

to the control, LA-treated group showed altered ($p < 0.05$) levels of serum oxidative stress markers, which was ameliorated with CFPD-treatment, especially at dose 250 mg/kg. Lead has potentials of triggering deleterious and irreversible health effects ranging from biochemical, physiological, neurological and behavioural dysfunctions in both humans and animals. One amongst the established mechanism of lead-triggered neurotoxicity is, the generation of free radicals. Agents with antioxidant properties have been reported to ameliorate toxicity in biological systems generated as a result of free radical production. Results suggest that CFPD has neuroprotective potentials against lead acetate-induced cerebellar and cerebral toxicity in Wistar rats which could be attributed to antioxidant properties.

Keywords: Histoarchitecture; Neurodegeneration; Neuroprotection; Neurotoxicity.

<https://doi.org/10.1016/j.ibror.2019.09.061>

Neurotoxicity

B1

Orally administered artesunate modulates activity of cell body defense in plasma and brain of female wistar rats

H.A. Abdulrahim^{1,*}, T.M. Salman², A.A. Feyitimi², N.A. Omeiza²

¹ Department of Medical Biochemistry

² Department of Physiology, Faculty of Basic Medical Sciences, College of Health Sciences, University of Ilorin, Ilorin, Nigeria

E-mail address: abdulrahim.ha@unilorin.edu.ng (H.A. Abdulrahim).

Artesunate is a potent and rapidly acting blood schizonticide used in the treatment of acute severe malaria, especially cerebral malaria. The present study evaluated the potential effects of varying doses of artesunate on some biochemical parameters in female Wistar rats. Fifteen (15) female Wistar rats weighing 150 ± 10 g were used and blindly randomized into three groups having five (5) animals each. Control received distilled water (0.2 ml/kg day 1–5), LDA and HDA were treated with oral artesunate (2.90 mg/kg day 1; 1.45 mg/kg day 2–5) and (8.70 mg/kg day 1; 4.35 mg/kg day 2–5) respectively. The results showed that artesunate significantly reduced the plasma potassium (K^+), calcium (Ca^{2+}), phosphorus (P), total protein, malondialdehyde (MDA), glutathione (GSH), phospholipids (PL) but raised hydrogen bicarbonate (HCO_3^-) and nitric oxide (NO) levels when compared with control. Furthermore, its effect in the brain tissue indicated marked elevations of sodium (Na^+), Ca^{2+} , HCO_3^- , P, MDA, with a concomitant decreases in NO levels when compared with control. The current findings suggest that artesunate could cause imbalance in the electrolytes and minerals, and alterations in prooxidant/antioxidant levels in plasma and brain tissue with ameliorative responses from cell body defense mechanisms. Therefore, dietary supplements are recommended along with therapeutic usage of artesunate.

Keywords: Artesunate; Biochemical Parameters; Prooxidants; Antioxidant; Dietary Supplements.

<https://doi.org/10.1016/j.ibror.2019.09.062>

B2

Assessment of the effect of post-natal lead exposure on the hippocampus of developing wistar rats

O.D. Olatomide*, S.S. Adebisi, S.A. Musa

Neuroscience Unit, Department of Human Anatomy, Faculty of Basic Medical Sciences, College of Health Sciences, Ahmadu Bello University, Zaria, Nigeria

E-mail address:

segundavis1995.olatomide@gmail.com

(O.D. Olatomide).

Human child development is regulated by the interactions of both endogenous and exogenous factors. One of the exogenous factors affecting early development of neurobehavior in children is the exposure to heavy metals. Lead is a highly toxic heavy metal found in every aspect of environmental and biological systems. It is readily absorbed through the gastrointestinal tract and distributes into soft body tissues such as kidney, bone marrow, liver and brain, but accumulates in the blood and bone. It is also believed that the greater absorptive capacity of the gastrointestinal tracts of children puts them at a higher risk than adults. The central and peripheral nervous systems are the most sensitive targets for lead induced toxicity. Lead has negative effect especially on the developing nervous system of the fetuses and young children, which absorbs a higher fraction of this metal. While peripheral effects in adults often go away when lead exposure ceases, evidence suggests that most of lead's effects on a child's central nervous system are irreversible. In this study we examined the effect of post-natal lead exposure on the cytoarchitecture of the hippocampus in developing Wistar rats by evaluating the effect of lead on body weight, hippocampus weight and CA3 regions of the hippocampus using Haematoxylin and Eosin and Cresyl fast violet. We hypothesized that post-natal lead exposure will not cause distortion in the hippocampus of developing Wistar rat. Ethical approval was obtained from the Ahmadu Bello University Committee for Animal Use and Care with approval number (ABUCAUC/2018/047). Nine non pregnant and three male apparently healthy Wistar rats weighing between (120 g–150 g) were purchased and acclimatized for two weeks prior to experimental study. Estrus cycle and mating was carried out using the method of Marcondes *et al.* (2002). Nine pregnant rats were randomly distributed into three groups of three rats each, consisting of control group (1) and experimental groups (2 and 3). Dams in control group were given distilled water while dams in Group 2 and group 3 were administered 60 mg/kg and 90 mg/kg bwt of lead acetate respectively from the day of parturition (post-natal day (PND) 1) to PND 21. The pups of the experimental groups (2 and 3) were exposed to lead acetate via lactation from dams' that were administered lead acetate via oral gavage from PND 1 – PND 21. On PND 22, all the pups were weighed, then euthanized using 75 mg/kg of ketamine intraperitoneally. The head was decapitated and the brain tissue was harvested by making a midline incision through the skull. The hippocampus was then extracted, weighed and fixed in Bouin's fluid for histological studies. The result showed a significant decrease ($p < 0.05$) in body weight of the pups in the control group when compared with the lead acetate treated groups. There was also a decrease in hippocampus weight although not significant ($p > 0.05$). We also observed chromatolysis of Nissl substance and distortion in cytoarchitecture of CA3 region of the hippocampus of Wistar rat's pups exposed to increasing doses of lead acetate via lactation from PND 1–21. Counter to our hypothesis, post-natal lead exposure caused distortion of the cytoarchitecture of the hippocampus of the pups possibly via oxidative stress, dissolution of Nissl substance and ability of lead to displace calcium.