distension of colon and rectum causes mechanically noxius situmulus. In this model, produced sitimulus is minimally invasive and reproducible. Physiological and behavioral parameters can be measured more easily and objective. This model mimics the pain syndromes occured in patients who are suffered from iritable colon syndrome. CRD causes an easily measuruble pressure response called as visceromotor (VMR) response. VMR involves external oblique muscles contraction and evaluated by measuring electromyographic (EMG) activity. To gain EMG activity the two nichrome electrodes are implanted into the external oblique muscles in anesthetisied rats. The EMG electrodes are tunneled subcutaneously and externalized at the nape of the neck for access during testing. After surgery, they are accustomed to manipulation and to light restraint in Bollman cages during a 6-day recovery period before commencing the experiments, to reduce motion artifacts and confounding effects due to stress-related responses.



Producing of Colorectal Distension

A flexible tygon plastic tubing is inserted into a latex balloon with the end of the balloon securely tied to the tube. The balloon is inserted intra-anally into the descending colon approximately 1–1.5 cm into the rectum. The tubing is taped to the base of the tail to prevent displacement. The rats are fully awake and placed inside Bollman cages during testing. CRD is produced by inflation of the balloon with air. The catheter is attached to a

bridge amplifier via a pressure transducer. The intracolonic pressure is monitored and recorded by a data acquisition system connected to the bridge amplifier. Briefly, starting at 0 mm Hg, intracolonic pressure is incremented in steps (10–20 mm Hg) over about 80 sec to a final pressure of 80 mm Hg. As the maximum VMR usually occurred in response to the 80 mmHg stimulus, EMG activity from the first 5 sec of this pressure is quantified and taken for data analysis. On the day of testing, five staircase distensions, at 5-min intervals, are given to establish the baseline response before drug administration. Staircase distension is administered 5, 15, 30, 60, 90, and 120 min after imipramine administration.



Keywords

Colorectal distension; Visceral pain; Electromyograph; Rat

Neurobiology of Migraine

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Introduction

Migraine is a common and complex brain disorder which affects up to 20% of women and 10% of men, with a peak incidence between 25 and 55 years of age. Migraine has a complex pathophysiology in which both central and peripheral components of the trigeminal pain pathway probably play a significant role, both in the symptoms and signs of the attack. Although the initial neurologic event in migraine headache development is still a controversial issue, it is recognized that migraine results from a primary dysfunction of the central nervous system occurring in the brain and/or the brainstem. This dysfunction leads to activation and sensitization of the trigeminovascular system. Trigeminovascular system activation can cause pain and central sensitization directly and it may also induce neurogenic inflammation, which may further add to the pain. Though what activates the trigeminovascular system is still not clear, there is growing evidence that migraine arises from a primary brain and/or brainstem dysfunction. Cortical spreading depression and brainstem dysfunctions are the two pivotal neurologic events to explicate the origination of migraine. Cortical spreading depression is a slowly propagating, sustained cortical neuronal depolarization wave followed by relatively long-lasting neuronal suppression over the cortex and is likely the physiologic correlate of human migraine aura. Dysfunction of brainstem nuclei and their connections to other important brain centers which play a role in the central control of nociception may activate the trigeminovascular system directly or may facilitate it's activation. These might contribute to the cascade of events that leads to other symptoms and signs of migraine.

Keywords

Migraine; Central Nervous System; Pain Neuron

Importance of TRP Channels, Oxidative Stress and Calcium Signaling in Diabetic Neuropathic Pain

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Abstract

Peripheral neuropathy is one of the most common early complications of diabetes in ~66% of these patients. Increasing the local Ca^{2+} concentration at the site of injury or in the spinal cord may contribute to the development or neuropathic pain. The main routes of extracellular Ca^{2+} influx to the cells are voltage-dependent Ca^{2+} channels. Recently, there are also some reports on TRP channels in the routes (1). Some subfamilies of the TRP channels such as TRPV1 (vanilloid) and TRPM2 (melastatin) were discovered within last 12 years and they are activated by oxidative stress and they are involved in Ca^{2+} homeostasis disruption in neuronal cells. Subtype of TRPM2 and TRPV1 cation channels is widely expressed in neuronal and the brain cells (2). TRPV1 is a gated by noxious heat, oxidative stress and the pungent ingredients of hot chili peppers (capsaicin) and it is inhibited by capsazepine. TRPM2 channels are also activated by oxidative stress and they are inhibited by 2-Aminoethoxydiphenyl borate (2-APB) and anthranilic acid (ACA). However, there is scarce report on activation and inhibition mechanisms of the channels. I will talk on the role of Ca^{2+} signaling through cation channels and oxidative stress in diabetic neuropathic pain of sensory neurons.

The pathogenesis of diabetic neuropathy involves the polyol pathway, advanced glycation end products, oxidative stress, protein kinase C activation, neurotrophism, and hypoxia. Experimental studies with respect to oxidative stress and Ca^{2+} signaling, inhibitor roles of antioxidants in diabetic neuropathic pain are also summarized in the talk. We hypothesize that deficits in insulin, triggers alterations of sensory neurone phenotype that are critical for the development of abnormal Ca^{2+} homeostasis and oxidative stress and associated mitochondrial dysfunction (3).

Oxidative stress-dependent Ca^{2+} over influxes through the TRP channels have also important role in diabetes and diabetic neuropathic pain. Such changes in transmission within the spinal cord may contribute to diabetic neuropathic pain. It seems that the

TRPC, TRPM and TRPV groups are mostly responsible from diabetic neuropathic pain.

In conclusion, I suggest that hyperglycaemia-dependent alterations of Ca^{2+} influx through cation channels, mitochondrial function and oxidative stress induced parallel pathophysiological mechanism in diabetic neuropathy. In this mechanism, impaired insulin signaling and Ca^{2+} influx through TRP and voltage gated Ca^{2+} channel activations triggers sensory neuron mitochondrial depolarization. The consequent increase in mitochondrial depolarization induces further ROS production and disrupts Ca^{2+} homeostatic mechanisms, particularly voltage gated Ca^{2+} channels. However, there is need future studies on antagonists of TRPV1, TRPM7 and TRPM2 channels in peripheral neuron.

Key words

Calcium Ion; Diabetes; Sensory Neurons; Pain; Oxidative Stress; Transient Receptor Potential Channels

References

1. Nazıroğlu M, Dikici DM, Dursun Ş. Role of oxidative stress and Ca^{2+} signaling on molecular pathways of neuropathic pain in diabetes: Focus on TRP channels. Neurochem Res. 2012;37:2065-2075.
2. Nazıroğlu M. Molecular role of catalase on oxidative stress-induced $Ca^{(2+)}$ signaling and TRP cation channel activation in nervous system. J Recept Signal Transduct Res. 2012;32(3):134-141.
3. Kostyuk E, Svichar N, Shishkin V, Kostyuk P. Role of mitochondrial dysfunction in calcium signalling alterations in dorsal root ganglion neurons of mice with experimentally-induced diabetes. Neuroscience 1999;90:535-541.

Automated Ion Channel Recordings and in Silico Modelling

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Abstract

Patch Clamp represents the Gold Standard in ion channel research. It is the only available approach offering real time voltage control in the biophysical characterization of ion channels and in the research of their pharmacological response. The conventional manual technique suffers from many limitations, such as a complex experimental setup and the need for specially trained scientists, who can perform only low numbers of experiments.

Automated patch clamp platforms try to address these limitations. The CytoPatch was developed to mimic the manual patch clamp technique. It produces data of the same quality that experienced electrophysiologists generate from their manual patch clamp setups. The system uses glass pipettes, which are integrated into a microchip with sophisticated microfluidic solution channels to support extremely rapid solution exchange experiments. The technology has been validated for a large number of ligand gated and voltage gated ion channels in both, cultured cells and primary cells, such as neurons, myocytes and leukemia cells.

Here we present results obtained for a variety of ion channels and cell types as well as a project, in which high quality automated patch clamp data were obtained with the CytoPatch instrument to validate and improve a structure based in silico approach for the prediction of the hERG liability of lead compounds. By experimentally determining the mechanism of hERG inhibition for a set of test compounds, the underlying model of hERG blockade on which the applied in silico model is based, could be verified.

Key words

Patch-Clamp; Cytopatch; Neurons

Comparisons of Ion Channel Expression Patterns in Brain and Heart in Hyperhomosisteinemic Mice

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Introduction

Elevated homocysteine levels are defined as hyperhomocysteinemia (HHcy), a disorder that is associated with cardiovascular and neurodegenerative diseases. HHcy caused apoptozis in brain and cardiovascular tissue by induced oxidative stress. Changes in the gene expressions for ion chanels in brain and heart tissues in HHcy have not been previously reported. Ion channels are involved in a large variety of cellular processes. Numerous families of ion channels are present in the different tissues. To characterize the distinct expression of this group of ion channels we have compared the mRNA expression levels of ion channel genes in brain and heart tissues of HHcy and control mice.

Material-Method

C57BL/6 J. mice were divided into two groups of 10 animals each: (1) control group and (2) methionine group. HHcy was induced by methionine administiration. After the animals were decapitated at the end of the 10th week, the blood was collected, brain and heart were removed. Ion channel expression were detected Real time PCR.

Results

This comparison revealed that the TRPM2, TRPC3, HCN4 and ASIC2 ion channels expression in brain tissues showed an upregulation of while heart tissues were unchanged in HHcy mice compared with control animals.

Discussion

To conclude, brain exhibit a very considerable and diverse ion channel expression pattern between HHcy and control animal. Hcyneurotoxicity in the CNS is related to increased cytosolic

Ca^{2+}. TRPM2,HCN4 and TRPC3 are Ca^{2+}. permeable ion chanels. Our results provide preliminary indication that Hhcy may be increased some ion channel expression in brain tissues, especially calcium ion chanels. Their detailed analysis could give an insight into their contribution to many cellular processes and even disease mechanisms.

Effects of Homocysteine on Ca^{2+} Signaling, Apoptosis, TRPV1 and TRPM2 Cation Channels in Hippocampus of Aged Mice

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Abstract

Homocysteine (Hcy) is a sulfur-containing amino acid is totally absent from any dietary source but instead is formed during metabolism of the essential amino acids methionine. Increased level of Hcy (15μmol/L<) plays important role in etiology of neurological diseases such as Alzheimer and Parkinson's diseases. Elevated amount of Hcy cause to oxidative stress in neurons and this increased oxidative stress parameters negatively affect Ca^{2+} entry and apoptosis through the cation channel activity and it may effect activation of oxidative stress-dependent activation of TRPM2 and TRPV1 in the hipocampal neurons. The main objective of the current study was to investigate effects of Hcy on Ca^{2+} influx through TRPV1 and TRPM2 channel activations, apoptosis, cytosolic reactive oxygen species (ROS) production, caspase activities in hippocampal neurons of aged mice.

Hipocampal cells were freshly isolated form the aged (10-12 months old) mice and they were divided into four main groups namely control, Hcy, Hcy+Ca^{2+} channel blockers and Hcy+apoptosis. After incubation of Hcy (250μM and 30 min) the neurons were incubated different channel blockers and they were stimulated with H_2O_2 (0.1 mM) or capsaicin (0.1 mM) concentrations for Ca^{2+} signaling experiments. Intracellular free Ca^{2+} concentration [Ca^{2+}]i was lower in Hcy group than in control although its concentrations were lower in Hcy+TRPM2 channel blockers (ACA and 2-APB), Hcy+SERCA inhibitor (thapsigargin), Hcy+NMDA receptor blocker (MK-801), Hcy+verapamil+diltiazem (voltage gated Ca^{+2} channel blockers), capsazepine (TRPV1 channel blocker) than in Hcy group. Apoptosis, ROS, caspase-3, caspase-9 values were also lower in Hcy group than in control although cell viability value was lower in Hcy group than in control.

In conclusion, the present study demonstrates that Hcy induced degenerative effects on oxidative stress, $[Ca^{2+}]i$ influx, ROS production and apoptosis through TRPM2 and TRPV1 channel activations in hippocampal neurons. Since hyperhomocysteinemia is a common feature of oxidative stress-induced neurological diseases of hippocampal neurons, our findings are relevant to the etiology of pathology in oxidative stress-induced neurological diseases of the hippocampal neurons.

Keywords

Homocysteine; Pain; Dorsal Root Ganglion; Hipocampus; Caspases; Calcium ion

Acknowledgement

The study was supported by Scientific Project Unit of Suleyman Demirel University (Project no: 3518-YL2-13).

The Effect of Melatonin on Biomechanical Properties of Radiation – Induced Deteriation of Rat's Bon

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Abstract

In this study, the radio-protective effects of melatonin on the radiation-damaged bones were investigated comparatively with the amifostin by using the biomechanical parameters.

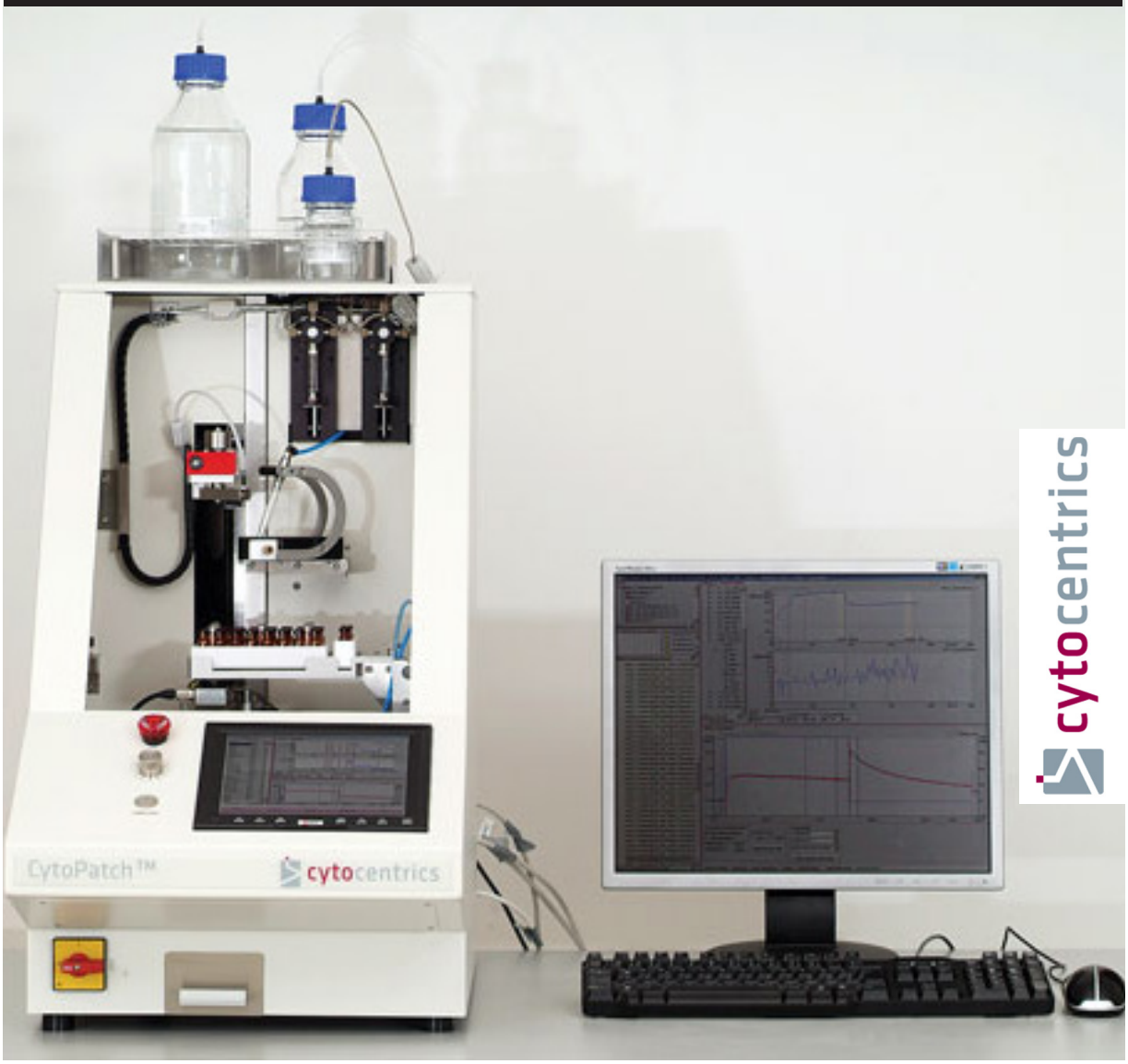
Fort this purpose, 40 female Sparague Dawley rats were divided into 5 groups. These are control group (C; n=8: saline, 1 ml i.p.), radiation (R; n=8; saline, 1 ml i.p.), radiation + melatonin 25 (R+M25; n=8: 25 mg/kg, i.p.), radiation + melatonin 50 (R+M50; n=8; 50 mg/kg i.p.) and radiation + amifostisin (R+WR; n=8; 200 mg/kg i.p.). A single dose of 50 Gy gamma radiation was exposed to the left legs of the rat groups of R, R+M25, R+M50 and R+WR. At the end of a four-week of experiment, measurements were done on the left femur of each rat by using 3 point bending test.

In the result of biomechanical analyzes, all parameters except for the young modulus of the R group, a decreasing were observed ($p < 0.05$). R+M25 and R+M50 groups, the parameters such as ultimate displacement, ultimate force, stiffness, and ultimate stress, ultimate strain were decreased according to K group but the ultimate displacement, ultimate force, toughness and energy absorption capacity parameters for R+M25 according to R group were increased statistically ($p < 0.05$). In the R+WR group, the ultimate displacement, ultimate force and energy absorption capacity increased those compared to R group, while the young modulus and toughness increased, Ultimate force and stiffness showed decreasing according to K group ($p < 0.05$).

According to these results, radiation treated to the rats reduce the bone quality; however application of melatonin showed a protective effect on bone structure same as Amifostin application.

Keywords

Radiation; Bone; Melatonin; Rat



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5th International Congress on Cell Membranes and Oxidative Stress:

Focus on Calcium Signaling and TRP Channels

Invitation

Dear Colleague;

The 4th International Congress on Cell Membranes and Oxidative Stress Focus on: Calcium Signaling and TRP Channels was organized by The Department of Biophysics, Medical Faculty of Suleyman Demirel University between 26-29 June 2012 in Isparta, Turkey. Three hundred five participants attended the congress. The abstracts of the oral presentation were published in Cell Membranes and Free Radical Research. We have since received a considerable amount of positive feedback encouraging us to organize a fifth congress, with even greater international participation. We have therefore decided to proceed with the organization of the "5th International Congress on Cell Membranes and Oxidative Stress Focus on: Calcium Signaling and TRP Channels. " It will be held between 9-12 September 2014. One of the most important aims of this Congress is to bring scientist from all around the world to Turkey, a country which has long served a role as a bridge between two continents. With this purpose we have invited scientists from abroad who are specialists in the topics of the Congress. Also we plan to organize some social activities during or around the congress, including a Pamukkale tour, which is famous for its travertines, and also Eğirdir lake tours. We will also organize two optional courses, one on patch-clamp and another on cell culture. We look forward to seeing you in Isparta in September 2014.

Speakers

Congress Language: English

Ca^{2+} signaling

Alexey Tepikin (Liverpool, UK)
ER-PM junctions: structure, dynamics and the roles in cell signaling

David I. Yule (Rochester, NY, USA)
Regulation of Ca^{2+} release through inositol 1,4,5-trisphosphate receptors

George G. Holz (Syracuse, NY, USA)
A novel PI-specific PLC-epsilon links cAMP sensor Epac2 activation to islet insulin secretion

Jonathan H. Jaggar (Memphis, TN, USA)
Vasoregulation by IP3 receptors in smooth muscle cells

Israel Sekler (Beer-Sheva, Israel)
The role of mithochondrial exchanger NCLX in cellular Ca^{2+} signaling

Johanna T. Lanner (Stockholm, Sweden)
Altered Ca^{2+} and redox handling in arthritis-induced skeletal muscle dysfunction

Kurt Beam (Aurora, CO, USA)
Altered calcium signaling in skeletal muscle caused by disease-causing mutations of the proteins mediating excitation-contraction couplin

TRP Channels

Indu S. Ambudkar (Bethesda MD, USA)
Regulation of TRPC1 and contribution to cell function

James W. Putney (North Carolina, USA)
Multiple Forms of the Store-operated Calcium Mediators, STIM1 and Orai1

Khaled Machaca (Doha, Qatar)
Role of IP3 receptor in vascular smooth muscle and hypertension development

Marc Freichel (Heidelberg, Germany)
The role of TRP channels for fertility and cardiac remodeling

Metiner Tosun (İzmir, Turkey)
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Mohamed Trebak (New York, USA)
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Mustafa Nazıroğlu (Isparta, Turkey)
Oxidative stress dependent activation of TRP channels in neurons

Scott Earley (Fort Collins, CO, USA)
TRPA1-Induced Endothelial Calcium Signals and Vasodilation

Stephan Huber (Tübingen, Germany)
TRP channels in irradiated tumor cells

Shmuel Muallem (Bethesda MD, USA)
Orai1, TRPCs or TRPMLs and TPCs

Yasuo Mori (Kyoto, Japan)
TRP channels in redox biology

Antioxidants

Cem Ekmekcioglu (Vienna, Austria)
The role of nutrition in health and behavior

José A. Pariente (Badajoz, Spain)
Melatonin and apoptosis in human leucocytes

Nic Savaskan (Erlangen, Germany)
Glioma-derived glutamate toxicity: Selenium in the limelight

Özcan Erel (Ankara, Turkey)
A Novel Method Measuring Thiol / Disuldife Homeostazis

Speakers

Oxidative Stress

Andreas Daiber (Mainz / Germany)
Crosstalk between mitochondrial and NADPH oxidase derived reactive oxygen and nitrogen species - implications for vascular function

Aron Fisher (Philadelphia, Pennsylvania, USA)
Role of Endothelial Katp channels in Ca^{2+}-mediated signaling with altered shear stress

Ismail Laher (Vancouver, BC, Canada)
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Hamid Akbarali (Virginia, USA)
Post-translational modification of calcium channels in colonic inflammation

Sven Horke (Mainz, Germany)
The role of Paraoxonases in redox and calcium homeostasis

Valerian E. Kagan (Pittsburgh, Pennsylvania, USA)
Asymmetry, oxidation and signaling "elimination" by two anionic phospholipids: cardiolipin and phosphatidylserine

Volker Ullrich (Konstanz, Germany)
NO and superoxide : a versatile couple in redox regulation

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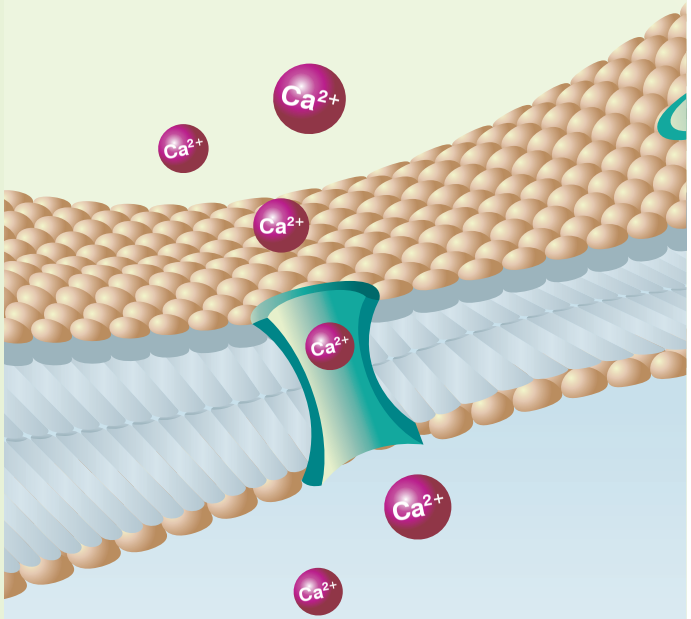
09-12 September 2014



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