Abstracts of Presentations at the 13th Conference of the Neuroscience Society of Nigeria

Characterization of prefrontal cortex microstructure and antioxidant status in an Alzheimer's-diabetes rat model

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Abstract

Diabetes mellitus (DM) and Alzheimer's disease (AD) do co-exist in several subjects. Most non-transgenic animal models of AD make use of oral treatment with aluminium chloride ($AlCl_3$) to induce neurodegeneration; besides, streptozotocin (STZ) can induce features of either AD or DM depending on the mode of treatment. The aim of this study was to characterize prefrontal microanatomy and antioxidant defence system in a rat model of AD with DM co-morbidity. Adult Wistar rats were randomly assigned to receive either intraperitoneal STZ (30 mg/kg/day for 3 days; to induce DM), oral $AlCl_3$ (500 mg/kg/day for 4 weeks; to induce AD); or both STZ and $AlCl_3$ (to induce AD with DM co-morbidity). Untreated rats served as controls. Blood glucose was evaluated during treatment. At euthanasia, prefrontal cortex was homogenized in PBS and the supernatants assayed for antioxidant enzymes (catalase, CAT; superoxide dismutase, SOD) and reduced glutathione (GSH). Moreover, prefrontal cortices were processed by the H&E and Congo red technique. In rats co-administered $AlCl_3$ and STZ (AD+DM rats), prefrontal levels of GSH reduced significantly ($p<0.05$), while reductions in SOD and CAT were not significant ($p>0.05$) compared with the control. Moreover, AD+DM rats, extensive neuronal cell loss was observed in the prefrontal cortex, but Congophilic deposits were not present. The neurodegenerative lesions and antioxidant deficits characteristic of this $AlCl_3$+STZ (AD+DM) rat model were more pronounced than similar lesions associated with mono-treatment with either STZ (DM) or $AlCl_3$ (AD) alone. The $AlCl_3$+STZ rat model could serve as a suitable option for the study of neurodegenerative diseases (such as Alzheimer's disease) with DM co-morbidity.

Key words: Alzheimer's disease, Diabetes mellitus, Aluminium chloride, Streptozotocin, Antioxidants, Prefrontal cortex

Sodium azide- induced degenerative changes in the dorsolateral prefrontal cortex of rats: attenuating mechanisms of kolaviron

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Abstract

Evolvement of therapeutic targets following neurodegeneration is of major biomedical importance. Kolaviron (Kv) is a biflavonoid complex isolated from seeds of *Garcina kola* - a common oral masticatory nut in West Africa known to hold medicinal value. Therefore, this study evaluated the therapeutic potentials of Kv on cells of the dorsolateral prefrontal cortex (DLPFC), before or after sodium azide (NaN_3)-induced neurodegeneration. Rats were randomly assigned into 5 groups (6/group) and treated daily (orally) as follows: 1 ml of corn-oil (vehicle of Kv, 21 days); Kv only (200 mg/kg) for 21 days; NaN_3 only (20 mg/kg for 5 days); NaN_3 (20 mg/kg for 5 days) followed by Kv (200 mg/kg for 21 days); Kv (200 mg/kg for 21 days) followed by NaN_3 (20 mg/kg for5 days). After treatments, rats were sacrificed and perfused transcardially (with 4% PFA) with brains fixed in specificity of techniques demonstrated. DLPFC was examined in histology (H&E), immunoperoxidase (GFAP), immunofluorescence (iNOS and NOS) and western blotting (MAPT, MAP2, Bax, BCL-2 and CAD). Quantitative analysis was done using ImageJ software and statistical analysis with Graphpad prism (ANOVA) at $P<0.05$. NaN_3 treatment induced neuronal damage, characterized by reduced relative brain weight, pyknosis, karyorrhesis, astrogliosis, axonal/dendritic damages and cytoskeletal dysregualation that subsequently resulted in increased expressions of apoptotic regulatory proteins. These degenerative changes were relatable to the observed iNOS and nNOS upregulations. However, Kv administration attenuated the NaN_3-initiated destructive molecular cascades in the DLPFC of rats through mechanisms that involved: modulation of stressor molecules and toxic proteins, prevention of stress related biochemical redox, preservation of neuronal integrity, cytoskeletal framework and subsequently, reduced the level of apoptotic regulatory proteins. We concluded that Kv conferred therapeutic benefits on NaN_3–induced neurodegeneration especially when administered before than after the damage in rats.

Key Words: Kolaviron, Sodium azide, Neurodegeneration; Cytoskeleton, Prefrontal Cortex.

Rauwolfia vomitoria and Gongronema latifolium combination protects the cerebellum

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Abstract
Rauwolfia vomitoria and *Gongronema latifolium* are two different medicinal plants whose combination has been reported to be beneficial to the nervous tissues. Individually these plants possess useful properties, though some adverse reports have been ascribed to *R. vomitoria*. This study therefore investigated the potential of the combination of these plants on cerebella protection. Twenty young male Wistar rats of average weight 125 g were divided equally into 4 groups. Oral doses of Tween 20™ (0.5 ml), 200 mg/kg of *R. vomitoria* (RV), 200 mg/kg of *G. latifolium* (GL) and the combination of both (RV + GL) were administered respectively to the control and groups 2 - 4 animals for 14 days. On day 15, the animals were sacrificed after ketamine sedation and perfuse-fixed with buffered-formalin. Each cerebellum was excised and processed for histomorphology, and immunolabelled for glial fibrillary acidic protein (GFAP) and Ki-67. Histomorphology result of the cerebella sections revealed slight atrophy of the cells in the molecular, Purkinje and granular layers with additional pyknosis in the molecular layer cells and karyorrhexis in the Purkinje cells of the RV group. The GL group had slight hypertrophy of the Purkinje cells, while the RV + GL group showed slight hypertrophy of cells of the Purkinje and granular layers. There was much expression of GFAP in the RV group, while the GL and RV + GL groups showed decreased GFAP expression. Ki-67 was positive in the cerebella sections of the RV, GL and RV+GL groups, all compared to the control group. In conclusion, RV+GL combination protects the cerebellum, modulates gliosis, while maintaining its proliferative state.

Keywords: *R. vomitoria*, *G. latifolium*, Cerebellum, Histomorphology, GFAP, Ki-67, Wistar rat

Black seed oil ameliorated scopolamine-induced memory dysfunction and cortico-hippocampal neural alterations in male wistar rats

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Abstract
This study is undertaken to evaluate cognitive enhancing and ameliorative effects of Black seed oil. These effects were investigated on scopolamine-induced dementia model in Morris water maze test (MWM) and Y maze test. We also examined the hippocampal histoarchitectural responses to scopolamine and Black seed oil (BSO). Scopolamine (1 mg/kg ip) was given to induce dementia, followed by oral administration of BSO (1 ml/kg) for 14 consecutive days. MWM and Y-maze paradigms were used to assess hippocampal and frontal dependent memory respectively, thereafter the rats were sacrificed and brains were removed for histopathologic studies. Scopolamine resulted in memory impairment, by delayed latency in the MWM (LTM 47 sec.; STM 40 sec.), reduced percentage alternation in the Y maze (58%) that was coupled by alterations in the cortico-hippocampal neurons. Post-treatment of rats with BSO mitigated scopolamine-induced amnesia, by reducing latency period (LTM 8.9 and 18 sec.; STM 8 and 24 sec.), increasing percentage alternation (96%) and histological changes. The observed anti-amnestic effect of BSO makes it a promising anti-amnesic agent for clinical trials in patients with cognitive impairment.

Keywords: Memory dysfunction, Cortico-hippocampal neurons, Working memory, Black seed oil, Scopolamine, Ameliorative efficacy

Progesterone an antioxidant; its involvement in oxidative stress produced by trimethyltin in the hippocampus of adult male Wistar rats

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Abstract
Reactive oxygen species (ROS) are normal by-products of mitochondrial respiratory chain activity. Increased concentration of reactive oxygen species results in oxidative stress. This made ROS concentration a central feature of all neurodegenerative disorders. Hence, this study is designed to investigate the antioxidant property of progesterone (PROG) following trimethyltin (TMT) induced neurotoxicity in the hippocampus of adult male Wistar rats. Twenty four adult male Wistar rats were used in the study and divided into three groups. Group A used as control were given 0.2 ml of normal saline (NS), group B were administered 8 mg/kg TMT start dose only, while group C were administered 8mg/kg start and 16 mg/kg of progesterone daily for five days. All treatments were done intra-peritoneally. The animals were sacrificed after 26 days. The brains were excised and homogenize for enzyme analysis. Superoxide dismutase (SOD); NS 88.3267 ± 3.6851 TMT 141.6667 ± 5.27046 ,*TMT-PROG 54.5833 ± 6.87437 .*Catalase (CAT); NS 71.3333 ± 3.33333, TMT 50.5000±4.40265, TMT-PROG 78.3333 ± 3.65756. malondialdehyde (MDA); NS 133.2667 ± 1.10202, TMT 163.6667 ± 7.53820,* TMT-PROG 127.3500 ± 3.51101. The result of this study revealed that, in the TMT treated, SOD and catalase activities were significantly reduced and MDA levels were significantly increased as compared to the animals that received only normal saline. While in the progesterone treated after TMT, the activities of SOD increased significantly, no difference in catalase activity and MDA was significantly reduced as compared to the rats that took only TMT values are presented as mean±SEM (*P<0.05). This study concludes that progesterone has the ability to regulate the antioxidants activities in the hippocampus of adult male Wistar rats.

Keywords: Reactive Oxygen species, Progesterone, Trimethyltin, Hippocampus, Superoxide dismutase, Catalase, Malondialdehyde

Building sustainable neuroscience capacity in Africa: the role of non-profit organizations

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Abstract
While advances in neuroscience are helping to improve many aspects of human life, inequalities exist in this field between Africa and more scientifically-advanced continents. Many African countries lack the infrastructure and appropriately-trained scientists for neuroscience education and research. Addressing these challenges would require the development of innovative approaches to help improve scientific competence for neuroscience across the continent. In recent years, science-based non-profit organisations (NPOs) have been supporting the African neuroscience community to build state-of-the-art scientific capacity for sustainable education and research. Some of these contributions have included: the establishment of training courses and workshops to introduce African scientists to powerful-yet-cost-effective experimental model systems; research infrastructural support and assistance to establish research institutes. Other contributions have come in the form of the promotion of scientific networking, public engagement and advocacy for improved neuroscience funding. Here, we discuss the contributions of NPOs to the development of neuroscience in Africa.

Keywords: Africa, Neuroscience, Higher education, Non-profit organization, Scientific capacity, Research funding.

Antidotal effects of *Nigella sativa* oil on acetaminophen induced neurotoxicity in the cerebellum of Wistar rats (*Rattus norvegicus*)

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Abstract
The use of naturally existing antioxidants as free radical scavengers has the potential to prevent, delay, or ameliorate many neurologic disorders. *Nigella sativa* (NS) has been used for thousands of years. It has been shown to possess antioxidant properties. The study aimed studying the possible antidotal effects of *NS* oil against Acetaminophen induced Neurotoxicity in the Cerebellum of Wistar rats. A total of 60 adult male Wistar rats were placed in six groups, A-F of 10 animals each. Group A served as the control, group B were treated with 28 ml/kg of *NS* oil daily for 14 days. Group C were treated with single dose of 2 g/kg Acetaminophen. Group D were treated with *NS* oil at 28 ml/kg daily for a period of 7 days orally before subsequent oral administration of Acetaminophen. Group E animals were administered Acetaminophen orally, followed by subsequent oral administration of *NS* oil for a period of 10 days. Group F animals were given *NS* oil at 28ml/kg weekly for 3 weeks. Haematoxylin and Cresyl fast violet staining were done. The results showed that acetaminophen indeed had a toxic effect on the cerebellum of the rats manifesting as degeneration of neuronal cell bodies. *NS* oil at 28ml/kg daily dose resulted in increased density of neuronal cell bodies. Both Acetaminophen treated and *NS* oil treated rats had reduced Glutathione significantly from groups B to F. In conclusion, it can be said that both *NS* and Acetaminophen induced oxidative stress in rat's brain. *NS* was unable to prevent neuronal cell bodies from degenerating after an Acetaminophen overdose.

Keywords: *Nigella sativa*, Acetaminophen, Glutathione, Cerebellum

Neuronal network models of epileptogenesis: a review

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Abstract
Epileptogenesis is the process that, following some trigger, transforms a normal brain to one that produces recurrent unprovoked seizures. The mechanisms involved are not fully understood but their study is of high importance as it may provide an opportunity for the primary prevention of epilepsy. In the search for the mechanisms that best explain the epileptogenic process, there is a growing body of evidence suggesting that the epilepsies are network level disorders. In this review, we briefly describe the concept of neuronal networks and highlight two methods used to analyze such networks. The first method, graph theory, is used to describe general characteristics of a network to facilitate comparison between normal and abnormal networks. The second, dynamic causal modelling is useful in the analysis of the pathways of seizure spread. Finally, the recurrent seizures, the end result of the epileptogenic process, are better understood as not simply due to molecular or cellular derangements, but rather a result of abnormalities in the neuronal circuitry. In conclusion, the neuronal networks models of epileptogenesis attempt to generate a parsimonious explanation for the varied and disparate phenomena associated with epilepsy and seizures and to understand epilepsy beyond merely recurrent seizures but as a dynamic property of physiologic neuronal systems. The model offers a hope for better and more refined treatments for epilepsy.

Key words: Epileptogenesis, neuronal networks, graph theory, causal modelling.

Effects of dichlorvos inhalation on the learning, memory and brain weights of adult Wistar rats

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Abstract

Dichlorvos is a volatile organophosphate that forms the active ingredient of locally formulated but popular insecticide and pesticide known as *Otapiapia* or *Madarar piapia*. It is an anti-acetylcholinestrase that binds irreversibly to acetylcholinesterase enzyme thereby leading to its inhibition. It is cheap in production, highly efficient and easily accessible thereby making it one of the most commonly abuse insecticide and pesticide. Although many studies were conducted on the hazardous effects of this chemical, less attention was paid to the learning, memory and brain weight. The study aims at determining the effect of dichlorvos inhalation on the learning, memory and brain weights in adult Wistar rats. Twenty five apparently healthy adult Wistar rats consisting of both sexes were randomly selected, divided into five groups and experimented for 28 days. Three groups were exposed to 11.25mg/l, 7.5 mg/l and 3.75 mg/l doses of dichlorvos in ethanol solution, whereas the two other groups were exposed to ambient air and ethanol solution (96% purity) as positive and negative controls respectively. After the experiments, the animals were made to undergo object recognition test (ORT) and their brains tissues were collected and weighed. Two samples *t* – tests and one-way ANOVA were carried out to determine the difference in the mean recognition time and brain weights respectively, in all the groups using Minitab (version 16) statistical package. Although variable degrees in mean recognition time and brain weights were obtained following dichlorvos inhalation, no statistically significant difference in the mean recognition time as well as brain weights were noticed in all the experimental groups. In conclusion, inhalation of dichlorvos during the test period did not have any effect on the mean recognition time or brain weights of adult Wistar rats.

Key words: Dichlorvos, Learning, Memory, Brain weights

Effect of insulin on learning and memory in mice during morris water maze and barnes maze tasks

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Abstract

Insulin is currently being reported to have many physiological effects, including effect on cognition. This study evaluated the effect of insulin on learning and memory in mice. Twelve mice each used for the Morris water maze and Barnes maze tests were divided into two and injected daily for seven days with insulin (10 I.U./kg/day) or water. Testing was done on the 5th, 6th and 7th days of treatment. Repeated measures ANOVA and Bonferroni *post-hoc* tests using SPSS statistical package compared the means. In Morris water maze test, there was reduction in latencies (between day 1 and 2) for both insulin (57.92 ± 5.5and 42.50 ± 5.5 seconds) and control (55.08 ± 5.9 and 30.79 ± 4.9 seconds) groups, while the difference in latencies between the two groups was not significant. There was no difference in time spent per quadrant, and in the number of platform crossings between the two groups in the probe trial (day 3). In Barnes maze test, primary latency remained the same between the first and second day; while the number of primary head searches significantly reduced between day 1 and day 2 for both the insulin (11.45 ± 1.9 and 4.17 ± 0.8) and control (19.95 ± 4.5 and 10.00 ± 3.2) groups. There was no difference in primary latency and primary head search between the two groups. There was also no difference between the groups in the time spent by animals per quadrant, number of head searches per quadrant and number of head dips in correct hole during the probe trial (day 3). It is concluded that insulin treatment did not impair or improve the ability of the mice to learn, or their long-term memory of the learned task.

Key words: Insulin, learning and memory, Morris water maze, Barnes maze, mice

Possible effect of monosodium glutamate on the performance of swiss albino mice in some learning and memory neurobehavioral paradigms

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Abstract
Monosodium glutamate (MSG) is the sodium salt of glutamic acid, it is a common food flavour enhancer. This study was aimed at evaluating the possible effect of MSG on learning and memory neurobehavioral paradigms of elevated plus maze for memory (EPM), Morris water maze (MWM), Y maze and novel object recognition test (NORT). 80 mice (20 for each behavioural paradigm) were used for the study. The animals were randomly divided into 4 groups of 5 mice each (n=5), control (10 mg/ml normal saline (10 ml/kg), groups II, III and IV were orally administered 1.5, 3 and 6 mg/kg of MSG respectively. Results obtained showed no statistical difference on acquisition and retention in EPM for memory between control and MSG-treated groups. Similarly, no difference was observed for latency to locate platform and frequency of platform crossings in MWM between control and MSG-treated groups. Further, there was no statisticaldifference in the time spent in arm C and percentage spontaneous alteration between the control group and the MSG-treated groups. Also, no difference was observed in time spent with object C between MSG-treated groups and control in NORT. However, statistical difference was observed between MSG at a dose of 3mg/kg and control group in number of visit to object C in NORT. This study suggests that MSG did not affect cognition in mice at the doses administered.

Keywords: Monosodium glutamate, Cognition, Elevated plus maze, Morris water maze, Y maze