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# 17th Global Neuroscience Conference

October 16-17, 2017 Osaka, Japan

## Scientific Tracks & Abstracts

## (Day 1)

Shang-Hsun Yang et al., J Neurol Neurosci 2017, 8:6
DOI: 10.21767/2171-6625-C1-005



## A miracle (miR-196a) in the fight against Huntington's disease

**Shang-Hsun Yang and Chang ChihYi**
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Huntington's Disease (HD) is a genetic disease and caused by a mutation in Huntingtin gene, leading to neuro-pathological symptoms. To date, there is no effective medicine for HD. Based on previous studies, transcriptional regulation is impaired during the progression of HD, and regulation of microRNA (miRNA) is one of affected mechanisms. Since HD leads to dysfunction of gene regulation and one miRNA could target to multiple pathways, it suggests miRNA could be one potential treatment for HD. In my laboratory, we identified one potential miRNA, miR-196a, from HD transgenic monkeys and found the neuro-protective effects of miR-196a on HD in cell, transgenic mouse and HD patient-derived induced pluripotent stem cell models. miR-196a could not only improve molecular, neuro-pathological and behavioral phenotypes in transgenic mouse models, but also suppress pathological aggregates in neurons derived from HD patients. Furthermore, we also investigate molecular mechanisms of miR-196a and show miR-196a could enhance cellular morphology, intracellular transport, synaptic plasticity, neuronal activity, learning and memory both *in vitro* and *in vivo*. In addition, miR-196a could work through binding to 3' untranslated region of *RAN* binding protein 10 (*RANBP10*) to suppress the protein expression, further enhancing the assembly of β-tubulin. Most importantly, overexpression of *RANBP10* led to worse neuronal morphology and severer pathological phenotypes in the HD transgenic mouse model, suggesting that miR-196a enhances neuronal morphology through suppressing *RANBP10* to provide neuro-protection in HD. These results suggest the important role of miR-196a on HD and might provide a new insight of therapeutic strategy for HD.

### Biography

Shang-Hsun Yang is an Associate Professor in Department of Physiology at National Cheng Kung University, Taiwan. He has completed his BSc at National Chung Hsing University in 1998, MSc at National Taiwan University in 2000 and PhD at Emory University, USA, in 2008. His research interests focus on the regulation of microRNAs on HD.

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**Notes:**

Eyleen Goh et al., J Neurol Neurosci 2017, 8:6
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## Choline as a nutraceutical for treating neurodevelopmental disorder: Rett syndrome

**Eyleen Goh, Chin E W, Lim W W, Ma D L and Rosales F J**
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Rett Syndrome (RTT) is a postnatal neurodevelopmental disorder that primarily affects girls, with 95% of RTT cases resulting from mutations in the Methyl-Cpg-Binding Protein 2 (*MECP2*) genes. To model RTT *in vitro*, a short hairpin RNA was used to knockdown the expression of *MECP2* in primary neurons. Abnormalities in the cholinergic system have been shown to be associated with the disorder. We found choline supplementation to *MECP2*-knockdown neurons increased their soma sizes and the complexity of their dendritic arbors. Through the use of specific inhibitors targeting each of the known physiological pathways of choline, synthesis of phosphatidylcholine from choline was found to be the most important pathway in bringing about the changes seen in choline-supplemented *MECP2*-knockdown neurons. Rescue of the morphological defects could lead to enhanced neurotransmission, as suggested by an observed trend of increased expression of selected synaptic proteins in choline-supplemented cells and differences in dendritic spine density and shape between wild type and *MECP2*-knockout mice, with choline or vehicle supplementation. In addition, choline supplementation to cultured hippocampal neurons restored mini excitatory postsynaptic current frequencies in *MECP2*-knockdown cells to control levels, while the amplitude was unchanged. Choline treatment to *MECP2*-knockout mice also rescued deficits in motor coordination, anxiety-like behavior and social interaction. Taken together, these data reveal a role of choline in modulating neuronal plasticity, possibly leading to behavioral changes and hence, a potential for using choline to treat RTT.3

### Biography

Eyleen Goh is a Senior Research Scientist at the National Neuroscience Institute and an Assistant Professor with the Duke-NUS Medical School in Singapore.

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## Young Researchers Forum

## (Day 1)

Chun-Wei Hsu et al., J Neurol Neurosci 2017, 8:6
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## The effect of mental countermeasures on fMRI-based concealed information tests

**Chun-Wei Hsu and Giorgio Ganis**
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Concealed Information Paradigms (CITs) have been developed to determine if an individual is familiar with a certain piece of information such as a crime-related item. The main logic of CITs is that recognition of an item of interest (probe) will generate a differential response, compared to suitable control items (irrelevant), that can be detected by monitoring behavioral, psychophysiological or neural variables. An important issue is an extent to which countermeasures used by suspects can reduce the accuracy of the CIT. Recent work has focused on neural variables measured with Functional Magnetic Resonance Imaging (fMRI) because at first sight, such variables may seem more resistant to countermeasures than more peripheral variables. Previous work has shown that hybrid physical and mental countermeasures can decrease the accuracy of fMRI-based CITs, but questions remain as to whether purely mental countermeasures can do so as well. Existing evidence shows that attentional and memory strategies can decrease the accuracy with which one can use fMRI to detect successful recognition in standard face recognition tasks. The aim of this fMRI study was to determine if such mental countermeasures are effective also with standard CITs. Participants (N=20) were tested under three conditions: no knowledge, concealed knowledge and countermeasures. Results based on regions of interest defined in previous CIT studies showed that the area under the curve (AUC) for discriminating no knowledge and concealed knowledge cases with multi-voxel pattern analyses was 0.86 without countermeasures. Critically, memory and attentional countermeasures significantly reduced the AUC to 0.74. These results indicate that purely mental countermeasures can reduce the accuracy of fMRI-based CITs, even without extensive training of participants.

### Biography

Chun-Wei Hsu is currently pursuing her PhD in Psychology at University of Plymouth, UK. She has completed her undergraduate degree at National Taiwan University, Taiwan. She has completed her Master's degree in Cognitive Neuroscience and Human Neuroimaging at University of Sheffield. She is interested in how people conduct high-level cognition in complex social interaction and how people evaluate expect the pay-offs and take action during the decision-making process.

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**Notes:**

Amira Elaalem et al., J Neurol Neurosci 2017, 8:6
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## Relative risk factors of Bell's palsy among Sudanese patients in Khartoum state 2016

**Amira Elaalem[1] and Omer El-Adil[2]**
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**Background & Aim:** Bell's palsy is the idiopathic seventh cranial nerve palsy. It is the most common cause of abrupt onset of unilateral facial weakness. The natural history of Bell's palsy is encouraging for most of the patients since total recovery of facial function is expected nevertheless additional long term poor outcomes occur in minority of them and can be devastating. Currently, no cause for Bell's palsy has been identified in the literature, but in Sudan there were very few information can be retrieved. The overall objective of the study was to assess the potential relative risk factors of Bell's palsy in Sudanese patients.

**Methodology:** This is analytic case control multicenter based study, conducted in Khartoum state physiotherapy centers (three in hospital and seven in private sectors) July-November 2016. The sample size was 70 cases of Bell's palsy and 140 controls from the same sample area. The cases that fulfilled the inclusion criteria were collected by a method of total coverage during the working hours. Well-constructed questionnaires were filled during the interview by a trained physiotherapist. The collected data was then analyzed using SPSS software program, version 20.

**Results:** The result showed the statistically significant factors (p value<0.05) are: Recurrence of Bell's palsy ten times more in affected patients, the hereditary factor 2.5 times more in affected patients. In contrast to the other factors was non statistical significance (p value>0.05) are recent vaccination, pregnancy, diabetes, hypertension, chronic diseases, immunosuppressive drugs, smoking and alcohol.

**Conclusion:** The study concluded that, statistical significant factors are frequency of recurrence of Bell's palsy, genetic susceptibility. The rest of risk factors were statistical non-significant. We recommend more studies to be done to determine the type of inheritance in Sudan and study the cause of recurrence.

### Biography

Amira Elaalem has completed her Master's degree in Neurology Physiotherapy and BSc in Physiotherapy at Al-Neelain University Faculty of Medicine and Health Science and currently pursuing MBBS degree.

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**Notes:**

Arunkumar Prasad et al., J Neurol Neurosci 2017, 8:6
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# CNS lymphatic drainage blockade exacerbates cerebral vasospasm and cerebral injury following subarachnoid hemorrhage and partially reversed by *Ginkgo biloba* extract

**Arunkumar Prasad, Bao-Liang Sun, Xuan Wang, Li Jia, Li-Li Jia, Xi-Chang Liu, Zi-Cui Cheng, Ming-Feng Yang, Cheng-Bi Zhengb, Lei-Lei Mao, Cun-Dong Fan, Zong-Yong Zhang, Da-Wei Li, Xiao-Yi and Yang**
Taishan Medical University, China

Recent studies suggest that CNS lymphatic drainage pathway to extra-cranial lymph compartments may play an important role in the removal of substances in the brain and Cerebrospinal Fluid (CSF). After the onset of Subarachnoid Hemorrhage (SAH), large amount of macromolecular substances, such as cellular lysates, proteins, peptides, were accumulated in the brain tissue and CSF, which contribute to cerebral vasospasm and cerebral injury. The present experiment was carried out to investigate the possible role of cerebral lymphatic drainage pathway in the development of cerebral vasospasm and related cerebral injury and the influence of Ginkgo biloba extract. Wistar rats were used in the experiment and animals were divided into different groups. SAH models were replicated by double cisternal injection of autologous arterial hemolysate. In some animals the main cerebral lymphatic drainage way out being blocked (Cerebral Lymphatic Blockade, CLB). Two different constituents, ginkgolides and ginkgo flavone, were given as interventions. It was found that SAH reduced the drainage of Evans Blue-Labeled Albumin (EBA) from the brain to the olfactory bulbs, cervical lymph nodes and abdominal para-aortic lymph nodes. A kinetic analysis of 125I-labeled human serum albumin (125I-HSA), a Cerebrospinal Fluid (CSF) tracer, showed that the clearance rate of macromolecules in the CSF was significantly reduced after SAH. Furthermore, SAH reduced the diameters of Basilar Artery (BA) and increased thickness of BA. Prominent cerebral injury was found after induction of SAH. The spasm of BA and cerebral injury were partially antagonized by ginkgolides and ginkgo flavone. It was concluded that cerebral lymphatic drainage pathway exerts intrinsic protective effects against cerebral vasospasm and cerebral injury by removal of macromolecular substances in the brain and subarachnoid spaces. Ginkgolides and ginkgo flavone may alleviate the exacerbated cerebral vasospasm and cerebral injury following SAH by CLB.

## Biography

Arunkumar Prasad is the President of the student organization in the field of academics and other activities in TSMU. He is currently working as a Young Researcher and pursuing his studies under guidance of Professor Baoliang Sun.

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**Notes:**

Yusuf Issa Olakunle et al., J Neurol Neurosci 2017, 8:6
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# Fibroblast growth factor 9 suppresses cell death through ERK signaling in Huntington's disease

**Yusuf Issa Olakunle[1,2], Pei-Hsun Cheng[2], Hsiu-Mei Chen[2] and Shang-Hsun Yang[1,2]**
[1]Academia Sinica, Taiwan
[2]National Cheng Kung University, Taiwan

Huntington's Disease (HD) is a heritable neurodegenerative disorder characterized by selective and progressive damage of medium spiny neurons in the striatum and there is no cure for HD to date. A type of Fibroblast Growth Factor (FGF), FGF9, has been reported to play pro-survival roles, in other neurodegenerative diseases, such as Parkinson's disease and Alzheimer's disease. With many similarities in the cellular and pathological mechanisms that eventually causes cell death in neurodegenerative diseases, we hypothesize that FGF9 might provide neuro-protective functions in HD. Here, we used STHdh$^{Q7/Q7}$ (WT) and STHdh$^{Q111/Q111}$ (HD) striatal knock-in cell lines as our models and examined the neuro-protective effects of FGF9 on HD. Employing MTT and PI staining assays to determine cell proliferation and survival respectively, we found that FGF9 enhanced cell proliferation and also increased cell survival under a starvation stress condition. In addition, we observed that FGF9 significantly up-regulated FGF signaling through ERK1/2, Akt, JNK and mTOR pathways and increased neuro-trophic factor (GDNF) and anti-apoptotic BcL-xL proteins in HD cells. Especially, ERK pathway plays a critical role in the effects of FGF9 on cell survival and GDNF regulation. These results not only show the neuro-protective effects of FGF9, but also clarify the critical mechanisms in HD cells, further providing therapeutic potential of FGF9 in HD.

## Biography

Yusuf Issa Olakunle is currently pursuing his PhD in Taiwan International Graduate Program (TIGP) Academia Sinica in Neuroscience at National Cheng Kung University (NCKU). He has completed his BSc degree in Physiology from Ahmadu Bello University, Nigeria in 2010 and MSc degree in Physiology from the University of Ibadan, Nigeria in 2014.

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## Scientific Tracks & Abstracts

## (Day 2)

Takakazu Yokokura, J Neurol Neurosci 2017, 8:6
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# Defining conserved spinal muscular atrophy gene networks that are involved in neuromuscular system using Drosophila SMA model

**Takakazu Yokokura**
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Spinal Muscular Atrophy (SMA) is a devastating inherited disorder characterized by progressive loss of motor activity, failure of neuromuscular synapses and muscle weakness. Genetic cause of SMA is mutation of Survival Motor Neuron 1 (*SMN1*) gene, while genetic factor determining severity of symptom is copy number of Survival Motor Neuron 2 (*SMN2*) gene, which only generate small amount of SMN protein due to skipping a functionally important exon at high frequency. As SMN has been considered as a key factor to regulate neuronal cell function cell autonomously, up-regulating SMN protein in spinal cord motor neurons at pre-symptomatic stages is the most advanced therapeutic approaches to prevent, or, at least, delay irreversible loss of motor neurons. However, the fact that SMA patients exhibit muscle weakness and experience fatigue suggests that it is little known underlying mechanism how low SMN levels affect to trans-synaptic biology at the neuromuscular junction (NMJ). Trans-synaptic structure and signaling at the NMJ play important roles for establishment and maintenance of neuromuscular connectivity and functions. As pathological observation in post-mortal NMJ specimen or rodent SMA models exhibited abnormality in neuromuscular connectivity, utilizing Drosophila NMJ, which is well characterize its structure and molecular mechanism, allow us to understand how low levels of SMN perturbs structure and molecular mechanism at the NMJ in depth. Severe SMN mutants exhibited two phenotypes in motor unit known as SMA pathology, loss of motor axon and abnormality at the NMJ. Modulation of trans-synaptic two canonical signaling pathways, BMP and FGF signaling, that have shown genetic interaction to SMN, can rescue the SMN defects. In addition, each pathway seems to modulate distinct aspect of SMA motor unit pathology.

## Biography

Takakazu Yokokura has his expertise in genetics and molecular biology and passion in finding approaches to cure devastated neurological disorders, such as Spinal Muscular Atrophy (SMA) and amyotrophic lateral sclerosis. His study has focused on elucidate underlying molecular mechanisms that low levels of SMN leads to manifestation of SMA neuromuscular pathological phenotypes.

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**Notes:**

Nikki Stang, J Neurol Neurosci 2017, 8:6
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# Implementing CranioSacral Therapy for Concussions, Traumatic Brain Injury and neurological disorders

**Nikki Stang**
My Traumatic Brain Injury, USA

CranioSacral Therapy is an International Program which is taught and ran through the Upledger Institute. www.upledgerinstitute.com. Craniosacral Therapy (CST) was invented by osteopathic physician John E. Upledger when he was a Professor of Biomechanics at Michigan State University. Following extensive scientific research he found that by using a very soft-touch therapy (about 5 grams of pressure), restraints are released in the craniosacral system, which is comprised of membranes and fluid that surround and protect the brain and spinal cord. CST can support the body's natural healing process as well as serve as a preventative health measure for a diverse range of health problems and neurological dysfunction including concussion, Traumatic Brain Injury, Alzheimer's and Dementia, and Central Nervous System Disorders. Nikki Stang will talk about her personal experience with TBI including the symptoms she was experiencing and how implementing CranioSacral Therapy with other modalities eliminated many of the symptoms associated with TBI that she was experiencing. CranioSacral Therapy has proven to be effective in increasing function and decreasing pain associated with head trauma.

## Biography

Nikki Stang was born in Denver, Colorado. In 2007 she attended UNLV to start a psychology degree. Shortly after attending college she moved to Peru where she volunteered in an all girls orphanage. She developed a passion for working with children and eventually found her calling in being a gym teacher for high risk youth in the city of Denver. In 2011 she began attending massage school to learn more about the body and how she personally could help people around the world who did not have access to medical care. In fall of 2011 she was accidentally headbutted in the mouth and suffered a traumatic brain injury while playing basketball with her students. Nikki was unaware of her TBI until 2013 when she was later diagnosed shortly after she was pregnant with her first child. Nikki has recovered from her accident and is now and advocate for brain injury survivors. She has taken classes in aromatherapy and healing touch through ISHA, craniosacral therapy through the Upledger Institute, and heart centered therapy through the Chikly Health Institute. She is currently living in Denver with her two young children and is a TBI advocate, motivational speaker, and is working on writing a book about her traumatic brain injury experiences.

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**Notes:**

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## Prenatal and early postnatal caffeine exposures influenced neuron and glial morphological destinies

**Owolabi O Joshua[1], Adefule A K[2] and Shallie Philemon D[2]**
[1]Babcock University, Nigeria
[2]Olabisi Onabanjo University, Nigeria

This study investigated the effects of caffeine consumption on the brain in pregnancy and early postnatal life. Thirty two (n=32) adult mice (*Mus musculus*) were used for the study. They were maintained in Anatomy Department animal holding and given chow and water ad libitum. They were divided into four groups: Group-A animals as control, Group-B animals given low dose caffeine 10 mg/kg body weight, Group-C animals given medium dose caffeine 50 mg/kg body weight and Group-D animals given high dose caffeine 120 mg/kg body weight. The treatment duration was divided into Phases I and II to assess pre and post natal effects, respectively. Caffeine was dissolved in distilled water and Group-A animals received distilled water as placebo. Animals mated prior to treatment commencement and pregnant mice were administered caffeine throughout pregnancy. Half number of offspring were sacrificed at birth, the rest were used in Phase II and treatment continued till postnatal Day 35, marking puberty. Animals were sacrificed and brain specimens were excised and processed. Specimens were subjected to gross morphological assessments. Histological and histochemical demonstrations were done using the H&E, Bielschowsky, Luxol Fast Blue and Feulgen DNA Staining techniques. Immuno histochemical-molecular properties were demonstrated using the GFAP technique. At parturition, 50 mg/kg body weight caffeine dose disrupted deeper cortical layers histoarchitecture and extended to other layers when dosage increased to 120 mg/kg body weight. Neuronal morphological heterogeneity was observed at doses higher than 10 mg/kg body weight and some effects persisted till puberty. Astrocyte morphologies and dendritic patterns of elaboration were persistently altered. Caffeine exposure altered the pattern of brain cells development by altering morphologies, patterns of elaboration and spatial distribution, also neuronal communication and basal metabolism. Effects were dose dependent and moderate dose might be beneficial to morphological and functional parameters.

### Biography

Owolabi O Joshua has received his PhD in Anatomy and Neuroscience and MBA with specialization in Clinical Research Management. He currently works as a Teacher and Researcher in the Ben Carson [Snr.] College of Medicine, Babcock University, Nigeria.

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**Notes:**

Arash Abdolmaleki, J Neurol Neurosci 2017, 8:6
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# Synergistic effects of Citicoline and bone marrow mesenchymal stem cells to improving regenerative capacity of acellular nerve allograft

**Arash Abdolmaleki**
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Acellular nerve graft is an alternative to autograft for the repair of short gaps associated with peripheral nerve injury. It provides a suitable three-dimensional structure that supports and guides axonal regeneration. However, outcomes associated with the use of acellular nerve grafting are often inferior to those achieved with autografts, particularly over long lesion gaps. Therefore, this experimental study was conducted to evaluate the effects of citicoline on the efficacy of acellular nerve allografts seeded with Bone Marrow Stem Cells (BMSCs) to bridge a 15 mm sciatic nerve gap. Seventy (70) rats were randomly allocated into seven groups (n=10 per group), including the healthy control group, sham surgery group, autograft group, acellular nerve scaffold (ANS) group, ANS+BMSCs group, ANS+BMSCs+100 mg/kg citicoline and ANS+BMSCs+200 mg/kg citicoline groups. The two experimental groups were treated daily with citicoline at the doses of 100 or 200 mg/kg for two weeks. After implantation, motor function was assessed and electrophysiological, histomorphometry and molecular tests were performed. Animals treated with citicoline immediately after implantation showed significantly better regeneration and motor function outcome compared with ANS group and ANS+BMSCs group. No significant difference was observed between the citicoline treatment (200 mg/kg) group and the autograft group. These findings suggest that citicoline treatment resulted in improved regenerative properties of cell-seeded nerve allografts, likely via increasing the viability and retention of transplanted BMSCs.

## Biography

Arash Abdolmaleki has his expertise in peripheral nerve regeneration. His PhD thesis was about the use of acellular nerve scaffolds enriched by bone marrow mesenchymal stem cells for enhance the regeneration of sciatic nerve after implantation in rats.

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**Notes:**

Tarfa M Peter et al., J Neurol Neurosci 2017, 8:6
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# The proliferative effect on the astrocytes of the brainstem of albino mice following long term ingestion of fresh and thermoxidized palm oil diets

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The astrocytic proliferation following chronic consumption of thermoxidized and fresh palm oil diets was studied on the brainstem of growing mice. 30 mice were divided into three groups A, B and C. 15 g of thermoxidized palm oil was mixed with 85 g of mice chow (15% w/w) and given to the group C animals. 15 g of fresh palm oil was mixed with 85 g of mice chow (15% w/w) and administered to the group B animals while group A animals were fed with normal mice chow. The astrocytes in the brainstem of mice in the thermoxidized palm oil group significantly increased in number (hyperplasia) and size (hypertrophy) when compared with animals in the control and fresh palm oil groups due to the presence of concomitant evolution of toxic byproducts (free radicals, peroxides, etc.). If the results obtained in mice are applicable to man, there is reason for concern regarding adverse consequences of chronic consumption of thermoxidized palm oil diet. This may be dangerous to health since it may result in astrocytic proliferation in brainstem, thereby making the animals susceptible to loss of motor function like control of cardiovascular system, respiration, gastrointestinal tract, eye movement, etc.

## Biography

Tarfa M Peter is presently working as a Professor at Gombe State University, Nigeria

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**Notes:**