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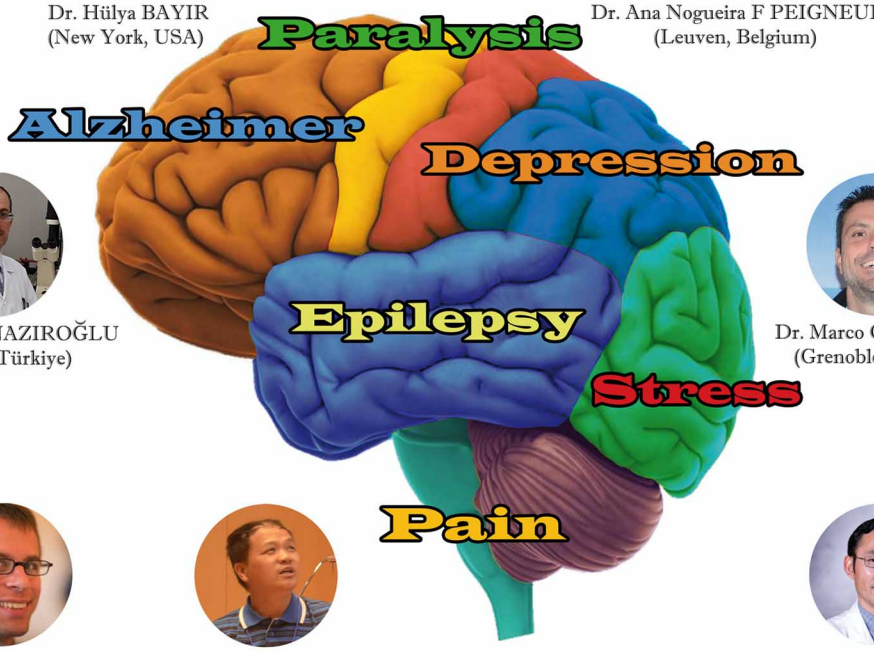

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AIM AND SCOPES
Journal of Cellular Neuroscience and Oxidative Stress is an online journal that publishes original research articles, reviews and short reviews on the molecular basis of biophysical, physiological and pharmacological processes that regulate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

Areas of particular interest are four topics. They are;

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

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

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

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

READERSHIP

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

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SPEAKERS

Speak No. 1

Ca^{2+} imaging in neuronal cells for TRP channel activation

Mustafa NAZIROĞLU
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The concentration of calcium ions (Ca^{2+}) outside of cells (1-3 mM) is ten thousand times greater than that within cells (50-100 nM). In neurons, calcium signaling links membrane excitability to the cell's biological purpose. Ca^{2+} channels are important for many different facets of brain activity. Synaptic plasticity requires Ca^{2+} signaling. Neuronal Ca^{2+} signaling involves many Ca^{2+} channels, including voltage-dependent calcium, NMDA-AMPA receptors, and transient receptor potential (TRP) channels. The internal endoplasmic reticulum depot releases Ca^{2+} through the action of ryanodine and inositol-1,4,5-triphosphate receptors. The exact control of the Ca^{2+} concertation in the cytosol within a very narrow range is facilitated by the PMCA pump, the Ca^{2+} pump of the plasma membrane, and the sarco/endoplasmic reticulum (SERCA) pump in the sarcoplasm and endoplasmic reticulum. The generation of cytosolic Ca^{2+} signals is greatly aided by the mitochondria. Thapsigargin inhibits the SERCA pumps. One ion channel that helps calcium enter the mitochondria quickly and in large quantities is the mitochondrial Ca^{2+} uniporter. Numerous dyes, including as rhodamine-123 (DHR123), DiOC6, and JC-1, are used to measure the rise in mitochondrial membrane depolarization but decrease in potential ($\Delta\Psi$) that is caused by the accumulation of Ca^{2+} within the mitochondria (Sivandzade et al. 2019). Reactive oxygen species (ROS) accumulate a result of the accumulation, and ROS are measured using fluorescent dyes such as DCFH-DA and MitoSOX mitochondria superoxide indicator in a laser scan confocal microscope. This indicates that in neuronal cells, oxidative stress, $\Delta\Psi$, and apoptosis are significantly influenced by the Ca^{2+} determination.

28 members make to the TRP superfamily. The voltage-gated and NMDA-AMPA receptor-gated Ca^{2+} channels have significantly distinct stimulators than TRP channels. For example, TRPV1, TRPM2, and TRPA1 are stimulated by capsaicin, a component of hot chili, and ADP-ribose, a DNA-damage product, and cinnamaldehyde, component of cinnamon. (Carrasco et al. 2018). Using laser scan confocal and fluorescent microscopes, scientists have long studied TRP channel stimulation-mediated Ca^{2+} in neurons using certain green fluorescent dyes, such as Fluo-3AM and Fluo-8AM (Yıldızhan and Nazıroğlu 2020). Fura-2 acetoxymethyl (AM) ester is used in spectrofluorometers and plate readers to do a ratiometric measurement of Ca^{2+} in neurons.

In summary, Fura-2 AM seems to be the optimal ratiometric method for analyzing TRP channels stimulated-Ca^{2+} analyses, while Fluo-3AM and Fluo-8AM are the most useful imaging dyes for the laser scan confocal microscope, according to the findings.

Keywords: Calcium signaling; Fluorescent dyes; Laser scan confocal microscope; TRP channels.

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SPEAKERS

Speak No. 2

Redox medicine in Traumatic Brain Injury – On the Road to Discovery and Repair

Hülya BAYIR
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No abstract

SPEAKERS

Speak No. 3

When inflammation strikes: a deep dive into the dorsal root ganglia

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Laboratory of Ion Channel Research, KU Leuven, Leuven – Belgium

Pain is an important physiological indicator that has a great impact on an organism's survival. The ability to perceive and react to noxious stimuli confers the capacity of animals, including humans, to avoid potentially dangerous situations. However, pain can persist beyond its alarm function, and become a problem of its own. It is estimated that 19% of adult Europeans suffer from moderate to severe chronic pain and still about half of these patients report inadequate pain management.

In the classical neurocentric view, the pain pathway starts when a noxious stimulus depolarizes the peripheral terminals of primary afferent neurons of the somatosensory system. The initiation of pain signals in nerve endings relies critically on a repertoire of specialized ion channels that are expressed in sensory neurons, such as TRPs, ASICs, Piezo and P2X among others. Intriguingly, emerging evidence indicates that the detection and transduction of pain stimuli is not only restricted to neuronal activity. For instance, several lines of evidence point to a key role for skin keratinocytes in the detection of thermal stimuli. More recently, a new type of Schwann cells (a type of glia in the peripheral nervous system, best known for providing myelination of peripheral axons) was identified as mechanosensitive cells in the skin, which convey nociceptive information to the adjacent DRG neurons. Nevertheless, the roles of non-neuronal cells in somatosensation and pain remain poorly understood. Notwithstanding the complete dominance of neuronal research for so many years, glial cells are now no longer considered simple support cells. In the past decades, it became clear that glial cells have an active role in numerous physiological and pathophysiological conditions, and the knowledge of glial cells' diverse physiological functions has increased ever since. Besides the aforementioned Schwann cells, there is a second type of peripheral glial cells that may be centrally involved in acute and chronic pain: the Satellite Glia Cells (SGCs).

In this lecture, we will discuss the molecular mechanisms underlying pain signaling and then delve into the molecular sensors that are present in this pathway. These sensors play a crucial role in detecting thermal, mechanical and chemical stimuli and are involved in a variety of pain syndromes, including neuropathic pain, migraine, inflammatory pain, among others. We will discuss the impact of peripheral inflammation on sensory neuron gene expression, and how this can lead to alterations in pain perception and the development of chronic pain conditions. We will explore the complex interplay of sensory neurons and satellite glial cells in the context of inflammation. Overall, this lecture will provide a comprehensive overview of the molecular mechanisms underlying pain signaling as well as the impact of peripheral inflammation in modulating pain perception. I will highlight and demonstrate the use of essential techniques in pain research, including calcium imaging, patch clamp, single cell sequencing and spatial transcriptomics.

Keywords: Pain, ion channels, satellite glial cells, inflammation, sensory neurons

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SPEAKERS

Speak No. 4

Development of new treatment for Alzheimer's disease: targeting cholesterol

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Alzheimer's disease (AD), the major cause of dementia, is an age-related mental disorder which impairs thinking, memory and day-to-day cognitive functioning, currently affecting over 34 million individuals worldwide. AD is a complex disease, associated with both environmental and genetic risk factors. AD is defined by atrophy of the cortex and hippocampus with pathological characteristic of the abnormal accumulation of extracellular amyloid β-protein (Aβ) plaques and intracellular neurofibrillary tangles (NFT), which result from hyperphosphorylated tau protein aggregation. It has been well documented that cholesterol is accumulated in the senile plaques of both AD rodent models and AD patients. According to genome-wide association studies, genes linked to cholesterol homeostasis, like the APOE gene, are associated with AD. In actuality, cholesterol and Aβ can interact directly, and excessive cholesterol enhances the development of Aβ plaques. The 5XFAD mouse model, which recapitulates the pathology of AD patients, is widely used for elucidating disease mechanism and developing new treatment. We also used this mouse model to investigate cholesterol dysregulation in the retina and in the brain.

During this presentation, I will provide an update on the state of cholesterol research in AD and discuss the results we obtained from the 5XFAD mice.

Keywords; Alzheimer's disease; Amyloid β-protein; Cholesterol; 5XFAD mice

SPEAKERS

Speak No. 5

Common methods for analyzing neuronal cell death/viability

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Neuronal cell death, a hallmark of neurodegenerative diseases and acute injuries to the nervous system, presents a complex and multifaceted challenge in neuroscience field. Quantifying neuronal cell death provides valuable information for basic research, disease understanding, drug development, toxicity assessment, and clinical applications, ultimately contributing to advancements in science and medicine. This short lecture provides an overview of common methodologies for analyzing neuronal cell death, focusing on imaging analysis of live and dead cells and cytotoxicity/viability assays.

Keywords: Cell death; Cell viability; MTT analyses

SPEAKERS

Speak No. 6

Current epilepsy treatment involves selective state-dependent blocking of voltage-gated ion channels

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Out of every 1000 people, 4 to 10 are estimated to have current epilepsy, which means they either still have seizures or need medication. Worldwide, an estimated 5 million people are diagnosed with epilepsy each year. The cause of epilepsy is still not entirely understood. Nonetheless, overactivation of voltage-gated calcium (VGCC) and sodium (VGSC) channels as well as inhibition of potassium and chloride channels are some of the suggested mechanisms. As a result, modern antiepileptic medications prevent an excessive inflow of calcium and sodium. Nevertheless, the antiepileptic medications used today either have unfavorable side effects or a restricted ability to guard against seizures.

VGCC and VGSC mutations are significant risk factors for a number of epilepsy types. Usually, the changes result in an increase in function by altering the biophysical characteristics of the channels. The development of medications that interact with several disease-causing targets is a contemporary tactic to increase the effectiveness of anticonvulsant medications and decrease their side effects. VGCC and one isoform of tetrodotoxin (TTX)-sensitive VGSC were found to be viable targets. Automated patch-clamping was utilized to test a greater number of chemicals. Stable transfected cell lines with consistently high expression levels were created and biophysically assessed prior to initiating the screening of chemical libraries.

Keywords: Epilepsy; Voltage gated sodium channels; State dependent inactivation; Automatic patch-clamp technique.

SPEAKERS

Speak No. 7

Basics of imaging Ca^{2+} and Na^{+} with low-affinity indicators

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The two cations that enter the cell when permeable ion channels open are Ca^{2+} and Na^{+}. Action potentials and excitatory synaptic potentials in neurons are generated by Na^{+} inflow, while Ca^{2+} primarily functions as a chemical messenger that initiates signaling in the cytoplasm. In addition to increasing the cytosolic Ca^{2+} content and binding to proteins in an unspecific way, Ca^{2+} influx through a particular Ca^{2+} channel can bind to proteins physically interacting with the channel in nanoscale domains. The process of measuring Ca^{2+} and Na^{+} experimentally involves adding a Ca^{2+}-sensitive fluorescent buffer, which modifies the physiological Ca^{2+} signaling. This limitation needs to be considered when planning research of this kind.

I'll give an example of how preclinical research might use Ca^{2+} and Na^{+} imaging techniques to look at the biophysical effects of channelopathies, which are the cause of many uncommon genetic illnesses.

Keywords: Calcium; Cation channels; Low-affinity indicators; Neuronal excitability.

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Oral Presentations

Oral Presentation 1

Mitochondrial translocator protein (TSPO)-induced calcium signaling stimulates oxidative injury in retina

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Age-related macular degeneration (AMD) is a factor in blindness in the elderly. There is still uncertainty regarding the cause of dry AMD. Nevertheless, accumulating evidence suggests that the primary cause of dry AMD disease is excessive production of mitochondrial reactive oxygen species in the membranes of retinal mitochondria (Lewis Luján et al. 2022). The primary cause of the overproduction of mitochondrial reactive oxygen species in the retina is an excess of calcium ion (Ca^{2+}), which enters mitochondria through their outer membrane and increases the depolarization of the mitochondrial membrane. A protein that binds to cholesterol, mitochondrial translocator protein (TSPO), is involved in the transport of cholesterol within mitochondria as well as other biological processes (Biswas et al. 2018).

Transient receptor potential melastatin 2 (TRPM2) is a Ca^{2+}-permeable cation channel. According to a recent report, the increase of Ca^{2+} concentration, apoptosis, mitochondrial membrane depolarization, and mitochondrial reactive oxygen species in retinal pigment cells can be attributed to the activation of TRPM2 (Özkaya et al. 2021). I will review current research on TSPO and calcium signaling in retinal cells in this session. I will concentrate on the role played by the TRPM2 channel in the oxidative damage to the retina caused by the TSPO gene.

In summary, research from the literature now available shows that TSPO stimulates TRPM2 in retinal cells, which in turn increases apoptosis and mitochondrial reactive oxygen species. It appears that TSPO deletion reduces mitochondrial reactive oxygen species-mediated oxidative cytotoxicity by influencing calcium signaling in retinal pigment cells.

Keywords: Age-related macular degeneration; Calcium signaling; Mitochondrial translocator protein; Oxidative stress;

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Oral Presentations

Oral Presentation 2

A review of the research on the expression levels of TRPM2 channels during brain development

Afra Gül YÖNET
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During the development of the brain, synaptic connections between nerve cells are being established with remarkable specificity. This is achieved by a series of steps such as synapses and contacts of axons and dendrites. Several triggers, including an increase in microglial reactive oxygen species (ROS), can affect the brains development. By increasing cation channel activation in response to the excessive Ca^{2+} influx, ROS are increased. The brain and neurons are particularly vulnerable to an increase in ROS due to three factors: (1) a lack of antioxidants; (2) a high concentration of polyunsaturated fatty acids; and (3) oxygen consumption. The perinatal brain development has an impact on the rises in ROS in the brain and neurons.

The stimulation of multiple cation channels causes the Ca^{2+} influx from the outside of the neurons. Transient receptor potential (TRP) is a cation channel superfamily. TRP melastatin 2 (TRPM2) channel is a subset of the TRP superfamily (Zong et al. 2024). An enzyme is located in the channel's C domain. The brain and neurons experience an increased Ca^{2+} influx if the enzyme is triggered by oxidative stress and ADP-ribose. In the course of the prenatal brain's development, numerous cellular and hormonal cues alter the amounts of TRPM2's mRNA and protein expression. According to the findings of a recent study, the levels of TRPM2 expression were identified in three neuron sections of the brain (neuronal, astrocytic, and microglial) and four crucial brain regions (cortex, striatum, and cerebellum) (Ratnam et al. 2018). Additionally, it has been documented that experimental animals' growing microglia can induce hypoxia-induced brain injury by upregulating the amounts of the mRNA TRPM2 expression (Turlova et al. 2018). I will go through the TRPM2 expression levels during brain development in the oral presentation.

The present literature results indicate that the increase of TRPM2 expression has a main role in the induction of PD and activation of microglia in the experimental animals.

Keywords: Brain development; mRNA expression; Oxidative stress; Parkinson’s disease; TRPM2 channel.

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Oral Presentations

Oral Presentation 3

Neurodegenerative disease, oxidative stress, and TRP channels

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Neurodegenerative disorders include a number of diseases. The prevalence of neurological disorders such as Parkinson's disease (PD) and Alzheimer's disease (AD) has increased significantly in the last ten years. Evidence is accumulating that neurological problems afflict 2–4% of the over-65 populations annually (Zong et al. 2024). Numerous physiological processes, including phagocytosis and mitochondrial ATP synthesis in the brain and neurons, produce free reactive oxygen species (ROS). It's unclear exactly what causes neurodegenerative diseases. However, increasing production of ROS causes the onset of neurodegenerative diseases, including PD and AD. Accordingly, it has been noted that some antioxidants offer protection against AD (Çınar and Nazıroğlu 2022).

Transient receptor potential (TRP) channels are Ca^{2+}-permeable cation channels. The TRP superfamily is divided into six subgroups, including TRP melastatin (TRPM), TRP canonical (TRPC), and TRP vanilloid (TRPV). ROS activate TRP channels in addition to a range of chemical, mechanical, and thermal stimuli, pH, osmolarity, and second messengers (Johnson et al. 2023). The high Ca^{2+} influx causes an increase in ROS production by raising TRP channel activity. ROS activates eleven of the 28 TRP channel members. TRP channels have been connected to a number of neurological disorders, such as PD and AD. I will focus on the most recent involvement of TRP channels, with a special emphasis on the recently identified functional roles of oxidative stress-dependent activated TRP channels in neurodegenerative disorders associated with changes in Ca^{2+} homeostasis.

The findings available in the literature today suggest that while blocking the channels has a therapeutic effect on neurodegenerative diseases, the activation of oxidative stress-dependent TRP channels plays a major role in their formation.

Keywords: Antioxidants; Neurodegenerative diseases; TRP channels; Oxidative stress.

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Oral Presentations

Oral Presentation 4

A relationship between optic nerve damage caused by cisplatin and TRPM2 channel activation-induced oxidative stress

Dilek ÖZKAYA
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Cisplatin is a popular and efficient chemotherapeutic drug that is used to treat several cancer types. The induction of apoptosis and the excessive formation of reactive oxygen species (ROS) are recognized as the anticancer activities of cisplatin; however, antioxidants like resveratrol and melatonin have been shown to control the excessive generation of ROS (Özkaya and Nazıroğlu 2020; Polat et al. 2023). Previous animal and clinical investigations have demonstrated the toxic effects of cisplatin on the retinal and corneal tissues, which may result in vision loss (Ferah Okkay et al. 2023; Polat et al. 2023). According to our recent study (Özkaya and Nazıroğlu 2020), it appears that the high ROS production brought on by the cisplatin treatment activates the TRPM2 channel by causing DNA damage and ADP-ribose formation.

Excessive Ca^{2+} influx in the optic nerve, mediated by TRPM2, then triggers the apoptotic pathway and death receptor signaling. Antioxidant therapies reduce the ROS that are produced in the optic nerve by TRPM2-stimulation. I will provide an overview of the current research on the effects of cisplatin on optic nerve damage and TRPM2 channel activation in the experimental animals during my oral presentation.

The results of the current literature suggest that TRPM2 activation-mediated ROS plays a major role in the optic nerve injury and apoptosis caused by cisplatin in experimental animals, even though the amounts of injury and apoptosis were decreased by the use of antioxidants like melatonin and resveratrol.

Keywords: Antioxidants; Cisplatin; Optic nerve injury; Oxidative stress; TRPM2 channel.

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Oral Presentations

Oral Presentation 5

A critical review on TRPM2 channel expression increase-induced induces neuronal cell death and oxidative stress

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It has long been recognized that voltage-gated calcium and chemical channels influence the Ca^{2+} influx in neural cells. Transient receptor potential, or TRP, is a novel channel superfamily that was identified in recent decades in addition to the well-known calcium channels (Nazıroğlu et al. 2020). There are 28 members of the TRP superfamily in mammals, and their processes of activation and inhibition differ significantly from those of chemical and voltage-gated calcium channels.

Molecular pathways are then activated, which in turn stimulates the pathways leading to apoptosis and neuronal death. According to a recent study, apoptotic neuronal cells had higher levels of TRPM2 expression (Malko et al. 2021). Moreover, it has been shown that an increase in TRPM2 expression results in the apoptosis of neurons (Ahlatcı et al. 2022). In the oral presentation, I will discuss the increase in oxidative stress and neuronal cell death caused by TRPM2 channel expression.

Recent studies have provided evidence that upregulating TRPM2 expression is essential for inducing oxidative stress and neuronal cell death in neurons.

Key words: Apoptosis; Oxidative stress; Neuron death; TRPM2 channel.

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Oral Presentations

Oral Presentation 6

The relationship between the brain and microbiota in Alzheimer's disease: Particular attention to calcium signaling and oxidative stress

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Alzheimer's disease is a degenerative condition of the degenerative condition of the central nervous system that is strongly correlated with aging. It is mostly characterized by increasing memory loss and cognitive decline, which limit physiological function and force patients to depend on caregivers (Murray et al. 2022). The network of connections between different parts of the body is known as the brain-gut axis. It provides a two-way channel of communication between the brain and gut microbiota and preserves the dynamic equilibrium of the microbial system, central nervous system, and intestinal tract (Zou et al. 2023). Through the immune system (inflammatory cytokines) and the neurological system (vague nerve), which runs from the brain to the intestines, the central nervous system influences intestinal permeability and motility. It alters the makeup and structure of the intestinal flora and has an impact on the immune system, endocrine system, neurological system, and release of endotoxins (like LPS), prostaglandins, inflammatory cytokines, and neurotransmitters. The alterations accelerate Alzheimer's disease's development and progression. Alzheimer's disease is primarily caused by deficits in the NMDA receptor and Ca^{2+} influx, and the use of antibiotics reduces the amount of intestinal microbiota in the brain, which in turn lowers the levels of NMDA receptors. Furthermore, oxidative stress plays a major part in the development of Alzheimer's disease. In combination with probiotics, polyphenols and polyphenol-combined nanoparticles have been demonstrated to increase gut bioavailability and blood-brain barrier permeability, thereby reducing oxidative stress, metabolic dysfunction, and inflammation associated with gut dysbiosis and, eventually, the onset and progression of disorders affecting the central nervous system, such as Alzheimer's disease (Scuto et al. 2024).

In conclusion, Alzheimer's disease can only be induced by upregulating oxidative stress via the gut bioavailability and blood-brain barrier permeability of antioxidants, while downregulating Ca^{2+} influx through the suppression of NMDA receptors.

Key words: Alzheimer’s disease; Brain gut microbiota axis; Calcium signaling; Oxidative stress.

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Oral Presentations

Oral Presentation 7

Present progress in experimental animal models of traumatic brain damage

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Many traumatic events, like sports accidents or car crashes that result in violent impacts to the head, can result in traumatic brain injury (TBI), the world's largest cause of death. The human brain is naturally shielded from small injuries by the surrounding soft and hard structures. The brain is vulnerable to injury from powerful hits because it has a limited capacity to withstand force (Fesharaki-Zadeh and Datta 2024).

The field of TBI research has advanced significantly through the use of animal models to imitate injury at the molecular, cellular, and organismal levels. When this study moves into a secondary phase with extended and developing secondary injuries, models are meant to assess the potential for therapeutic intervention (Orbach et al. 2024). Early models examined the biophysical features of TBI, while more recent studies have focused on how these lesions modify molecular cascades. The weight drop model, which simulates the impact of a steel ball rolling down a few-meter track on an object, the piston-driven closed head injury model, which causes injury by a piston, and the Maryland model, which mimics the impact of a steel ball rolling down a track, are the three most commonly used injury simulations used in animal experiments (Freeman-Jones et al. 2023).

In the oral session, I will go over the most recent advances in the experimental animal models of TBI. By developing and evaluating novel models, we can significantly enhance our comprehension of the natural history of brain injury caused by trauma.

Key words: Brain; Experimental animals; Maryland model; Traumatic brain injury.

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Oral Presentations

Oral Presentation 8

Antioxidant treatment reduces sciatic nerve injury-induced TRPV1 channel stimulation and oxidative stress

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There are multiple reasons that can lead to sciatic nerve injury (SCI), including compression, ischemia, and direct destruction. Numerous pathophysiological processes, including oxidative stress and apoptosis, are triggered following a SCI injury. The overproduction of reactive oxygen species (ROS) causes oxidative stress. The SCI produces an excessive amount of ROS due to the action of ischemia, and direct damage. St. John's wort, or Hypericum perforatum, is a medicinal plant. Hypericin as a component of Hypericum perforatum has potent antioxidant properties through the upregulation of enzymatic and nonenzymatic antioxidants. According to new findings, Hypericum perforatum may have a protective effect on the healing of wounds and nerves, including sciatic nerve injury in experimental animals (Uslusoy et al. 2017). Another antioxidant is quercetin. As a flavonoid, quercetin treatment reduced SCI-injury induced oxidative stress in the rodents (Fideles et al. 2023).

Ca^{2+} influx is increased by a variety of channels, including voltage- and chemical-gated calcium channels, transient receptor potential (TRP) cation channels, and others. The TRP vanilloid 1 (TRPV1) subfamily activates in response to ROS. Recent studies have shown that TRPV1 channel activation aggravates rodent sciatic nerve damage (Silva-Cardoso et al. 2021). Conversely, SCI-induced damage, pain, and apoptosis were controlled by antioxidant blocking of the channels. Furthermore, Hypericum perforatum was found to lessen the pain and increase the Ca^{2+} influx induced by SCI (Uslusoy et al. 2017). During my oral presentation, I will go over the most recent findings regarding the impact of antioxidants on TRPV1 channels in SCI.

Keywords: Antioxidants; Oxidative stress; Sciatic nerve injury; TRPV1

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Oral Presentations

Oral Presentation 9

The interaction between sevoflurane anesthesia and calcium signaling in cerebral ischemia

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Reactive oxygen species (ROS) are produced in excess when there is oxidative stress. Numerous pathological and physiological activities result in the production of ROS. Brain ischemia plays a crucial part in the pathophysiological process. One typical neurological complication following anesthetic surgery is postoperative cognitive impairment, which has grown to be a significant problem influencing older patients' postoperative recovery and quality of life. A popular clinical inhalation anesthetic, sevoflurane, has a stronger effect on postoperative cognitive performance in rats by inhibiting ROS, Ca^{2+} influx, and inflammation (Zhang et al. 2022; Liu et al. 2023).

Numerous channels, including transient receptor potential (TRP) cation channels, voltage- and chemical-gated calcium channels, and others, increase Ca^{2+} influx. ROS triggers the activation of the TRP vanilloid 1 and TRP melastatin 7 subfamilies. When there is cerebral ischemia, anesthetic drugs are neuroprotective. In experimental models of ischemia, a variety of anesthetic protective mechanisms have been investigated, including the mitigation of TRP channel activation excitotoxicity through TRP channel blockage. Because anesthetics like isoflurane prevent intracellular Ca^{2+} influx in rats by blocking the TRP channel, which causes cognitive impairment, I will discuss the most recent research on the effects of sevoflurane anesthesia on TRP channels and oxidative stress in cerebral ischemia during my oral presentation.

Keywords: Cerebral ischemia; Oxidative stress; Sevoflurane anesthesia; TRP channels.

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Oral Presentations

Oral Presentation 10

Effect of TRPV1 inhibition in epilepsy

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Between 1% and 2% of people suffer from epilepsy globally. Worldwide, an estimated 5 million people are diagnosed with epilepsy each year. Modern antiepileptic drugs can help reduce the frequency of epileptic episodes, yet the exact etiology of epilepsy is still unknown. Oxidative stress and changes in calcium channel function are two of the proposed pathways. The buildup of calcium ions (Ca^{2+}) in the neurons of the hippocampus has long been recognized as a significant factor in the development of epilepsy. The Ca^{2+} permeable transient receptor (TRP) superfamily includes TRP vanilloid 1 (TRPV1). Capsaicin dramatically activates TRPV1 (Zavala-Tecuapetla et al. 2020). Oxidative stress also stimulates the TRPV1 channel, while capsazepine and 5'-iodoresiniferatoxin (IRTX) inhibit it. The growing body of research on the therapeutic potential of TRPV1 continuously supports the hypothesis that TRPV1 channel blockage likely explains many of the benefits associated with epilepsy, such as improved apoptosis, antioxidant, and controlled Ca^{2+} entry (Pasierski and Szulczyk 2022). A limited number of recent studies have connected TRPV1 to epilepsy, indicating that this could be a novel target for potential drugs to treat the disorder (Nazıroğlu and Övey 2015). Consequently, oxidative stress causes TRPV1 to become active and has an epileptic effect; strong inhibitors such as IRTX may reduce this effect. Thus, hippocampal TRPV1 inhibition may represent a new target in the fight against epileptic seizures and apoptosis. The results of the research thus far point to a role for Ca^{2+} accumulation via the TRPV1 channel in the development of epileptic seizures.

Keywords: Antiepileptic drugs; Oxidative stress; Seizures; TRPV1 channel.

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Oral Presentations

Oral Presentation 11

High speed calcium imaging on hippocampal slices

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Calcium imaging is a valuable technique in neuroscience, as changes in calcium ion concentration provide crucial insights into neural activity. However, it is limited by photo-bleaching, photo-toxicity, and high noise levels, which complicate achieving high-quality spatiotemporal resolution (Robbins et al., 2021).

We aimed to study rapid neural activity within the hippocampus using calcium imaging. To this end, we employed a novel camera (Kinetix Scientific CMOS) capable of recording at up to 80 kHz with 8-bit resolution. Hippocampal brain slices from mice (~3rd postnatal week) were dyed with Fluo-4, a Ca^{2+} indicator, to identify calcium signals, and bicuculline was used to induce visible network activity following direct stimulation on the slice. We developed MATLAB scripts for photobleaching correction and signal strength enhancement through filtering.

Our results demonstrate that 8-bit recordings provide remarkable spatiotemporal resolution, capturing rapid calcium changes effectively. Also, the adjustable frame rate allowed us to balance the need for temporal resolution with the desire to image larger areas. This method may provide detailed insights into the fast dynamics of neuronal function and dysfunction across various neurological conditions without requiring genetic modification. Although, integrating GEVIs and GECIs with high-speed imaging may enhance spatial precision, benefiting from their targeted localization (Suzuki et al., 2016).

Combining this approach with patch-clamp techniques would enable exceptional acquisition speeds, if spatial resolution at the cellular level is traded for temporal resolution (Canepari & Ross, 2023). Additionally, improving data analysis techniques and computational methods could yield deeper insights into the complex dynamics observed in neural networks.

Keywords: Calcium Imaging; Fluo-4; Bicuculline; MATLAB; Hippocampus.

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Poster Presentations

Poster No, 1

Optimization study to increase transduction efficiency in T cells converted to chimeric antigen receptor T cells via lentiviral vector

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Lentiviral vectors are commonly preferred in cellular immunotherapy, especially in the genetic modification of Chimeric Antigen Receptor T cells used in the treatment of hematological malignancies.

Three variables are fundamentally important in the success of transduction efficiency. The infection unit/mL (IFU/mL) of the lentiviral vector, Multiplicity of infection (MOI) and the volume of the culture medium affect the results. IFU indicates the number of viral vectors capable of infection per unit volume. MOI is the measure of how many viruses per target cell to be infected.

In our optimization study, 6 groups, including the control group, were created with different MOI numbers by keeping the IFU and culture volume variables constant. The aim is to show that MOI does not only depend on the number of cells but also the number of T cells contacting the lentiviral vector is important. Our hypothesis is that even if the number of vectors per cell in a certain volume decreases, the probability of contact between the lentiviral vector and the T cell will increase and therefore the transduction efficiency will increase.

These results show that although the probability of T cell-viral vector contact increases by increasing the number of cells per unit volume, there is no linear relationship between them and transduction efficiency.

As a result, it was determined by flow cytometry that there were 25.6%, 23.7%, 19%, 18.6%, 16.2% and 0% CAR-T cells in the 1st, 2nd, 3rd, 4th, 5th and control groups, respectively. It is more recommended to conduct further experiments to find the optimal cell number and culture volume.

Keywords: Lentiviral vector; Transduciton; Multiplicity of infection; Infectios units; Chimeric Antigen Recetor T cell.

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