



The Biochemical Effects of Aluminum Intoxication on Serum Lipid Profile of Male Wistar Albino Rats.

PN Ogueche^{1,3}, IC Maduka¹, MN Ugwu² and O Obidoa³

¹Department of Human Biochemistry, Faculty of Basic Medical Sciences, College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria.

²Department of Medical Biochemistry, Faculty of Basic Medical Sciences, Cross River State University of Technology, Calabar, Cross River State, Nigeria.

³Department of Biochemistry, Faculty of Biological Sciences, University of Nigeria, Nigeria.

ARTICLE INFO

Article history:

Received: 29 July 2017;

Received in revised form:

1 September 2017;

Accepted: 11 September 2017;

Keywords

Aluminum,
Intoxication,
Serum Lipid Profile,
Total cholesterol,
Low Density Lipoprotein.

ABSTRACT

This study was designed to investigate the biochemical effects of graded doses of aluminum on some serum lipid profile (SLP) of male wistar albino rats. A total of twenty-four male albino rats of 10-12 weeks of age were used for the study. They were randomly assigned to six groups (Groups A-D) of six rats each. The treatment groups-A to C were administered aluminum as aluminum chloride (AlCl₃) : 0.38, 3.8, and 38 mg/kg body weight while group D received 0.2ml normal saline which served as vehicle. Assay of the SLP were carried out using standard biochemical methods after 14 days. The results showed that serum total cholesterol of the treatment group administered 38mg/kg decreased significantly (p<0.05) relative to the control whereas the serum low density lipoprotein (LDL) of the treatment groups administered 3.8mg/kg and 38mg/kg decreased significantly (p<0.05) relative to the control after 14 days of treatment. The treatment group administered 0.38mg/kg showed a decrease in serum total cholesterol and low density lipoprotein but were not statistically significant (p>0.05).

© 2017 Elixir All rights reserved.

Introduction

Human systems have inorganic elements as their integral constituents (Ogueche et al., 2009). These inorganic elements may be either classified as bulk elements or trace elements, which aid in the growth and metabolism. Aluminum is neither classified as bulk nor trace element yet, finds its way into the human system. Aluminum is ubiquitous, being the third most prevalent element and abundant metal in the earth's surface mainly in the combined form as silicates, oxides, and hydroxides (WHO, 1997). It is released into the environment both natural and anthropological sources. Aluminum has a variety of applications such as in food industry (as a packaging foil and drying agents), pharmaceutical industry (as an anticholinesterase and antiperspirant), and engineering works (in construction of roofing sheets, vehicle parts), etc (Abbasali et al., 2005, Dominigo, 1995, Agrawal et al., 1996). Perhaps the numerous applications of aluminum is because of its light-weight, corrosion-free, and relative inexpensiveness (Kandiah and Kies, 1994). Aluminum is a known neurotoxin that can predispose individuals to certain diseases such as Alzheimer's, dementia, Parkinsonism, and amyotrophic lateral sclerosis (Wurtman, 1985, Alferey et al., 1976). It also affects some body structures like the skeletal systems, brain tissues, and blood cells (Ajoy et al., 1990, Mestaghanmi et al., 2002). The mechanism of aluminum toxicity is poorly understood. Aluminum is absorbed by cells through transferring receptors similar to iron absorption (Skiillen and Moshtaghi, 1997). It transverses across the cell membrane and enters into the blood circulation where it binds to the serum proteins particularly transferrin (Moshtaghi and

Ani, 1992). The target tissues for aluminum burden are bones, brain, kidneys, and liver (Ajoy et al., 1990). However, in spite of the aluminum burden on some tissues, little is known about the serum lipid profile of aluminum toxicity and this warrants the study.

Materials and Methods

Materials: Animals used for this study were male Wistar albino rats aged between 8-10 weeks with body weight range of 150-205g. They were obtained from the animal house of the Faculty of Veterinary Medicine, University of Nigeria, Nsukka. All chemicals used in this study were of high quality and analytical grade. The Institutional Animal Ethics Committee approved the study before the experiment and certified all experimental protocols.

Experimental Design: Twenty- four male rats were housed in four separate cages of six rats each and acclimatized for five days. The four test groups include: **Group A**, administered 0.38mg/kg body weight of aluminum, **Group B**, administered 3.8mg/kg body weight of aluminum, **Group C**, administered 38mg/kg body weight of aluminum, while **Group D**, (control) administered 0.2ml of normal saline which also served vehicle for the dissolution of the toxicant. All Chemicals used in this study were of analytical grade. The route of administration was per oral (p/o) exposure. All groups were fed with commercial feed (grower's mash) and water *ad libitum* for fourteen (14) days. The experiment was replicated thrice and their results were pooled together. Blood was collected from each group i.e. control and the three test groups on day 14, through the median cantus vein in the eyes of the rats with the aid of a capillary tube and transferred into

plastic test tubes. This was later centrifuged at 2000xg in separate test tubes and serum/sera collected after discarding the supernatant. The animals were later sacrificed. The following parameters: the total cholesterol and low density lipoprotein (LDL) were assayed by the methods of King and Wooten, 1959 and Burstein and Samaille, 1958 respectively using the serum of animals from the various groups.

Statistical Analysis:

Standard error mean (\pm SEM) of replicate experiments with triplicate samples were taken for each analysis. Significant differences of results were established by one way analysis of variance (ANOVA) while differences between groups were assessed by student's independent t-test. The acceptance level of significance was $p < 0.05$ using a 2-tail distribution.

Results and discussion

The results of serum total cholesterol (mg/100ml) are shown in Table 1. Total cholesterol of the test group treated with 38mg/kg body weight of $AlCl_3$ significantly decreased ($p < 0.05$) relative to the control group, while the other test groups given 0.38mg/kg and 3.8mg/kg body weight of $AlCl_3$ decreased non-significantly ($p > 0.05$) after the fourteenth day of exposure compared to the control. There was no significant difference ($p > 0.05$) in the cholesterol level within the aluminum treated groups, however, the aluminum-treated group with 38 mg/kg showed the least serum total cholesterol after the fourteen days of treatment.

Table 1 . Total Serum Cholesterol (mg / 100ml).

Groups /Days	control	0.38mg/kg	3.8mg/kg	38mg/kg
14	196.94 \pm 8.95	192.20 \pm 9.0	186.90 \pm 10.0	178.70 \pm 12.6*

In Table 2, however, after fourteen days of aluminum treatment, the low density lipoprotein (LDL) of the test groups given 3.8mg/kg and 38mg/kg decreased significantly ($p < 0.05$) relative to the control group, while the test group given 0.38mg/kg decreased non-significantly ($p > 0.05$). However, the test group given 38mg/kg had the least LDL on the fourteenth day of treatment. Results from this study showed a significant decrease ($p < 0.05$) in total cholesterol and low density lipoprotein (LDL) of the test group given 3.8mg/kg and 38mg/kg body weight of $AlCl_3$ compared to the control group after fourteen days of treatment.

Table 2.0. Low Density Lipoprotein (LDL) mg/100ml.

Groups /Days	control	0.38mg/kg	3.8mg/kg	38mg/kg
14	396.00 \pm 25.2	382.00 \pm 20	355.60 \pm 21.0	346.90 \pm 30.0*

* Significantly Different Between the Control and Test Groups.

Discussion

Toxic metals are widely found in our environment. Human are exposed to these metals from numerous sources: contaminated air, water, soil and food (Deloncle et al., 1999; and Elstner and Osswald, 1994). These results agree with the findings of Chang *et al.*, (1998). In normal tissue, there is a balance between the production and scavenging of reactive oxygen metabolites (ROMs). According to Sies, (1997), oxidative stress occurs when the rate of cellular antioxidant depletion exceeds the rate of replacement. The consequence of such is tissue damage and may lead to cell death (Nwanguma *et al.*, 1999). Cholesterol is found in cell membranes where it function and regulate the fluidity of the cell membrane.

Nevertheless, a positive association between serum cholesterol concentration and coronary heart disease (CHD) have been reported elsewhere (Castelli, 1986; Tyroler, 1987; Austin, 1988; Barbir et al., 1988 and Manninen et al., 1989). However, in our study the results showed no associated cardiac dysfunctions following the reductions in total cholesterol and low density lipoprotein (LDL). And cholesterol is transported in LDL particles. In addition, there is a strong correlation between a high serum cholesterol concentration and coronary heart disease (CHD), especially atherosclerosis or hardening of the arteries. From our results, we infer that Al exposure to rats may not predispose rats to diseases associated with cardiac events.

References

- Abbasali, K.M., Zhila, T. and Farshad, N. (2005). Developmental toxicity of aluminium from high doses of $AlCl_3$ in Mice. *J of Appl Res.* **5**: 4.
- Agrawal, S.K, Ayyash, L., Gourley, C.S., Levey, J., Faber, K. and Haghes, C.L. (Jr). (1996). Evaluation of developmental Neuroendocrine and reproductive toxicology of aluminium. *Food Chem. Toxicol.* **34**: 49-53.
- Ajoy, K.R., Geecta, T. and Archuna, S. (1990). Effects of aluminium sulphate on human leukocyte chromosomes in vitro. *Mutat. Res.* **244**: 179-183.
- Alferey, A.C., Legendre, G.R. and Kachny, W.D. (1976). The dialysis encephalopathy syndrome. Possible aluminium intoxication. *N. Engl. J. Med.* **294**:184-188.
- Austin, M.A. (1988): Epidemiologic associations between hypertriglyceridemia and coronary heart disease, in: Seminars in Thrombosis and Hemostasis. New York, Thieme Medical Publishers, Inc, pp 137-142.
- Barbir, M., Wile, D., Trayner, I., Aber, V.R., Thompson, G.R. (1988). High prevalence of hypertriglyceridaemia and apolipoprotein abnormalities in coronary artery disease. *Br. Heart J.* **60**: 397-403
- Burstein, M. and Samaille, J. (1958). Determination of Serum Lipoprotein after Selective Precipitation by heparin. *Press. Med.* **66**: 974-975.
- Castelli, W.P. (1986) The triglyceride issue: A view from Framingham. *Am Heart J.* **112**:432-437
- Chang, G., Denofrio, D., Desai, S. Kelley, M.P. Rader, D.J., Acker, M.A. and Loh, E. (1998). Lipoprotein (a) levels and Heart Transplantation Atherosclerosis. *Am Heart J.* **36** (2): 329-334.
- Deloncle, R., Huguet, F., Babin, P., Fernandez, B., Quellard, N. and Guillard, O. (1999). Chronic administration of aluminum L glutamate in young mature rats: Effects on iron levels and lipid peroxidation in selected brain areas. *Toxicol. Lett.* **104**: 65-73.
- Dominigo, J.L. (1995). Reproductive and developmental toxicity of aluminium: a review. *Neurotoxicol Teratol.* **17**: 515-521.
- Elstner, E.F. and Osswald, W. (1994). Mechanism of O₂ activation during plant stress. *Prod. R. Soc. Edinb. Sect. B.* **102**: 131-154.
- Kandiah, J. and Kies, C. (1994). Aluminium concentration in tissues of rats: effect of soft drink packaging. *Biometals.* **7**(1): 57-60.
- King, E.J and Wooten, I.D. (1959). Total Cholesterol Determination in Plasma/Serum. In: *Microanalysis in Medical Biochemistry.* Churchill, London. p 42.

- Manninen, V., Huttunen, J.K., Tenkanen, L., Heinonen, O.P., Minttairi, M., Frick, M.H. (1989). High-density lipoprotein cholesterol as a risk factor for coronary heart disease in the Helsinki Heart Study, in Miller NE (ed): High Density Lipoproteins and Atherosclerosis II. Amsterdam, Excerpta Medica, ICS 826, pp 35-42.
- Mestaghanmi, H., El-Amrani, S. and Saile, R. (2002). Effect of aluminium chloride administration during gestation in rats. *Stal.* **27**: 73-81.
- Moshtaghie, A.A., and Ani, M. (1992). Comparative binding study of aluminium and chromium to human transferrin. *Biol. Trace Elem. Res.* **32**: 39-46.
- Nwanguma, B.C., Achebe, A.C., Ezeanyika, L.U.S. and Eze, L.C. (1999). Toxicity of oxidized fats: Tissue levels of lipid peroxides in rats fed by a thermally oxidized corn oil diet. *Food and chem. Toxicol.* **37**: 413-416.
- Skillen, A.W. and Moshtaghie, A.A. (1985). The effect of aluminium on the interaction between transferrin and its receptor on the placenta membrane. In: Aluminium and other elements in renal disease. A Taylor ed. Brailier tindal. London. Pp 85-89.
- Sies, H. (1997). Oxidative Stress: Oxidants and antioxidants. *Exp. Physiol.* **82**: 291-295.
- Tyroler HA (1987). Review of lipid-lowering clinical trial in relation to observational epidemiologic studies. *Circulation*; **76**:515-522
- WHO, (1997). International programme on chemical safety. Environ. Health Criteria 194. Aluminium. Pp. 1-3. Review.
- Wurtman, R.J. (1985). Alzheimer's disease. *Sci. Am.* **252**: 62-66, 72-74.