# Abstracts Presented at the 22nd Conference of the Neuroscience Society of Nigeria

## S1.01: Ameliorative effects of ethanol extract of *Lavandula angustifolia* on cyanide-induced hippocampal toxicity in male Wistar rats

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**Abstract**
The hippocampus, essential for memory and learning, is highly susceptible to oxidative damage. Cyanide, a known neurotoxin, disrupts cellular respiration and induces oxidative stress, particularly affecting high-metabolic regions like the hippocampus. Given lavender's established antioxidant, anti-inflammatory, and neuroprotective properties, this study investigates the curative potential of ethanol extract of *Lavandula angustifolia* (English lavender) against cyanide-induced hippocampal toxicity in male Wistar rats. A total of 25 adult male Wistar rats were randomly divided into five groups, A-E. Group A (negative control) received normal saline, Group B received 6 mg/kg of cyanide extract from cassava roots orally for 14 days, and thereafter groups C, D, and E received 100 mg/kg, 150 mg/kg, and 200 mg/kg of body weight, respectively, of ethanol extract of *L. angustifolia* for another 14 days. On day 29, blood samples were collected via retro-orbital puncture, stored in plain tubes, and analysed for oxidative stress markers malondialdehyde (MDA), glutathione (GSH), and catalase (CAT) using ELISA kits. The rats were sacrificed by chloroform inhalation, the hippocampus was dissected and fixed in 10% formal saline and processed for routine haematoxylin and eosin. Group B showed significant elevation of MDA levels and reduction of GSH and CAT, indicating oxidative stress. Groups C, D, and E, particularly at higher doses, showed normal levels of these biomarkers, suggesting reduced oxidative stress. Histologically, lavender-treated groups preserved hippocampal architecture compared to the cyanide-only group, which displayed spongiform changes and neuronal damage. In conclusion, the ethanol extract of *Lavandula angustifolia* conferred a dose-dependent ameliorative effect against cyanide-induced oxidative stress and histological damage in the hippocampus.

**Keywords**: English lavender, Cyanide, Oxidative stress, Hippocampus

## S1.02: Depression, anxiety, and associated factors among individuals receiving antiretroviral therapy: A mixed-methods study in selected tertiary hospitals in Anambra State

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**Abstract**
As of 2024, Nigeria continues to face a significant public health challenge with HIV, recording an estimated prevalence rate of 1.4% among adults aged 15 to 64 years. This translates to approximately 2 million people currently living with HIV. Despite progress in HIV care, stigma and discrimination still hinder national efforts. Mental health issues like depression and anxiety are common among PLWHA, affecting treatment adherence and outcomes. This study assessed levels of depression and anxiety and their associated factors as part of comprehensive care. Over six months, the study was conducted in tertiary hospitals in Anambra State, involving HIV-positive individuals aged 17 and above on antiretroviral therapy. Using a mixed-methods approach, data were collected through the PHQ-9 (for depression) and GAD-7 (for anxiety), alongside qualitative interviews. Analysis involved descriptive statistics and chi-square tests using SPSS Version 26 ($P<0.05$). Among 110 participants (71.1% female), 78.9% reported low depression and 70.0% reported low anxiety levels. Depression showed significant associations with marital status, educational level, and duration since diagnosis. Anxiety was significantly linked to age and education. Gender and occupational status were not significantly associated with either con-

dition. These findings suggest that while depression and anxiety levels were generally low, sociodemographic factors still play a role in mental health among PLWHA. Addressing these factors is vital in designing effective mental health interventions within HIV care.

**S1.03: Polyherbal formulation ameliorates rotenone-induced motor and non-motor dysfunctions in rats by modulating glutamine-glutamate metabolism, tyrosine hydroxylase and DAT expression**

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**Abstract**
Glutamatergic neurotransmission is involved in many cognitive, sensory and autonomic activities. Impaired glutamate homeostasis has important neuropathological consequences and has been associated with Parkinson's disease. Thus, keeping extracellular glutamate levels in a physiological range is crucial to ensure proper neural transmission and synaptic plasticity. This study investigated the effect of a polyherbal formulation (PHF) comprising bitter kola seeds, hog plum leaves, red onions and Aidan fruits on rotenone-induced motor and non-motor dysfunction of Parkinson's disease (PD) in male Wistar rats. Male Wistar rats were randomly divided into six groups (n=10): namely, Group 1, control; Group 2: rotenone (2 mg/kg); Groups 3-5: 2 mg/kg rotenone + 50 or 100 mg/kg PHF or 10 mg/kg L-DOPA, respectively; and Group 6: 100 mg/kg PHF only. PD was induced by subcutaneous administration of rotenone (2 mg/kg) for 30 days, and rats were then administered PHF orally for 3 weeks before sacrifice. Motor function was evaluated using behavioural tests, after which the rats were sacrificed. Biochemical analyses were performed to evaluate markers of excitotoxicity, including the activities of glutamine synthetase, glutaminase and glutamate dehydrogenase, as well as dopamine levels. Immunohistochemical analyses of dopamine active transporter (DAT) and tyrosine hydroxylase expression in the striatum were also conducted. The results showed that rotenone administration elicited significant motor deficits and disrupted glutamine-glutamate and dopamine metabolism. These alterations were attenuated by treatment with PHF. Immunohistochemical and histological evaluations revealed that rotenone caused dopaminergic neuronal loss and histoarchitectural distortions in the striatum, both of which were substantially ameliorated by PHF treatment. In conclusion, this study revealed that the polyherbal formulation exhibits therapeutic potential in ameliorating the symptoms and pathological changes associated with rotenone-induced Parkinson's disease in rats.

**Keywords:** Parkinson's disease, polyherbal formulation, neuroprotection

**S1.04: Ethanol leaf extract of *Aloe barbadensis* (Aloe vera) mitigates mercury-induced Alzheimer's-like symptoms in the basal ganglia of Albino Wistar rats**

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**Abstract**
Mercury exposure has been linked with numerous neurological disorders that frequently trigger Alzheimer's disease through oxidative stress processes. This study aims to determine the ameliorative potential of *Aloe barbadensis* on mercury-induced Alzheimer's-like symptoms in the basal ganglia of Wistar rats. Thirty-five Wistar rats with an average weight of 150 g were randomly divided into five groups designated A-E with seven rats per group. Group A served as the control and did not receive any treatment; group B received 5 mg/kg mercury chloride only for three weeks; group C received 500 mg/kg of ethanol extract of Aloe barbadensis for 3 weeks; and groups D and E were administered 5 mg/kg mercury chloride for 3 weeks, followed by administration of 250 mg/kg and 500 mg/kg ethanol extract of *Aloe barbadensis*, respectively, for 3 weeks. All administrations were via oral gavage. The anxiety index and recognition memory were evaluated using open field and novel object recognition tests. Brain tissue obtained was homogenised for estimation of superoxide dismutase (SOD), malondialdehyde (MDA), acetylcholinesterase (AChE) and glutamate levels and also processed for routine haematoxylin and eosin and silver Bielschowsky staining. Results of the neurobehavioural tests showed a significant ($p<0.05$) increase in the anxiety index and a significant decrease ($p<0.05$) in the recognition index in group B. There was a significant ($p<0.05$) decrease

in SOD, AChE, and glutamate levels in group B compared with groups A, C, D, and E, while there was a significant increase ($p<0.05$) in MDA levels in group B. A histological study of the basal ganglia showed pyknotic nuclei in group B, while silver Bielschowsky stain revealed amyloid plaque deposition in group B. These results revealed that mercury chloride caused oxidative stress, anxiety, reduced AChE and glutamate levels, pyknosis, and the deposition of amyloid plaques on the basal ganglia. The *Aloe barbadensis* ethanol extract mitigated these effects and may be useful in the management of Alzheimer's symptoms.

**Keywords**: Mercury chloride, oxidative stress, amyloid plaques, Alzheimer's disease, *Aloe barbadensis*

### S1.05: Modulation of neuroinflammatory and cognitive dysfunction pathways by *Stachytarpheta jamaicensis* in diabetic rat brains

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**Abstract**
Diabetes-induced neuroinflammation and cognitive decline, particularly within the hippocampal CA3c region, are driven by chronic hyperglycemia and oxidative stress. *Stachytarpheta jamaicensis* (SJ), a medicinal plant with known antioxidant and anti-inflammatory properties, was investigated for its neuroprotective potential. Adult male Wistar rats (n = 25) were assigned to five groups: healthy control, diabetic control, low- and high-dose SJ, and ibuprofen-treated. After streptozotocin (STZ)-induced diabetes, treatments were administered orally for two weeks. Neurobehavioural performance was assessed using open field and novel object recognition tests. TNF-α and IL-6 levels were quantified from brain homogenates using ELISA, and hippocampal CA3c subregion histology was evaluated via H\&E staining. SJ-treated groups, especially at high doses, showed improved cognitive performance, reduced cytokine levels, and preserved neuronal integrity. Effects were comparable to ibuprofen. This study provides compelling evidence of SJ's therapeutic potential in mitigating diabetic encephalopathy and supports further exploration of its bioactive constituents.

**Keywords**: *Stachytarpheta jamaicensis*, diabetes, neuroinflammation, hippocampus

### S1.06: The emerging roles of the RP1-111D6.3 gene in the converging pathology of Alzheimer's disease and diabetes

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**Abstract**
Alzheimer's disease (AD) and type 2 diabetes mellitus (T2DM) are progressive, multifactorial disorders with increasing global prevalence. Emerging clinical and molecular evidence suggests that T2DM significantly contributes to the onset and progression of AD through overlapping pathophysiological mechanisms, including chronic insulin resistance, mitochondrial dysfunction, oxidative stress, neuroinflammation, and aberrant protein aggregation. These shared disturbances ultimately lead to synaptic failure, cognitive impairment, and neuronal loss. Despite these observed clinical associations, the genetic and molecular mediators underpinning this convergence remain insufficiently characterised. Recent systems-level genomic analyses have identified RP1-111D6.3 as a candidate gene directly associated with both AD and T2DM. RP1-111D6.3 is embedded in gene networks related to key signalling pathways such as Phosphoinositide 3-Kinase/AKT serine/threonine kinase (P13K/AKT), Mitogen-Activated Protein Kinase (MAPK), and CREB-binding protein (CREBBP) pathways known to regulate neuronal survival, synaptic plasticity, glucose metabolism, and insulin signalling. Disruption of these pathways can lead to tau hyperphosphorylation, amyloid accumulation, and neuronal energy failure, a hallmark of AD that is exacerbated by diabetic pathology. Moreover, RP1-111D6.3 may play a role in brain insulin resistance, lipid dysregulation, and autophagy impairment, which is a critical process linking peripheral metabolic disease to central nervous system degeneration. By modulating these interconnected pathways, RP1-111D6.3 represents a promising target for understanding the molecular overlap between diabetes and AD. Clarifying the role of RP1-111D6.3 could provide novel insights into the patho-

genesis of both disorders and guide future therapeutic interventions aimed at mitigating cognitive decline in individuals with metabolic dysfunction.

**Keywords:** RP1-111D6.3, Alzheimer's disease, type 2 diabetes, insulin resistance, and neurodegeneration.

### S1.07: Neuroprotective effect of turmeric on calabash chalk-induced toxicity on mice hippocampus

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**Abstract**
Calabash chalk, a geophagic material commonly consumed in parts of Africa, contains neurotoxic heavy metals such as lead and arsenic, which are implicated in hippocampal damage and cognitive impairments. Turmeric (*Curcuma longa*), renowned for its antioxidant and anti-inflammatory properties, may offer neuroprotection against such toxicity. This study investigates the neuroprotective effects of turmeric on calabash chalk-induced hippocampal toxicity in mice. The aim is to evaluate the efficacy of turmeric in mitigating calabash chalk-induced neurotoxicity, focusing on hippocampal function, oxidative stress, and inflammation. Sixty albino mice (20-22 g) were divided into six groups: control, low-dose turmeric (300 mg/kg), high-dose turmeric (500 mg/kg), calabash chalk (400 mg/kg), and co-treatment groups (calabash chalk + turmeric). Neurobehavioural tests (open field, Y-maze, novel object recognition) assessed locomotion, anxiety, and memory. Biochemical analysis measured oxidative stress markers (MDA, GSH, SOD, and catalase) and inflammatory cytokines (TNF-α and IL-6). Hippocampal histology was examined using H&E staining. Statistical significance was determined via ANOVA ($p \leq 0.05$). Calabash chalk exposure increased anxiety-like behaviour, impaired memory, and elevated oxidative stress and inflammation. Histological analysis revealed hippocampal pyramidal cell degeneration. Turmeric co-treatment, particularly at high doses, restored locomotor activity, reduced oxidative stress, lowered pro-inflammatory cytokines and ameliorated hippocampal damage. Turmeric exhibits significant neuroprotective effects against calabash chalk-induced hippocampal toxicity, likely via antioxidant and anti-inflammatory mechanisms. These findings highlight its potential as a therapeutic agent for neurotoxicity, warranting further clinical exploration.

**Keywords:** Calabash chalk, turmeric, neuroprotection, hippocampus, mice model

### S1.08: Translational insights into neuroprotection: Whole *Saigon cinnamon* as a therapeutic candidate for post-stroke cognitive dysfunction

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**Abstract**
Motivated by nature's pharmacological potential, we investigated the neuroprotective effects of whole *Saigon cinnamon* in experimental models of stroke and cognitive decline. Using adult male Wistar rats, neurological insults resembling stroke were induced via daily intraperitoneal administration of pathophysiological agents for one week. The animals were then treated with *Saigon cinnamon* extract (250 mg/kg orally) for 28 days across pretreatment, co-treatment, and post-treatment regimens to evaluate therapeutic windows. Behavioural assessments, including Barnes Maze, Novel Object Recognition, Object Location, Open Field, Sucrose Splash, Rotarod and Hot Plate tests, provided a comprehensive evaluation of cognitive and emotional outcomes following brain injury and treatment. Biochemical analyses measured oxidative stress markers (SOD, catalase, GSH, MDA, NO), inflammatory cytokines (TNF-α, IL-6, IL-10, NF-κB), neurotrophic/apoptotic markers (BDNF, Caspase 3, S100B), and neurotransmitter regulation (acetylcholinesterase, serotonin). Histological studies (haematoxylin & eosin, Golgi-Cox staining) assessed neuronal survival and dendritic integrity, particularly within the hippocampus. Results demonstrated significant cognitive and behavioural improvements in cinnamon-treated rats, alongside reduced neuroinflammation and oxidative damage, enhanced antioxidant defences, and preservation of neuronal architecture. The consistent

benefits across treatment windows highlight the robust therapeutic potential of whole *Saigon cinnamon*. These findings provide critical preclinical evidence supporting the multi-target neuroprotective effects of *Saigon cinnamon* and underscore its promise for translational application in mitigating stroke-induced cognitive impairment. This study lays the groundwork for future clinical exploration of botanical therapies aimed at neurodegenerative disease and stroke recovery.

**Keywords:** *Saigon cinnamon*, Neuroprotection, Stroke, Cognitive impairment, Oxidative stress, Neuroinflammation

### S1.09: Modulating brain metabolism and neurodegeneration: Therapeutic potential of whole alligator pepper in Alzheimer's-like neuropathology

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**Abstract**
Alzheimer's disease (AD) is a progressive neurodegenerative disorder marked by cognitive impairment, oxidative stress, and chronic neuroinflammation. With growing interest in nutraceuticals for neurotherapeutic intervention, this study investigated the neuroprotective and therapeutic effects of *Aframomum melegueta* (alligator pepper) in a scopolamine-induced Alzheimer's-like model in Wistar rats. Adult rats were divided into six groups, including control, scopolamine-only, and treatment groups administered whole *A. melegueta* orally at 50 mg/kg and 100 mg/kg. The whole *A. melegueta* was given in both pretreatment and post-treatment paradigms to evaluate its protective and therapeutic efficacy, respectively. Behavioural assessments included the Morris Water Maze, Y-Maze, Novel Object Recognition, Object Location Test, and Open Field Test to evaluate learning, memory, and anxiety-related behaviours. Biochemical analyses of brain homogenates revealed that *A. melegueta* significantly reduced oxidative stress by elevating antioxidant enzymes—superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH)—while decreasing malondialdehyde (MDA) levels. In addition, the extract effectively downregulated proinflammatory cytokines tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), indicating anti-inflammatory activity. Behavioural outcomes showed marked improvements in cognitive performance and spatial memory, with greater efficacy observed at the 100 mg/kg dose. Toxicological evaluations confirmed the safety of both doses, with no significant adverse effects on liver or kidney function. These findings suggest that *Aframomum melegueta* offers dual protective and therapeutic benefits in Alzheimer's-like neuropathology, likely mediated through antioxidant and anti-inflammatory pathways. The study supports its potential as a safe, multi-target nutraceutical for managing neurodegenerative diseases.

**Keywords:** *Aframomum melegueta*, Alligator pepper Alzheimer's model, Oxidative stress, TNF-α, IL-6

### S1.010: Effects of methanol extract of *Olax subscorpioidea* Oliv. (Olacaceae) in carrageenan-induced foot pad oedema in chicks and brewer's yeast-induced pyrexia in Wistar rats

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**Abstract**
*Olax subscorpioidea* leaf is used traditionally for the treatment of venereal diseases, rheumatoid arthritis, toothache, dermatitis, fever and pain management. The methanol extract of the leaf has not been scientifically validated. The objective of this research was to evaluate the extract for possible anti-inflammatory activity in a carrageenan-induced footpad oedema model, as well as its antipyretic activity in brewer's yeast-induced pyrexia in rats, and to characterise the phytochemical composition of the extract. Foot volume in carrageenan-induced foot oedema was measured using a plethysmometer before and after treatment with 25, 50, 100 and 200 mg/kg of the extract and 1 mg/kg dexamethasone as the standard drug. The extract showed a decrease ($p<0.001$) in oedema with a maximal inhibition of 73.54% at a dose of 50 mg/kg, which was comparable to that of dexamethasone (67.78%). Also, methanol extract of *Olax subscorpioidea* caused a significant reduction

(p<0.05) in yeast-induced pyrexia in rats. Alkaloids, flavonoids, tannins, steroids and cardiac glycosides, found in the phytochemical analysis, could have been responsible for the activity of the extract. The GC-MS method was used to screen the compounds present in the methanol extract. Identification of sixty bioactive phytochemical compounds in the methanol extract of *Olax subscorpioidea* (MEOS) was based on the peak area, retention time, molecular weight, molecular formula, and pharmacological actions with that of literature and by interpretation of mass spectra. In GC-MS analysis, major and minor compounds were present; among the major phytochemical constituents screened were cis-vaccenic acid, n-hexadecanoic acid, 9,12,15-octadecatrienoic acid, methyl ester (Z,Z,Z), 1-amino-2,6-dimethylpiperidine, and hexadecanoic acid, methyl ester. Some of the identified compounds possess biological properties such as anti-inflammatory, antifungal, antibacterial, anti-tumour, and antioxidant. Hence, the methanol extract of *Olax subscorpioidea* contains phytocompounds with useful biological activities and thus exhibits anti-inflammatory and antipyretic activities, thereby confirming the use of the plant for inflammation in traditional medicine.

**Keywords:** GC-MS, Inflammation, Fever, Pain, Carrageenan, Chicks

### S1.011: Efficacy of eugenol in *N,N'-dimethyl-4,4'-bipyridinium dichloride* dysregulation of $Na^+/K^+/Ca^{2+}$ channel, cytochrome C, and behavioural deficits in Wistar rats

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**Abstract**
Ion channel disruption causes mitochondrial aberrations, altering the level of cytochrome C (Cyt-C). To evaluate the efficacy of eugenol in the $Na^+/K^+/Ca^{2+}$ channel, Cyt-C dysregulation, and behavioural deficit. Forty male Wistar rats were acclimatised for two weeks and assigned to five groups of 8 rats per group. Group A served as the control group. Groups B, C, D, and E received 10 mg/kg of paraquat (PQ); thereafter, C, D, and E were treated with 200, 400, and 600 mg/kg of eugenol, respectively, while Group B was not treated with eugenol and served as the PQ group. PQ and eugenol were administered orally for 28 and 14 days, respectively. Social-interactive and depression-like behaviours were studied using a sociability chamber and a tail suspension box. The interstitial level of Na+/K+ increased while $Ca^{2+}$ decreased significantly in group B, which was reversed by eugenol treatment. Cyt-C, caspase cleavage, and full-length cytokeratin-18 levels significantly increased in the untreated group and were reduced by eugenol ($p < 0.05$). Social interaction and mobility were improved by eugenol treatment. Dopamine and gamma-glutamate transferase levels were increased and decreased, respectively, by eugenol ($p < 0.05$). Microscopically, there was a dopaminergic neuronal loss, formation of Lewy bodies, and pyknosis in the substantia nigra of group B. Eugenol alleviated the mitochondrial physiological imbalance by regulating the nakca2 channel and increasing antioxidant levels.

**Keywords:** Cytochrome C; Dysregulation, Eugenol; $Na^+/K^+/Ca^{2+}$; Paraquat

### S1.012: Natural intervention for cognitive decline: unveiling the multi-target neurotherapeutic potential of *Saigon cinnamon* in Alzheimer's disease

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**Abstract**
Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterised by memory impairment, cholinergic dysfunction, oxidative stress, and neuroinflammation. This study investigates the multi-target neurotherapeutic efficacy of *Cinnamomum loureiroi* (*Saigon cinnamon*) in a scopolamine-induced AD-like model in Wistar rats, with a focus on cognitive, biochemical, and histological outcomes. Following acute toxicity evaluation, a safe oral dose of 200 mg/kg *Saigon cinnamon* extract was administered daily for 28 days. Alzheimer's-like neuropathology was induced via intrahippocampal scopolamine (5 µg/µL). Behavioural assessments using the Morris water maze (MWM) and Y-maze showed significant improvement in spatial learning and working memory among treated rats. Biochemical assays revealed increased antioxidant activity,

including superoxide dismutase (SOD), reduced glutathione (GSH), and catalase, alongside decreased oxidative stress markers such as malondialdehyde (MDA) and nitric oxide (NO). *Saigon cinnamon* downregulated pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), suppressed nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) activation, and elevated anti-inflammatory interleukin-10 (IL-10) and serotonin levels, suggesting modulation of inflammatory and mood-related pathways. Acetylcholinesterase (AChE) inhibition indicated improved cholinergic transmission. Histopathological analysis confirmed neuronal preservation and enhanced dendritic arborisation in the hippocampus. These findings demonstrate that *Saigon cinnamon* exerts a multi-target neuroprotective effect through antioxidant, anti-inflammatory, cholinergic, and serotonergic mechanisms, supporting its potential as a natural therapeutic intervention for cognitive decline in Alzheimer's disease.

**Keywords:** Alzheimer's disease, *Saigon cinnamon*, *Cinnamomum loureiroi*, Cognitive decline, neuroinflammation, Cholinergic transmission

### S1.013: Cerebellar histology, microglia status, and nuclear areas expressing alpha-synuclein and parvalbumin in rats exposed to a high-fat diet and bisphenol-A - roles of *Vernonia ambigua*

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**Abstract**
Research on the cerebellum has focused on its functions, susceptibility to individual toxic chemicals, and responses to natural products aimed at reducing observed or anticipated abnormalities. However, there is still a lack of information regarding the combined effects of toxic substances on cerebellar histoarchitecture, immune responses, and proteins that are related to motor and synaptic coordination. This study examined the effects of ethanol leaf extract of *Vernonia ambigua* (ELEVA) on cerebellar histology, expressions of microglia marker (CEMM), alpha-synuclein (CEA), and parvalbumin (CEP) in male Wistar rats exposed to a high-fat diet (HFD) and bisphenol-A (BPA). A total of 42 rats were divided into six groups. Groups 1, 2, 3, and 4 received feed and water, 12.6 mg/kg body weight (b.w.) of BPA only, HFD only, and HFD with 12.6 mg/kg b.w. of BPA, respectively, for 84 days. Groups 5 and 6 received 50 mg/kg and 100 mg/kg b.w. of VA, respectively, for 28 days following 84 days of HFD and BPA exposure. On days 85 and 113, the brains were excised and fixed in 10% neutral buffered formalin for routine histological and immunohistochemical analyses. The significance level was set at $P < 0.05$. Group 4 exhibited enlarged and vacuolated glial cells and distorted Purkinje cell bodies (PCBs) with spiny projections from the cell membrane. Group 6 showed more PCBs appearing normal, contrary to the findings in group 4. This study observed a significant decrease in CEA in group 5 and CEP in groups 5 and 6 compared to groups 1 and 4. ELEVA showed a concentration-dependent ability to reduce the distortion of PCBs and microglial inactivity. Since alpha-synuclein and parvalbumin are vital for maintaining synaptic integrity and firing rates of Purkinje cells, respectively, ELEVA may positively influence neuronal functions by reducing CEA and CEP.

**Keywords**: Purkinje Cells, Spiny projections, firing rates, synaptic integrity

### S1.014: Ameliorative effect of papain on LPS-induced neuroinflammation and memory loss

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**Abstract**
Neuroinflammation and memory loss have been established to be triggered by lipopolysaccharide (LPS) and are implicated in neurodegenerative diseases. Papain, characterised from *Carica papaya,* is an isolated cysteine protease (proteolytic enzyme) that exhibits antioxidant and anti-inflammatory properties. Its ameliorative potential in LPS-induced neuroinflammation and memory loss remains unexplored. This study explored the potential of papain in mitigating LPS-induced neuroinflammation and memory loss in BALB/c mice. Forty-two (42) male BALB/c mice (30±2 g) were given a coadministration of intraperitoneal injection of 1 mg/kg of LPS oral treatment with papain at three doses (50 mg, 100 mg and 200 mg/kg b.w.) for 7 days. During treatment, spatial cognition and working memory were evaluated by the Morris water maze, NOR and Y-maze behavioural tests. After treatment, animals were euthanised and sacrificed, serum was obtained from whole blood, and the brain tissues were excised. The serum collected was used for biochemical anal-

yses of antioxidant enzymes, SOD, catalase and GSH, while the hippocampal and prefrontal tissue homogenates were assayed for inflammatory markers and acetylcholinesterase activities. The remaining prefrontal cortex and hippocampal tissues were fixed in formo-saline for histology and immunofluorescence procedures. Behavioural analyses showed a reversal of working memory in the mice exposed to LPS. The biochemical estimations revealed that lipid peroxidation with increased MDA was reversed significantly following treatment with [50 mg/kg and 100 mg/kg] of papain, while serum antioxidant levels of GSH and catalase were restored to near normal. Likewise, LPS injection significantly altered IL-1β and IL-4 levels, increased reactive microglia expression (P2RY12) and decreased AChE activity in the prefrontal cortex and hippocampus. However, these changes were ameliorated following a treatment with papain. This study concludes that papain significantly ameliorates the effects of LPS-induced oxidative stress, neuroinflammation and memory loss through an increase in levels of antioxidant markers and a decreased expression of P2RY12 both at the prefrontal cortex and hippocampus.

**Keywords:** Neuroinflammation, Lipopolysaccharide, Papain, Memory impairment

### S1.015: Modulation of oxidative stress and neurotransmitter dynamics by *Rauwolfia vomitoria* and clozapine in a psychosis model

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**Abstract**
Psychosis is closely linked to oxidative stress and dysregulation of neurotransmitter systems. This study aimed to investigate the effects of *Rauwolfia vomitoria* (*R. vomitoria*), clozapine, and their combined administration on oxidative stress and neurotransmitter profiles within a psychosis model. Adult rats were assigned to seven groups: control, untreated psychosis, *R. vomitoria* (low/high dose), clozapine, and their combinations. Post-treatment, levels of T-AOC, MDA, glutamate, GABA, and serotonin were measured. Data were analyzed using one-way ANOVA with post hoc comparisons. Group GP2 displayed a marked decrease in T-AOC relative to GP1 and all treated cohorts ($p < 0.0001$), underscoring the presence of oxidative stress in the untreated psychosis model. Treatment with *R. vomitoria*, clozapine, and their combinations resulted in significant restoration of T-AOC levels. Notably, GP7 demonstrated elevated glutamate levels compared to all other groups ($p < 0.05$), indicating heightened excitatory neurotransmission associated with high-dose combinatorial therapy. Moreover, GABA levels were significantly diminished in GP3 compared to GP2 and GP4 ($p < 0.05$), suggesting a dose-dependent modulation effect. Serotonin levels did not exhibit significant variation across the groups ($p = 0.497$). Untreated psychosis shows oxidative stress and glutamatergic hyperactivity. *R. vomitoria* and clozapine restore antioxidant capacity and modulate neurotransmitters. High-dose *R. vomitoria* improves GABA levels, while its combination with clozapine increases glutamate, highlighting the need for dose optimisation. These findings support the use of antioxidant strategies in psychosis management and the mechanism through which R. vomitoria and clozapine affect neurotransmission.

**Keywords**: Oxidative stress, Neurotransmitters, *Rauolfia vomitoria*, Clozapine, Psychosis

### S1.016: Mercury chloride-induced neurotransmitter alterations in the prefrontal cortex in Wistar rats: Modulatory role of *Ginkgo biloba* extract (EGB 761)

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**Abstract**
Mercuric chloride ($HgCl_2$) is a potent neurotoxic agent that disrupts neuronal function and neurotransmitter regulation. The prefrontal cortex (PFC) is critical for cognitive processes and behaviour. *Ginkgo biloba* extract (EGB761) has garnered attention for its potential therapeutic effects on cognitive function and neurotransmitter regulation. This study

evaluates the effects of EGB761 on $HgCl_2$-induced neurotoxicity on the PFC of Wistar rats. A total of 30 adult Wistar rats were randomly divided into 5 groups of 6 rats each. The control group, 5 mg/kg $HgCl_2$, 5 mg/kg $HgCl_2$ + 500 mg/kg A. A., 5 mg/kg $HgCl_2$ + 100 mg/kg EGB761, and 5 mg/kg $HgCl_2$ + 500 mg/kg EGB761. Administration was done orally for 21 days. The Y-maze was used to assess memory and learning; tissue homogenate was used to assess monoamine oxidase (MAO) and acetylcholinesterase (AChE). The result showed a significant ($p < 0.05$) decrease in %ALT in the $HgCl_2$-treated group. EGB761 showed a significant ($p < 0.05$) increase in working memory and a significant ($p < 0.05$) decrease in AchE and MAO activity levels in the EGB761-treated groups. EGB761 ameliorated cognitive deficit and changes in AchE and MAO activity. In conclusion, findings revealed that EGB761 has ameliorative properties against $HgCl_2$-induced insult on the PFC.

**Keywords**: *Ginkgo biloba*, Memory, Acetylcholine, Dopamine, Neurotoxicity

### S1.017: Evaluation of the ameliorative effect of ethanol extract of *Xylopia aethiopica* and vitamin E on the histomorphology of smokeless tobacco-exposed cerebral cortex of adult male Wistar rats

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**Abstract**
The World Health Organization (WHO) estimates that about 80% of the world's population rely on unconventional plant-based medicines as their primary medical intervention. Exposure to tobacco nicotine has been associated with alterations in the normal function of the brain and the nervous system. Nicotine has been shown to induce a dose-dependent increase in neuronal activity in a distributed system of brain regions. This study evaluates the ameliorative effect of ethanolic extract of *Xylopia aethiopica* and vitamin E on the histomorphology of the smokeless tobacco-exposed cerebral cortex of adult male Wistar rats. Twenty-five (25) adult male Wistar rats weighing between 180 and 200 g were randomised into five groups of five (5) rats each. Group A (negative control) was given feed and water only. Group B (Positive Control) received 4 mg/kg of smokeless tobacco. Group C was administered with 4 mg/kg of smokeless tobacco and 5 g of ethanolic extract of *Xylopia aethiopica*. Group D received 4 mg/kg of smokeless tobacco and 10 g of ethanolic extract of *Xylopia aethiopica*. Group E: 4 mg/kg of smokeless tobacco and 0.2 ml of vitamin E. The treatment lasted for 60 days, and subsequently the animals were sacrificed and their cerebrums harvested and processed using normal histological techniques and stained with H&E. The results demonstrated normal neural cells in the negative control group and extensive infiltration of inflammatory cells into the cerebrum with the positive control group. Group C indicated the presence of microglial cells in the cerebrum, and enhanced perfused tissues of the cerebrum in Group D, which is the same as in Group E. In conclusion, *Xylopia aethiopica* and vitamin E exhibited anti-inflammatory and ameliorative effects on the smokeless tobacco-exposed cerebrum.

**Keywords**: Smokeless tobacco, *Xylopia aethiopica*, Vitamin E, Cerebrum, Inflammatory cells

### S1.018: Anti-seizure effects of *Datura stramonium*: Impact on neurobehaviour, antioxidant enzymes, and inflammatory markers in the hippocampus and amygdala of adult female Wistar rats using an isoniazid-induced epileptic model

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**Abstract**
Epilepsy, marked by recurrent seizures and neurochemical imbalances, remains a treatment challenge, particularly in resource-limited settings. *Datura Stramonium* (*DS*), a plant rich in tropane alkaloids like atropine, scopolamine, and hyoscyamine, has

demonstrated potential pharmacological effects on the nervous system. However, its antiepileptic and neuroprotective properties are not well studied. *Aims and Objectives:* This study assessed the antiseizure potential of *DS* and its effects on neurobehavioural parameters, antioxidant enzyme activity, and inflammatory markers in an isoniazid-induced epileptic model in adult female Wistar rats. *Material and Methods: DS* extract was prepared using a standard extraction protocol. Group 1 was fed with water and standard feed only; group 2 was induced with 300 mg/kg of isoniazid only; group 3 was given low-dose *DS* extract (60 mg/kg) only; group 4 was given high-dose *DS* extract (120 mg/kg) only; group 5 (standard drug) was pre-treated with carbamazepine (200 mg/kg), and a seizure was induced with isoniazid (300 mg/kg); groups 6 and 7 were pre-treated with low (60 mg/kg) and high (120 mg/kg) doses of *DS* extract, respectively, and induced with isoniazid (300 mg/kg). Seizure traits were monitored while the threshold and latency were scored for anti-epileptic assessment. Oxidative stress levels were assessed using the superoxide dismutase, catalase, and malondialdehyde biomarkers, and TNF-α levels were also assessed for proinflammatory reactions. The histology of the hippocampus and amygdala, stained with haematoxylin and eosin, was studied. *Results:* The results revealed that low-dose *DS* (60 mg/kg) significantly reduced seizure latency, increased the seizure threshold, showed mild anxiolytic effects, improved cognitive abilities in behaviour, and also exhibited neuroprotective effects on the hippocampal and amygdala cytoarchitecture against isoniazid-induced epilepsy. *DS* extract also showed significant anti-inflammatory and antioxidant abilities. *Conclusion:* These findings suggest that *DS* possesses neuroprotective, antioxidative, and anti-inflammatory effects, as well as being a potent anti-epileptic agent.

**Keywords:** *Datura stramonium*, Isoniazid-induced Epilepsy, Anti-seizure, Neurotoxicity, Neuroprotection

### S1.019: Effect of selective alcoholic bitters on hippocampal histomorphology, oxidative enzymes and behaviour in rats

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**Abstract**
Alcohol bitters consumption is on the increase, especially among male adults, and may pose neurological disorders. This study evaluated the effect of selected alcoholic bitters on the hippocampal tissue, oxidative enzymes and behaviour in rats. Twenty (20) adult male Wistar rats were randomly divided into 4 groups of 5 animals each; Group 1 received water *ad libitum*. Group 2 received 0.66 mL/kg b.w. of action bitters. Group 3 received 0.75 mL/kg b.w. of Balomo bitters. Group 4 received 0.66 mL/kg b.w. of Odogwu bitters. Administration was done daily using an orogastric tube and lasted for 28 days. On the last day, a neurobehavioural test was carried out using the T-maze spontaneous alternation test. On the 29th day, the animals were anaesthetised with 50 mg/kg of ketamine injection intraperitoneally. Blood was aspirated via cardiac puncture, and serum was obtained for antioxidative assay. The brain tissues were perfused, harvested and fixed in 10% buffered formalin for histological processing. Results showed a significant increase ($p<0.05$) in the brain weight of the test groups compared to the control group and a significant decrease in the body weight of the test groups compared to the control. Histologically, changes that occurred were decreased granule cell numbers, nuclei degeneration and cellular vacuolations in the alcoholic bitters group compared to the control. SOD and CAT tests carried out showed a significant ($p<0.05$) decrease in the action bitters group compared to the control, respectively. MDA showed a significant ($p<0.05$) increase in the alcoholic bitters groups compared to the control, respectively. The reward alternation test showed increased alternation in the action bitters group compared to other treated groups and the control at $p < 0.05$, respectively. In conclusion, alcoholic bitters pose deteriorative effects on the histomorphology of the hippocampus and induce oxidative stress and memory deficit; however, aromatic bitters pose more detrimental effects.

**Keywords**: Alcoholic bitters, histology, oxidative damage, behaviour, hippocampus

### S1.020: Neurotoxic risk from urban dust: Evidence of cadmium and lead exposure in vehicle and motor park environments in Abuja, Nigeria

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**Abstract**
Environmental degradation in urban settings presents growing concerns for neurological health, particularly through the accumulation of potentially toxic elements (PTEs) in particulate matter. This study assessed the concentration, sources, and health risks of PTEs—including Fe, As, Cd, Zn, Cu, Mn, Pb, Cr, Co, and Ni—in in-vehicle and motor park dusts across Abuja, Nigeria. Using atomic absorption spectroscopy and positive matrix factorisation (PMF), five anthropogenic pollution sources were identified, such as vehicular emissions, brake and engine wear, and coal combustion. Notably, cadmium (Cd) levels surpassed geochemical background thresholds by over 1.6 times. Although aggregate health risk indices for both carcinogenic and noncarcinogenic effects fell within globally accepted limits, Cd posed a disproportionately high risk, particularly via ingestion. Children demonstrated heightened vulnerability, raising concern about neurodevelopmental exposure. Cd and Pb, two prevalent neurotoxic metals in the dust samples, are associated with oxidative stress, synaptic disruption, and neuronal apoptosis—all of which can impair cognitive function and behaviour, especially in developing brains. These findings align with the sub-theme “Neurotoxic Aspect of Environmental Degradation”, as they expose overlooked urban exposure routes affecting neurological health. Dust in vehicles and motor parks—ubiquitous in Nigerian cities—functions as a persistent reservoir of neurotoxicants. Bridging the gap between neuroscience research and environmental health practice in Nigeria requires integrated monitoring and urban policy interventions. Emphasising low-emission transportation, phasing out hazardous vehicle components, and redesigning urban spaces to minimize dust exposure are crucial steps forward. This study provides significant evidence regarding the neurotoxicological effects of urban pollution and emphasises the necessity for interdisciplinary collaboration to enhance public and neurological health.

**Keywords**: Environmental pollution, Neurotoxicity syndromes, Lead poisoning; Cadmium, Urban health

**S1.021: Mitigating roles of eugenol against lead-altered neurochemicals and motor activity and anxiety behaviours in Wistar rats**

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**Abstract**
Lead neurotoxicity has been associated with significant alterations in behaviour and neuronal integrity. This study evaluated the mitigating role of eugenol on lead-induced changes in anxiety-related behaviour and specific neurochemical markers in adult female Wistar rats. The thirty (30) adult Wistar rats used in this study were randomly assigned into five groups of six rats per group after a 14-day acclimatisation period. Group A was the control; Group B was the lead group and received 15 mg/kg of lead; Group C received 15 mg/kg of lead and 400 mg/kg of eugenol; Group D was given 15 mg/kg of lead and 600 mg/kg of eugenol; while Group E received 600 mg/kg of eugenol only accordingly. Anxiety-related behaviour and motor activities were assessed using the elevated plus maze (EPM) and the PhD Field Test, while biochemical analyses focused on dopamine, serotonin, alpha-synuclein levels, glutathione peroxidase (GPx), and malondialdehyde (MDA). Lead exposure resulted in a significant increase in time spent in the closed arms and a decrease in time spent in the open arms compared to the control ($p<0.05$), indicating heightened anxiety. The time spent in the closed arms decreased in the eugenol-treated groups while increasing in the open arms compared to the lead group ($p<0.05$). Additionally, dopamine and serotonin levels were significantly reduced, while alpha-synuclein expression was markedly elevated in the lead group compared to the control ($p<0.05$). Treatment with eugenol reversed these effects, as evidenced by improved performance in the EPM, restoration of dopamine levels, and reduction in alpha-synuclein expression ($p<0.05$). These findings suggest that eugenol possesses anxiolytic and neurocurative properties capable of mitigating lead-in behavioural and neurochemical disruptions.

**Keywords**: Alpha-synuclein, Anxiety, Eugenol, Lead, Neurochemical, Dopamine

**S1.022: Perinatal iron deficiency alters hemorheological indices and hippocampal acetylcholinesterase activity, impairing cognitive function in neonatal Wistar rats**

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**Abstract**
Perinatal iron deficiency (PID) is associated with neurodevelopmental impairments, but the underlying mechanisms remain unclear. This study investigated the effects of PID on cognitive function, hemorheological indices and hippocampal acetylcholinesterase (AChE) activity in neonatal Wistar rats. Pregnant rats were fed iron-deficient (IDD) or iron-sufficient (ISD) diets. Cognitive function was assessed using the novel object recognition (NOR) test at postnatal days (PND) 15, 22, and 30. Blood and hippocampal tissues were collected to measure hemorheological indices and AChE activity. PID significantly reduced cognitive performance, hemorheological indices, and hippocampal AChE activity at PND 15 compared to controls. These findings suggest that PID-induced cognitive deficits may be mediated by altered haemodynamics and cholinergic neurotransmission in the hippocampus.

**Keywords**: Perinatal, Iron deficiency, Hemorheological indices, Acetylcholinesterase

## S1.023: Haematologic effects of traumatic brain injury in adult Wistar rats

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**Abstract**
Traumatic brain injury occurs due to direct or indirect mechanical forces acting on the brain, leading to acute effects such as shearing injuries, contusions, and haematomas. Damage to the brain's vasculature and parenchyma contributes to the breakdown of the blood-brain barrier, which triggers secondary effects, including oedema, inflammation, and hyperexcitability. These changes initiate a cascade of complications that emerge hours to days after the initial injury. Delayed responses to TBI include neovascularisation, impaired cerebral blood flow, glial cell dysfunction, and cell degeneration. This study aimed to examine the haematologic effects of traumatic brain injury in adult Wistar rats. Forty adult Wistar rats were randomly assigned to five groups (A–E). Group A served as control. Groups B, C, D and E were induced with 50 g, 100 g, 150 g and 200 g weights, respectively, using the Marmarou method. Blood was collected a week before the trauma to establish a baseline and subsequently at 5 hours, 24 hours, 7 days, and 21 days post-trauma. The results of haematology showed a decrease in red blood cell count and its differentials across all traumatised groups at every interval compared to the control group. In contrast, platelet counts, white blood cells, and its components increased in all traumatised groups at every interval compared to the control group. These findings indicate that TBI significantly affects the overall blood count.

**Keywords:** Haematology, Traumatic brain injury.

## S1.024: Effect of beetroot juice extract on combined ingestion of coffee and *Garcinia kola* (bitter kola) on the cerebellum of adult male Wistar rats

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**Abstract**
The impact of various plant-based materials and beverages on brain function and structure has been a topic of scientific concern. This study investigated the effects of beetroot juice extract on the cerebellum following combined ingestion of coffee and *Garcinia kola*. The research assessed the neurobehavioural performance, body weight changes, and histological alterations in the cerebellum caused by the consumption of these substances. Fifty (50) adult male Wistar rats were randomly assigned to ten groups of 5 rats each. Group 1 served as the normal control; Groups 2 and 3 received low and high doses of coffee (362.3 mg/kg and 1086.8 mg/kg), respectively, while Groups 4 and 5 received low and high doses of *Garcinia kola* (273.9 mg/kg and 821.6 mg/kg), respectively. Group 6 received a combination of low doses of coffee and *Garcinia kola,* while Group 7 received a combination of high doses of

coffee and *Garcinia kola*. Group 8 received low-dose coffee + low-dose *G. kola* + beetroot juice (3.5 mL/kg), while Group 9 received high-dose coffee + high-dose *Garcinia kola* + beetroot juice (3.5 mL/kg). Group 10 received only beetroot juice (3.5 mL/kg). Results indicated significant weight loss in the group that received high doses of coffee and *Garcinia kola* combinations. Histological analysis revealed severe cerebellar damage, decreased Nissl substance staining and significant astrocyte proliferation in groups treated with high doses of coffee and *Garcinia kola*. The combination of both substances further worsened these neurotoxic effects, leading to the highest performance index in the beam walking test for the group receiving the highest doses. Conversely, the addition of beetroot juice extract showed promising neuroprotective effects. It is concluded that high doses of coffee and *Garcinia kola* have detrimental effects on cerebellar health and motor function, while beetroot juice possesses protective potential.

**Keywords:** Coffee, *Garcinia kola*, Beetroot juice, Cerebellum

### S1.025: Neuroprotective effect of ascorbic acid in LPS-induced neuroinflammation in male Swiss mice

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**Abstract**
Neuroinflammation has emerged as a central mechanism in the pathogenesis of numerous neurodegenerative diseases, often resulting in synaptic dysfunction, cognitive impairment, and neuronal loss. Lipopolysaccharide (LPS), a component of Gram-negative bacterial cell walls, is commonly employed in experimental models to induce neuroinflammation via microglial activation and pro-inflammatory cytokine release. Ascorbic acid (vitamin C), a naturally occurring neuroprotective compound, has demonstrated immunomodulatory effects that may attenuate such pathological responses. This study evaluated the neuroprotective potential of ascorbic acid in male Swiss albino mice subjected to LPS-induced neuroinflammation. Fifteen mice were randomly assigned to three groups (n = 5 each): Group A (Control) received normal saline, Group B (LPS) received a single intraperitoneal dose of LPS (2.5 mg/kg), while Group C (LPS + Ascorbic Acid) received LPS followed by oral administration of ascorbic acid (200 mg/kg) for five consecutive days. Cognitive performance was assessed using the Y-maze spontaneous alternation paradigm, while histological evaluations were performed on brain tissues using haematoxylin and eosin, cresyl fast violet, Feulgen, and silver staining techniques to assess general cytoarchitecture, Nissl substance distribution, nuclear morphology, and axonal integrity, respectively. The findings indicated that LPS administration significantly impaired cognitive performance and induced neuropathological changes, including neuronal shrinkage, chromatolysis, nuclear fragmentation, and axonal degeneration. Notably, ascorbic acid co-treatment resulted in a significant improvement in cognitive function, accompanied by preservation of neuronal morphology, restoration of Nissl granules, normalised nuclear architecture, and attenuated axonal damage. These findings suggest that ascorbic acid may help protect the brain from inflammation-related damage. This supports its potential usefulness as a complementary treatment for managing conditions involving neuroinflammation and neurodegeneration.

**Keywords:** Neuroinflammation, Ascorbic Acid, Lipopolysaccharide (LPS), Cognitive Function

### S1.026: Naringin attenuates hypothalamic oxidative stress, inflammation, and lead load in a dual-hit model of depression and lead-induced neurotoxicity

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**Abstract**
Depression is a multifactorial neuropsychiatric disorder with increasing global prevalence. Environmental toxins like lead are known to exacerbate neurobehavioural dysfunctions, yet the combined impact of chronic stress and lead-induced neurotoxicity remains underexplored. This study introduces a novel dual-hit model of depression combining chronic unpredictable mild stress (CUMS) and lead exposure and investigates the therapeutic potential of naringin, a citrus flavonoid with antioxidant properties. Forty-two adult male Wistar rats (170-190 g) were randomly assigned to seven groups (n=6): control (saline), CUMS + lead (120 mg/kg), CUMS + lead + naringin (2.5, 5, and 10 mg/kg), CUMS + lead + fluoxetine (10 mg/kg) + vitamin E (0.2 mL/kg), and naringin (5 mg/kg) alone. Treatments lasted 28 days. Behavioural tests (EPM, Y-Maze, Sucrose Preference Test, Tail Suspension Test) were conducted to assess depressive and anxiety-like behaviours. Biochemical assays of the hypothalamus were used to evaluate oxidative stress markers (MDA, SOD, GST) and inflammatory cytokines (IL-1β, TNF-α), while serum cortisol and glucose were assessed to determine systemic stress response. Lead accumulation in the hypothalamus was quantified via Inductively Coupled Plasma Mass Spectrometry (ICP-MS) to evaluate naringin's effect on lead bioaccumulation. Histological analysis of the hypothalamus and adrenal glands assessed cytoarchitectural integrity of the HPA axis. The CUMS+lead group showed significant behavioural deficits, increased MDA, IL-1β, TNF-α, cortisol, glucose, and hypothalamic lead levels, along with structural damage. The naringin-treated groups exhibited dose-dependent behavioural recovery, biochemical normalisation, reduced hypothalamic lead levels, and preserved histoarchitecture. These findings demonstrate that naringin exerts antidepressant, antioxidant, anti-inflammatory, and metal-chelating effects and can restore HPA axis integrity in a comorbid model of depression and lead neurotoxicity. This supports its potential as a natural therapeutic agent for stress-related neuropsychiatric disorders aggravated by environmental neurotoxic comorbidity.

**Keywords**: Naringin, Lead neurotoxicity, Depression, Chronic unpredictable mild stress, Hypothalamic–pituitary–adrenal axis.

**S1.027: Socio-emotional, anxiety and behavioural difficulties in children with autism spectrum disorder: Prevalence, patterns, and carer stress in south-eastern Nigeria.**

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**Abstract**
Children with Autism Spectrum Disorder (ASD) often exhibit anxiety, emotional problems, and behavioural difficulties; yet data on these manifestations in sub-Saharan African contexts remains scarce. This study examined the prevalence and patterns of these difficulties and how they affect parents and carers to highlight interventions that fit the local culture.
A cross-sectional study was conducted among 214 carers of children with ASD in Enugu and Anambra States. Standard tools used included the Anxiety Scale for Children (ASC), the Social Emotional and Behavioural Difficulties Scale (SEBDS), the Carer Coping Strategies Scale (CCSS), and the Carer Stress Scale (CSS). Descriptive statistics, Pearson's correlations, t-tests, and chi-square analyses were used. A majority of children (79.2%) showed anxiety symptoms, with 44.2% rated as "sometimes" anxious and 12.4% as "always" anxious. Difficulties in making friends (38.3%) and understanding others emotionally (44.4%) were widespread. Behavioural issues like aggression towards siblings (39.4%) and avoidance of social settings (29.4%) were common. Statistically significant correlations were found between children's difficulties and carer stress, for example, feeling frustrated ($r=0.372$, $p<0.001$) and feeling overwhelmed ($r=0.276$, $p<0.001$). Coping strategies such as seeking social support ($r = 0.221–0.333$) and relaxation strategies ($r = 0.124–0.303$) showed modest but consistent mitigating effects on carer distress. Chi-square analyses showed that boys aged 6–12 were significantly more likely to exhibit externalising behaviours ($\chi^2=65.056–90.093$, $p<0.001$). Children with autism in southeastern Nigeria often struggle with emotional and behavioural problems. This affects the mental health of their carers. Programs that encourage emotional support-

seeking and stress management for carers could offer protective benefits and enhance caregiving outcomes in under-resourced regions.

**Keywords**: Autism spectrum disorder, Anxiety, Socio-emotional challenges and behavioural difficulties, Carer stress and coping strategy, Nigeria

### S1.028: Neuroprotection by the sub-fraction of *Stephania dinklagei* roots on monosodium glutamate-induced excitotoxicity in mice: Modulation of oxido-nitrergic stress, neuroinflammation and BDNF level

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**Abstract**
*S. dinklagei* is a shrub whose aerial roots are used in southeastern Nigeria for the treatment of mental disorders. This study, therefore, investigated the neuroprotective effects of its sub-fraction on monosodium glutamate (MSG)-induced excitotoxicity, implicated in neurological disorders. The aerial roots of *S. dinklagei* were extracted with 80% methanol, after which the crude extract was partitioned, and the active fraction was subjected to vacuum liquid chromatography to obtain the sub-fractions. Mice were distributed into 5 groups of 8 mice each: Group 1 was the control, and Group 2 represented the MSG-induced group, which was left untreated. Groups 3-5 were induced with MSG and treated with 20 mg/kg memantine and 5 and 20 mg/kg of the sub-fraction (F10), respectively. Animals were orally administered the respective treatments and induced with MSG (2 g/kg; i.p.) after 1 h for 21 days. Mice were thereafter sacrificed, and the prefrontal cortex, hippocampus, and striatum were isolated for the evaluation of oxidative and nitrergic stress following standard protocols. Tumour necrosis factor-alpha (TNF-α), interleukin 6 (IL-6) and brain-derived neurotrophic factor (BDNF) concentrations were also assessed in the brain tissues using ELISA kits, while the histological analysis of brain tissues was achieved using Nissl stain. The decreased glutathione level, superoxide dismutase, catalase and glutathione S-transferase activities in the MSG group were increased in the treated groups. Malondialdehyde, nitrite, TNF-α and IL-6 levels were decreased in the F10-treated groups, while the concentration of BDNF increased significantly ($p < 0.05$) compared to the MSG group. Nissl stain showed atrophy and pyknosis of neurons in the MSG group, with 20 mg/kg F10 leading to increased neuronal count relative to the other groups. The neuroprotective effects of F10 are attributed to its modulation of oxido-nitrergic stress, neuroinflammation and BDNF level in mice, demonstrating its potential in neurological disorders.

### S1.029: Evaluation of the effect of vitamin C on serum oxidative parameters of male Wistar rats exposed to *Cannabis sativa*

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**Abstract**
This research investigates the impact of vitamin C on oxidative stress induced by *Cannabis sativa* in male Wistar rats. Oxidative stress arises from an imbalance between antioxidants and reactive oxygen species (ROS), which is associated with various clinical conditions. In addition to its recognised psychotropic properties, *Cannabis sativa* is known to induce oxidative stress. Vitamin C, a potent antioxidant, is believed to alleviate this stress. The experiment involved twenty male Wistar rats, which were acclimatised for 14 days prior to the study's initiation. The rats were randomly assigned to four groups, each consisting of five individuals: a control group receiving only food and water and three experimental groups (B, C, and D) that were administered 2 mg/kg, 4 mg/kg, and 6 mg/kg of body weight (BW) of *Cannabis sativa*, respectively, alongside 4 mg/kg of vitamin C. The study maintained strict adherence to the experimental protocol, measuring oxidative stress parameters such as glutathione (GSH), catalase, and malondialdehyde (MDA). Additionally, glutathione peroxidase (GPx), lactate dehydrogenase (LDH), superoxide dismutase (SOD), catalase (CAT), and total antioxidant capacity (TAC) were evaluated. The results indicated that the groups treated with both vitamin C and *Cannabis sativa* extracts exhibited a significant enhancement in antioxidant activity, effectively mitigating the indicators of oxidative stress compared to the control group. These findings underscore the potential therapeutic role of vitamin C in addressing oxidative stress-related disorders, suggesting its protective effects against oxidative stress induced by *Cannabis sativa*.

**Keywords**: Psychotropic, Reactive oxygen species, Antioxidants

### S1.030: 3,4-Methylenedioxymethamphetamine (MDMA) withdrawal affects social behaviour and neurotransmitter levels in male Sprague-Dawley rats

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**Abstract**
3,4-Methylenedioxymethamphetamine (MDMA) is a synthetic psychostimulant with serotonin-releasing properties, and it is known for its emotional and social effects, abuse potential, and neurotoxicity. Despite extensive research on its acute effects, little is known about its withdrawal impact. This study investigates MDMA and its withdrawal effects on social interaction and prefrontal cortex (PFC) neurotransmitter levels in male Sprague-Dawley rats. Fifteen rats were divided into three groups: control, MDMA-only, and MDMA with a 30-day withdrawal period. MDMA was administered orally at 100 mg/kg for 30 days, with social behavioural assessments being conducted every 10 days throughout a 60-day experimental period. Social behaviours assessed included contact-following, boxing, and total interaction time using an open field apparatus. PFC homogenates were collected and analysed for dopamine and serotonin on day 30 of MDMA exposure and post-withdrawal (day 60) via the ELISA method. Data was analysed with one-way ANOVA followed by *the Bonferroni post-hoc test* using GraphPad Prism with the level of significance set at $p<0.05$. Results showed that MDMA significantly increased contact-following time on day 10 and remained elevated until day 30 before decreasing from day 40 onwards. Boxing time initially decreased by day 20, then spiked significantly on day 40, persisting through day 60 ($P<0.05$). The total social interaction time showed a significant increase on day 30 (50.98±1.83 s) and a decrease on day 60 (20.19±0.63 s) ($p<0.05$). Biochemically, MDMA increased PFC serotonin and dopamine levels at day 30 when compared with the control group ($P<0.05$); in contrast, both neurotransmitter levels failed to return to baseline following MDMA withdrawal at day 60 ($P<0.05$). These findings suggest that MDMA enhances social interaction during active exposure. However, the observed behavioural changes following withdrawal suggest a tendency towards MDMA-induced antisocial-like outcomes, mediated by alterations in serotonergic and dopaminergic neurotransmission.

**Keywords**: MDMA, Psychostimulant, social- interaction, substance use disorders, Neurotransmitters

### S1.031: Omega-3 fish oil improves histoarchitectural features and increases medial prefrontal cortex volume and pyramidal cell number in male adult Wistar rats

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**Abstract**
Sleep deprivation poses a public health risk, and it is linked to neurodegenerative diseases like Alzheimer's. Omega-3 fish oil (O3FO) has demonstrated significant antioxidant, anti-apoptotic, and anti-inflammatory properties. This study aimed to evaluate the effects of O3FO on histo-architectural features, volume, and pyramidal cell numbers following sleep deprivation in Wistar rats. Twenty male Wistar rats (150-200 g) were randomly divided into four groups (n=5). Group I received 2 ml/kg distilled water, while Groups II, III, and IV underwent 18 hours of daily sleep deprivation for 21 days using the modified multiple-platform method. Groups III and IV were administered 200 mg/kg and 400 mg/kg of omega-3 fish oil, respectively. On day 22, rats were euthanised via intraperitoneal ketamine injection. Medial prefrontal cortex (mPFC) tissues were processed with haematoxylin and eosin staining for histoarchitecture assessment. Cavalieri's principle and physical dissector methods estimated mPFC volume and pyramidal cell counts in Layers III and V. Histological examinations revealed normal histoarchitectural features in the control group, showing healthy pyramidal cells, glial cells, and blood vessels. In contrast, the sleep-deprived group exhibited significant distortions, including cytoplasmic vacuolation, karyolysis, and pyknosis. The 200 mg/kg omega-3 fish oil group showed improvements similar to controls, while the 400 mg/kg group displayed cytoplasmic disorientation and degenerative changes. Stereological assessments indicated a significant decrease in mPFC volume and pyramidal cell count in the sleep-deprived group compared to controls. Notably, the 200 mg/kg group showed an increase in cell numbers compared to the sleep-deprived-only group. Overall, the sleep-deprived group had fewer pyrami-

dal cells than controls, with a slight increase in the 400 mg/kg group, though not statistically significant. The findings suggest that omega-3 fish oil treatment improved histo-architectural features and increased both the volume and number of pyramidal cells in the prefrontal cortex of sleep-deprived Wistar rats.

**Keywords:** Omega-3, Neuroprotective, Histo-architecture, Sleep deprivation, Stereological

### S1.032: Effect of dietary modification and intermittent fasting on neurobehaviour and oxidative stress markers in the brain

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**Abstract**
Dietary modifications, including intermittent fasting, can help reduce oxidative damage and improve behavioural health. This study aimed to investigate the effect of dietary modification and intermittent fasting on neurobehaviour and oxidative stress biomarkers in the brain. Thirty-six Wistar rats weighing 100-120 g were divided into 6 groups (n=6): Control – fed with a normal diet, IF – fasted and fed with a normal diet, HPD – fed with a high-protein diet, IF+HPD – fasted and fed with a high-protein diet, HFD – fed with a high-fat diet, and IF+HFD – fasted and fed with a high-fat diet. After a period of eight weeks, a neurobehavioural test was conducted using an elevated plus maze and a light and dark box. The brain was obtained for the oxidative stress assay. The results show that HFD, in combination with intermittent fasting, increases anxiety and MDA levels, while significantly decreasing CAT, GSH and nitrite levels compared to the control group. However, intermittent fasting and HPD only increased antioxidant levels significantly and had no significant difference in MDA level compared to the control group. This study indicates HFD as well as combination with intermittent fasting have detrimental effects on the brain.

**Keywords:** Diet, Oxidative stress, Anxiety, Intermittent fasting

### S1.033: Histomorphological and stereological characterisation of the pons of adult and juvenile African grasscutter (*Thrynomys swinderianus*)

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**Abstract**
The rodent species have become a remarkable tool in biomedical research, serving as an animal model of choice based on their physiology and genetic relativity to humans. Recently, the giant rodent, the African grasscutter (AGC), has been identified with unique characteristics, including hearing, movement, balance and reflexes regulated by structures of the brainstem, that provide an evolutionary advantage for survival in its natural habitat, hence its relevance as a tool in neuroscience investigations. This study characterised the pons in both adult and juvenile AGC using histomorphologic and stereologic approaches. Eight AGCs (adult and juvenile; n=4) were procured for this study. Brains were harvested, gross morphological features observed and dimensions measured (pontine breadth and length). Subsequently, sagittal and coronal histological paraffin sections of the brain were processed for light microscopic examination and stained with haematoxylin and eosin stains to demonstrate histoarchitectural features of the pons with a focus on age-related differences. Stereologically estimate the number of pyramidal cells and global volume of the pons from serial sections, sectioned at 8 *μm* using the Physical Fractionator Probe and Cavalieri Estimator, respectively. Comparison of gross features revealed structural similarities and variations between adult and juvenile specimens, including differences in dimensions and surface features of the pons. Light microscopy demonstrated similar pontine histoarchitectural features across the age groups. However, certain variations, including clear differentiation in neuronal layers and tissue composition and distribution and density of neuronal cell bodies, reflecting functional variations in the brainstem across the age groups, were observed. Stereological estimation revealed differences in the pyramidal cell count and global volumes of the pons across the age groups. Findings offer significant insights into the developmental neuroanatomy of the AGC, highlighting age-dependent morphological adaptations in the brainstem, and contribute to the broader understanding of rodent neurobiology.

**Keywords**: Dimensions, Histoarchitecture, Pyramidal cell count, Global volume