# Special Issue - Abstracts

# Abstracts of Orient Neuron Nexus 1(1), 2(1), and 3(1)

## ORIENT NEURON NEXUS NO. 1(1) 2010: 2-6 RESEARCH COMMUNICATION

### UNCOVERING NEURAL TUBE DEFECTS IN HUMAN THROUGH CANDIDATE GENE EXPRESSION PATTERNS IN MOUSE EMBRYOS

**Nor Linda Abdullah, Mustakiza Muslimin, Hoo Wan Mu, Fahimah Noor Ngah, Sheikh Mohd Norhafiz Abdul Aziz, Nor Syazwani Yip, Noraishah Mydin Abdul Aziz***

*Department of Parasitology, Faculty of Medicine, University Malaya, 50603 Kuala Lumpur, Malaysia*

ABSTRACT

Neural tube defects (NTDs) are the leading cause of disability in human arising from the malformation of central nervous system. The genes responsible and their involvement in causing neural tube defects in humans are poorly understood. Gene expression analysis in a whole organism enables the identification of the possible role of the gene being studied, If the gene is expressed in a particular tissue at a certain period of development, this spatiotemporal pattern of the gene of interest signals the possibility that the gene serves a function of being switched on in those tissue at the particular time. In this report, we have identified possible gene candidates in the mouse which may be required for the development of the neural tube, the precursor to the brain and the spinal cord. Development of the brain occurs by closure of the anterior neuropore (forms the cranial neural tube) while the spinal cord forms due to resolution of the posterior neuropore (forms the caudal neural tube). The genes Tiam1 and T-cadherin were found to be likely candidate genes for the development of the spinal cord and may serve as potential human NTDs genes.

*Keywords: neural tube development, Eph receptor tyrosine kinase, ephrin ligand, neurulation, gene expression*

** Corresponding author:*
*Dr Noraishah Mydin Abdul Aziz, Department of Parasitology, Faculty of Medicine, University Malaya, 50603 Kuala Lumpur, Malaysia. Email: noisha@um.edu.my*

## ORIENT NEURON NEXUS NO. 1(1) 2010:7-12 REVIEW ARTICLE

### SHORT-TERM CHRONIC ALCOHOL CONSUMPTION REDUCES CEREBELLAR DENTATE NUCLEUS IN THE RATS

**Tsui-Chin Wang, Naiphinich Kotchabhakdi***

*Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya, Nakornpathom 73170, Thailand*

ABSTRACT

Alcohol is a widely acknowledged neurotoxicant to the vulnerable cerebellar system. Most of recent study have focused on the intoxication of the premature cerebellar neurons during the development stages. Cerebellar dentate nucleus is regognised as the only input and final output in the cerebro-cerebellar circuitry, which connects several cortical area via ventral regions of thalamus. Thus, any toxic insults to the integral roles of this nucleus will influence the normal functions of the cerebellum. In this study, we used young adult animals, 8 weeks old Sprague-Dawley rats, to examine the permanent loss of the cerebellar dentate nuclei after daily alcohol voluntary administration for three weeks. The animals were fed with different concentration of ethanol in fixed volume with liquid diet supplement to abserve and estimate the total cell loss in cerebellar dentate nucleus. The large and small neurons were digitally counted in serial sections and the relative densities of the two populations of neurons of dentate nucleus were estimated and compared in relation to the effects of different concentrations of alcohol versus that in non alcoholic group. The quantitative analysis showed a significant reduction in the total cell numbers of the cerebellar dentate nucleus in both large and small neuronal cells in dose-dependent fashion despite short-term, chronic alcohol exposure.

*Keywords: cerebellar dentate nucleus, alcohol, neuronal reduction, Nissl stain*

**Corresponding author:*
*Dr Naiphinich Kotchabhakdi, Director of Salaya Stem Cell Research and Development Project, Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Salaya Campus, Nakornpathom 73170 Thailand. Email: scnkc@mahidol.ac.th*

## ORIENT NEURON NEXUS NO. 1(1) 2010:13-16 REVIEW ARTICLE

### NORMOBARIC HYPEROXIA TREATMENT IN TRAUMATIC BRAIN INJURY: A FOCUS ON BASAL GANGLIA

**Fatin Azwa Haruddin**

*Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia*

ABSTRACT

Traumatic brain injury (TBI) is known to inflict significant morbidity and mortality worldwide. In severe TBI cases, the resulting physical and cognitive impairment incur high management and rehabilitation costs that crucially involve monitoring intracranial pressure (ICP) and improving brain oxygenation. Normobaric Hyperoxia Treatment (NBOT)

 © Penerbit Universiti Sains Malaysia, 2014
For permission, please email:mjms.usm@gmail.com 

is a therapeutic strategy to improve brain oxygen metabolism and to decrease ICP by reducing tissue swelling and deactivating toxin. NBOT is administrated by increasing the inspired oxygen concentration to 100% in normal atmospheric pressure. Previous studies involving NBOT had explored its effectiveness to salvage the TBI-related cognitive and motor deficits. However, the focus of these studies has frequently been on the cortical lesions despite the known facts that TBI often inflicts tissue damage to the subcortical areas such as the basal ganglia. There are growing evidence to support recent functional theories thet implicate a pivotal role of the basal ganglia in regulating normal movements and cognitive deficits may involve the different affected brain regions. This mini review attempts to highlight the key processes involved in the pathophysiology of severe TBI and offers insight into the role of NBOT by exploring its potential effects on the cerebral energy metabolism and gene expression pattern of dopamine receptor in mouse model.

*Keywords: traumatic barain injury, fluid percussion injury, normobaric hyperoxia treatment, brain energy metabolism, basal ganglia*

** Corresponding author:*
*Ms Fatin Azwa Haruddin, (formerly MSc candidate) Department of Neurosciences, School of Medical Sciences, Universiti Sains Malayasia, 16150 Kubang Kerian, Kelantan, Malaysia. Email: fatinazwa@gmail.com*

ORIENT NEURON NEXUS NO. 2(1) 2011:2-7
REVIEW ARTICLE

## GLUTAMATE RECEPTORS AND TRANSPORTERS AS COMPARATIVE CUES TO THE GLUTAMATERGIC CIRCUITS OF THE AVIAN AND MAMMALIAN BRAIN

**Islam MR[1,2]*, Muazaimi M[1], Abdullah JM[1]**

[1] *Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia*
[2] *Department of Anatomy and Histology, Bangladesh Agricultural University, Mymensingh-2022, Bangladesh*

ABSTRACT

Glutamate is the principal excitatory neurotransmitter in the central nervous system, and plays important roles in both physiological and pathological neuronal processes. Current understanding of the exact mechanism involved in glutamate-induced neuronal excitotoxicity, in which excessive glutamate cause neuronal dysfunction and degeneration, whether acute or chronic, remain elusive. Conditions, due to acute insults such as ischemia and traumatic brain injury, and chronic neurodegenerative disorders such as multiple sclerosis and motor neuron disease, suffer from the lack of translation neuroprotection in clinical setting to tackle glutamate excitotoxicity despite steady growth of animal studies that revealed complex cell death pathway interactions. In additional, glutamates are also released by non-neuronal cells including astrocytes and oligodendroglia. Thus, attempts to elucidate this complexity are closely related to our understanding of glutamatergic circuitry in the brain. Neuronal cells develop a glutamatergic system at glutamatergic synapses that utilise glutamate as an intracellular signalling molecule to characterise the output, input, and termination of this signalling. As to signal input, various kinds of glutamate receptors have been identified and cg = characterised. Na+-dependent glutamate transporters at the plasma membrane are responsible for the signal termination through sequestration of glutamate from the synaptic cleft. The signal output system comprise vesicular storage and subsequent exocytosis of glutamate by using vesicular glutamate transporters. Similar to mammalian brain, the regional differences of glutamatergic neurons and glutamate receptor neurons suggest many glutamatergic projections in the avian brain, as supported by recent evidence of glutamate-related genes distribution. Glutamatergic target areas are expected to show high activity of glutamate transporters that remove release glutamate from the synaptic clefts. This review summarizes and compares glutamatergic circuits in the avian and mammalian brain, particularly in the olfactory pathway, the pallial organization of glutamatergic neurons and connection with the striatum, hippocampal-septal pathway. Visual and auditory pathways, and granule cell-Punkinje cell pathway in the cerebellum. Comparative appreciation of these glutamatergic circuits, particularly with the localisation and/or expression of specific subtypes of glutamate transporters would provide the morphological basis for physiological and pharmacological design that supplement existing animal studies of the current proposed mechanisms that underlie glutamate-induced neuronal excitotoxicity.

*Keywords: central nervous system, vesicular glutamate transporters, glutamate receptors, mRNA expression, excitotoxicity*

** Corresponding author:*
*Dr M Rafiqul Islam, PhD, TWAS–USM Postdoctoral Fellow, Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia. Email: rafiqah77@yahoo.com*

ORIENT NEURON NEXUS NO. 2(1) 2011:10-14
REVIEW ARTICLE

## ROLE OF A7 NICOTINIC ACETYLCHOLINE RECEPTOR SUBTYPE IN NEUROPROTECTION: AN OVERVIEW

**Muthuraju S, Abdullah JM***

*Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia*

ABSTRACT

Neuronal cell death results from various circumstances such as hypoxia, ischemic and neurodegenerative diseases (NDs). In these events, the resulting modification of

neurotransmitter, either excitatory or inhibitory, mediate much of the neuronal damage. However, this consequence depands upon their pre and post synaptic receptor activities which are the key mechanism for signal regulation. Among these, acetylcholine (Ach) is a well known neurotransmitter which is predominantly involved in the neuroprotection as well as cognitive functions through its receptors activity, particularly the nicotinic subtypes. Several lines of evidence suggest that among these subtypes, α7 nicotinic acetylcholine receptor (α7nAChR) offers much promise for neuroprotective role in relation to the central nervous system (CNS) disorders like schizophrenia and Alzheimer's disease (AD). Several lines of evidence exist to show the potential mechanisms in which this nAChR subtype and its agonists such as nicotine, that trigger the α7nAChR-mediated suppression of neuronal cell death. This review focused in the potential role of α7nAChR in neuroprotection by examining recent experimental data, both in vitro and in vivo, that argue for the neuroprotective role of α7nAChR in the CNS.

*Keywords: neuroprotection, α7nAChR nicotinic acetylcholine receptor, hypoxia, glutamate, ethanol, oxygen-glucose deprivation*

** Corresponding author:*
*Prof Dr Jafri Malin Abdullah, MD PhD, Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia*
*Email:brainsciences@gamil.com*

ORIENT NEURON NEXUS NO. 3(1) 2012:1-7
REVIEW ARTICLE

## THE POTENTIAL OF MALAYSIAN SEA CUCUMBER EXTRACTS IN REGENERATIVE MEDICINE: HOW FAR WE CAN GO?

**Azim Patar[1], Hasnan Jaafar[2], Syed Mohsin Syed Sahil Jamalullail[2], Jafri Malin Abdullah[2]**

[1] *Regenerative Medicine Institute (REMEDI), National Centre for Biomaterial Engineering Science (NCBES), National University of Ireland Galway, Ireland*
[2] *Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia*

ABSTRACT

Historically and continually ever since the time immemorial, sea cucumber extracts in Malaysia have been extensively utilized by the locals as home-based remedies to treat a wide range of ailments, including rheumatoid arthritis, abdominal pain, liver damage, and heart diseases. One species of many interest, Stichopus variegatus (also known as S. hermanii), and its reputed efficacy of this species particulary on the central nervous system injuries is remains unexplored. However, the limited data on the bioactive compounds and its efficacy studies currently does not support the fact that this species had a future medication development in regenerative medicine especially in spinal cord injury and traumatic brain injury. This review provides the current and past data on regenerative properties of sea cucumber extracts in Malaysia, toxicology studies and the treatment strategies towards understanding the possible mechanism of actions of sea cucumber extracts particularly in spinal cord injuries.

*Keywords: Stichopus variegates, regeneration, nervous system, medicine, Malaysia*

** Corresponding author:*
*Azim Patar, Regenerative Medicine Institute (REMEDI), National Centre for Biomaterial*
*Engineering Science (NCBES), National University of Ireland Galway, Ireland*
*E-mail: azim.patar@gmail.com, Tel: +353 91 495166, Fax: +353 91 495547*

ORIENT NEURON NEXUS NO. 3(1) 2012:8-13
REVIEW ARTICLE

## THE OUTCOME OF MILD TRAUMATIC BRAIN INJURY

**Mohamad Rafiqul Islam[1,2] and J M Abdullah[1]***

[1] *Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kota Bharu, Kelantan, Malaysia*
[2] *Department of Anatomy and Histology, Bangladesh Agricultural University, Mymensigh-2202, Bangladesh*

ABSTRACT

Mild traumatic brain injury has emerged is one of the most common neuronal insults and can lead to long-term disabilities. Our limited understanding of the underlying pathological changes makes it difficult to predict the outcome of mild traumatic brain injury. A neuronal degeneration, initial axonal swelling, axonal degeneration or injury, apoptotic cell deaths are common in early outcomes of mild traumatic brain injury. In addition, recent evidence suggests that mild traumatic brain injury may induce long-term neurodegenerative processes, has been found to continue even years after injury in humans, and seems to play a key role in the development of Alzheimer's disease-like pathological changes. Here we review the current understanding of mild traumatic brain injury that may represent important therapeutic targets in the treatment of mild traumatic brain injury and potentially the mitigation of chronic neurodegeneration.

*Keywords: traumatic brain injury, neuronal degeneration, post-traumatic stress disorder*

*Corresponding author:*
*Prof Jafri Malin Abdullah, Department of Neurosciences, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kota Bharu, Kelantan, Malaysia. Email: brainsciences@gmail.com, tel: +609-7676300, fax: +609-7673833.*

ORIENT NEURON NEXUS NO. 3(1) 2012:14-18
REVIEW ARTICLE

# THE VALUE OF ZIZYPHUS MAURITIANA AND MYRISTICA FRAGRANS AS A NEW DRUG DISCOVERY IN THE FIELD OF NEUROSCIENCE

**Ahmad Tarmizi Che Has**

*Faculty of Pharmacy, Pharmacy and Bank Building (A15), Camperdown Campus, University of Sydney, Sydney NSW 2006*

ABSTRACT

Zizyphus mauritiana has been used to soften human rigor mortis for centuries. The deceased is bath with it to make handling of the corpse easier during preparation. The mechanism of its action is not well known. Myristica fragrans has been used to treat epilepsy in developing countries by making it into a tea drink. It effects on the central nervous system is not well studied.

*Keywords: Zizyphus mauritiana, myristica fragrans, neuroscience, drug, discovery, ethnopharmacology*

*Corresponding author:*
*Ahmad Tarmizi Che Has, Faculty of Pharmacy, Pharmacy and Bank Building (A15), Camperdown Campus, University of Sydney, Sydney NSW2006. Email: ache4838@uni.sydney.edu.au*