hydroxylase-TH and Monoamine Oxidase B– MAO–B), microglia activation (CD11b), pro-inflammatory (IL–1β), and oxidative stress (p47phox) biomarkers using Western immunoblot. Data was analyzed by one-way ANOVA (Tukey *post hoc*), with the level of significance set at $p < 0.05$.

6-hydroxydopamine-induced striatal lesion caused deficit in the contralateral use of forepaw (cylinder test), a reduction in the number of line crossed (open field test) and cognitive impairment (Novel object recognition test). Intervention withVD_3 or VD_3 + L-DOPA significantly reversed these motor-cognitive declines. Western blot data showed that PD-induced mice treated with VD_3 or VD_3 + L-DOPA resulted in a marked increase in the expression of the rate limiting enzyme-TH, and a reduction in MAO-B (catabolic enzyme) expression. Similarly, microglia activation, neuro-inflammation, and oxidative stress were down-regulated as depicted by reduction in the striatal expressions of CD11b, IL–1β and p47phox respectively. Interestingly, PD mice treated with VD_3 + L-DOPA showed a better outcome in the behavioral and western blot data compared to other treatment groups.

VD_3 showed neuroprotective effects against Parkinson's disease by modulating dopamine neurotransmission, oxidative stress, microglia activation and neuro-inflammation, which are some of the key factors implicated in the pathophysiology and progression of PD. These factors in turn could have protected against further neurodegeneration of the nigrostriatal pathway thereby sparing the dopaminergic neurons from the cytotoxic effect of 6-hydroxydopamine, and thus, enhancing the striatal release of dopamine, the neurotransmitter that regulate movement. This may possibly explain the consequent improvement in the motor-cognitive behavioral decline in the PD-induced mice. These neuroprotective actions of VD_3 suggest that, apart from altering the progression of PD, it may also reduce L-DOPA dosage thereby reducing the short comings of L-DOPA therapy associated with dosage and prolonged use in the treatment of Parkinson disease.

Keywords: Dopamine, Vitamin D, 6-Hydroxydopamine, Parkinson's disease.

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A31

Protective role of *adansonia digitata* on lead-induced neurotoxicity in the hippocampus of adult wistar rats

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Lead contamination is a known cause of neurotoxicity mainly involving hippocampal damage, which invariably impair brain functions and cognition. *Adansonia digitata* (A.D), also known as Baobab, has been shown to contain a high amount of vitamin C hence possess antioxidant properties. This study investigated the neuroprotective effect of aqueous leaf extract of *A.D* on lead induced hippocampal oxidative stress in adult Wistar rats.The leaves were procured from a farm in Zaria, Kaduna State and aqueous extraction was carried out.A total of 24 Wistar rats were divided into 6 groups (A-F) of 4 rats each. Group A(control) received normal saline, B received lead acetate (50 $mgkg^{-1}$ body weight), C; received 50$mgkg^{-1}$ body weight$CaNa_2EDTA$ and lead acetate (50 $mgkg^{-1}$ body weight), while groups D, E and F received 500 $mgkg^{-1}$, 1000 $mgkg^{-1}$, and 1500 $mgkg^{-1}$ body weight respectively of aqueous extract of A.D, as well as lead acetate (50 $mgkg^{-1}$ body weight). The experiment lasted for 14 days. At the end of the experiment, the rats were sacrificed and hippocampus harvested. A portion was fixed in Bouins fluid and processed for routine H&E while the other was homogenised and the tissue homogenate was assayed for the activities of the oxidative stress markers; superoxide dismutase (SOD) and malondialdehyde (MDA). Group B (lead acetate group) showed increase in SOD and MDA levels as well as alterations in the histoarchitecture of the hippocampus. However, the groups treated with $CaNa_2EDTA$ as well as the extracts showed significant decrease in SOD and MDA levels in hippocampus of Wistar rats with the best result at the highest dose of the extract (1500 mg/kg). The histoarchitecture of the hippocampus was also significantly ($P < 0.05$) preserved in the extract as well as the $CaNa_2EDTA$ treated groups. Crude aqueous extract of *Adansonia digitate* shows a dose dependent decrease on the oxidative stress induced by lead acetate in the hippocampus of adult Wistar rats.

Keywords: Oxidative stress; Superoxide dismutase (SOD); Malondialdehyde (MDA); Hippocampus; *Adansonia digitata*.

Ethical approval was obtained from Research Ethics Committee of the Faculty of Basic Medical Sciences, ESUT with reference no. ESUT/FBMS/422a.

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A32

Ameliorative effect of mimo2 (a novel compound from *Moringa oleifera* leaves) against vanadium-induced neurotoxicity

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Moringa oleifera (MO) is a shrub belonging to the family Moringaceae and various reports exist on the medicinal usefulness of its crude extract, including effect on some neurodegenerative diseases. Vanadium (V), a transition metal emitted into the atmosphere during fossil burning and gas flaring, is implicated in various neurodegenerative conditions. However, the ameliorative effect of a pure compound from MO has not been documented. This study was therefore designed to assess the neurotherapeutic properties of a pure compound isolated from MO leaves against vanadium-induced neurotoxicity in mice. A bioassay-guided fractionation was employed to separate the fractions of the methanol extract of MO leaves. Ferric reducing antioxidant potential assay was used to assess the fraction with the highest anti-oxidant potential, while preparative HPLC was employed to isolate the compound. Nuclear magnetic resonance was employed to elucidate the structure of the

pure compound obtained, which was named MIMO2. Cell culture assays (Dihydroethidium and Comet assay tests) using immortalised mouse hippocampal cell lines (HT22) were used to assess the effect of MIMO2 on vanadium neurotoxicity. Eighty-four 2-week old mice were randomly and equally divided into seven groups, and dosed intraperitoneally for 14 days, in the following groups: controls were water and DMSO, vanadium 3 mg/kg (V), MIMO2 5 mg/kg (M5), MIMO2 10 mg/kg (M10), M5+V, and M10+V. Hanging wire and open field neurobehavioural tests were carried out on day 14, while all animals were humanely sacrificed and perfused on day 15. Histological examination on the brain included H&E, Cresyl Violet (for hippocampal neuronal count in cornu Ammonis 1 and 3 regions), immunohistochemistry (for microglia and astrocytes, with sterological count for microglia), Black Gold II histochemistry and triple immunofluorescence with confocal imaging. Data were analysed using descriptive statistics and ANOVA at $\alpha_{0.05}$. The concurrent administration of MIMO2 and V in HT22 cells resulted in a significant reduction of the immuno-expression of reactive oxygen species (33% reduction) and vanadium-induced DNA damage (52% reduction). Administration of M10 resulted in a significant amelioration of the neurobehavioural deficits caused by vanadium. In V group, histology showed Purkinje cell degeneration, depletion and focal multiple layering, with cerebral gliosis, neuronal clumping and degeneration. All these neuropathologies were considerably reduced with the administration of M10. Cresyl Violet stain showed significant amelioration of vanadium-induced neuronal loss in the cornu Ammonis 1 region of M10+V ($4.9\pm2.3 \times 10^{-5}$/sqμm) compared to V ($3.9\pm1.9 \times 10^{-5}$/sqμm). For H&E and Cresyl Violet, no appreciable differences were observed in M5 and M5+V compared to controls and V group, respectively. The somatosensory cortex showed microglia and astrocytic hyperplasia and hypertrophy evident in the vanadium group (12.5±1.5%, area covered by microglia), which was significantly ameliorated in M10+V (9.3±2.3%). Black Gold II histochemistry showed severe vanadium-induced pantropic demyelination, particularly in the middle band of the corpus callosum, somatosensory and motor cortices, which were significantly alleviated in M10+V. This is the first documented report of the isolation of MIMO2 from *Moringa oleifera* leaves. MIMO2 displayed good neurotherapeutic activity against vanadium-induced neurotoxicity both *in vitro* and *in vivo* in mice.

Keywords: *Moringa oleifera* leaves, MIMO2, Vanadium neurotoxicity, Neurotherapeutic activity.

Ethical approval: Ethical approval was obtained from the Animal Ethical Committee of the University of Ibadan, ethical code number UI-ACUREC/App/2016/028.

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A33

Cholecalciferol attenuates 1-Methyl-4-Phenyl-1,2,3,6-tetrahydropyridine-induced parkinson's like-disease in brain damage

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Parkinson's disease is the motor neurodegenerative disorder which affects the dopaminergic neurons and causes significant loss of dopamine with 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) as a selective neurotoxin in the nigrostriatal pathway. Cholecalciferol (Vit.D_3) has been described as an active neurosteriod with antioxidant properties ubiquitously present in the brain. The study hypothesized that stimulation of vitamin D receptor by cholecalciferol could reduce autophagic cell death and degeneration following a state of drug-induced Parkinsonism in mice. The aim of the research was to investigate the attenuating effects of cholecalciferol on striatum and substantia nigra in mice model of MPTP-induced Parkinson's disease. Fifty adult male mice weighing about 25–35 g were randomly selected and assigned into 5 groups for 30 days of study. Group 1 received normal saline, Group 2 and 3 received MPTP and Vit.D_3 at low and high dose (MPTP:20 mg/kg and Vit.D_3: 50 mg/kg;100 mg/kg). Group 4 received MPTP only. The route of administration is intraperitoneal. The mice were then subjected to motor neurobehavioural test and biochemical assay (superoxide dismutase and catalase) was done with neuropathological evaluations of the brain. The results obtained showed a significant reduction in the estimated markers of oxidative stress with high dose of vitamin D_3 following MPTP induction. There was also statistical significant reduction in the expression of glia fibrillary acidic proteins (GFAP)-immuno-positive cells in the substantia nigra of the experimental mice when compared with the control group. It can be inferred that the administration of Vitamin D_3 was associated with significant attenuation of focal effects linked with MPTP in mice model of Parkinson's disease.

Keywords: Aging, Neurodegeneration, Dopaminergic neuron, Vitamin D_3, Environmental toxins.

Ethical approval: It was approved by Afe Babalola University Ethical Committee.

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