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RESEARCH PAPER

EFFECT OF POTASH ON RENAL PROFILE OF ALBINO WISTAR RATS

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ABSTRACT

This study is intended to investigate the effect of Potash on renal function. Twenty four (24) Albino wistar rats with mean weight of 126.3±23.9g were used. They were subdivided into four groups -A, B, C and D (n=6 each); with A serving as control while B, C and D served as test groups. Group A received normal feed and water while B, C and D received 3.0g/kg, 6.0g/kg and 9.0g/kg of potash per body weight for 21 days respectively. At the end of the experiment, the animals were sacrificed under light anesthesia to obtain blood samples for the estimation of renal function parameters. Sodium, Potassium, Chloride, Urea, Uric acid and Creatinine was analyzed using standard analytical chemical methods. The results showed a significant ($P<0.05$) reduction in body weight and serum levels of sodium, potassium, chloride, urea and creatinine among the test groups. Serum levels of uric acid in the test groups were not significantly different ($P>0.05$) from that of the control. Our findings therefore, suggest that potash may have dosage dependent nephrotoxic and weight reduction potentials and by implication, may induce growth retardation. This indicates therefore, that potash is toxic to the kidney and there is need for further studies.

Key words:

INTRODUCTION

Various environmental chemicals and industrial pollutants have useful and harmful potentials (Moore, 2003). Of interest in this study is potash which is the common name for various mined or manufactured salts containing potassium in water-soluble forms (Jasinski, 2008). The name is derived from the combination of the words "pot" and "ash". Potash is also a name given to a naturally occurring reddish coloured mixture of the minerals sylvite (KCl) and halite (NaCl) that form a rock called sylvinite. The commercial products produced from sylvinite are referred to as muriate of potash and often symbolized as "K" (NRCAN 2008). The variable uses of potash have tremendously led to the study of its effect on various body organs. This study therefore, is designed to evaluate the effects of potash on kidney function using adult albino wistar rats as the animal model.

MATERIALS AND METHOD

Experimental animals: Twenty four (24) Adult Albino Wistar rats of comparable sizes and weights ranging from 150 to 200g were procured from the animal farm of Anthonio services Nigeria, Ekpoma, Edo State, where they were allowed to acclimatize for two (2) weeks. They were kept in wire mesh cages with tripod that separated the animal from its faeces to prevent contamination. During this period of acclimatization, the rats were fed with growers' mash

and water provided ad libitum. The animals were maintained and utilized in accordance with the standard guide for the care and use of Laboratory animals.

Animal grouping: The animals were divided into four groups of six (6) rats each. Group A served as the control while groups B, C and D served as the test groups treated with graded doses of potash.

Substance of study: Considerable quantities of Potash were purchased from Kersmond grocery stores, Ekpoma, Edo State. The Potash purchased was carefully poured on a clean dry plastic container. From this container it was measured using Electric Balance and packaged in small plastic envelopes and then stored pending usage. The substance preparation process was performed with maximum care in order to avoid any form of contamination.

Experimental Procedure: The test groups, B, C and D were administered 3.0g/kg; 6.0g/kg and 9.0g/kg of the measured potash respectively. The respective potash doses were prepared by mixing with feed to add up to 100g. Group A received normal feed (100g) and water only. The administration period lasted for a period of 21 days.

Sample collection: Blood samples (5mls) of each rat were obtained at the end of the administration (3 weeks) under chloroform anaesthesia and dispensed into plain containers labeled appropriately (A, B, C and D). The samples were centrifuged for 5 minutes at 3000 rpm within two hours after collection into clean dry plain containers which are labeled corresponding to the initial blood sample container. The serum obtained was stored frozen until the time of analysis. Laboratory analysis was carried out for sodium, potassium, chloride, creatinine, Urea and Uric acid.

Estimation of Biochemical parameters: Estimation of serum creatinine was done using the modified Alkaline Picrate method described by Fabiny and Ertingshausen (1971) while serum urea was done using the Urease berthelot method described by Sims (2006). Enzymatic colorimeric method (Uricase) was employed for uric acid as described by Cheesbrough (2005) while sodium and potassium was estimated by flame emission photometry described by Scoog (2005). Also, chloride was estimated based on Schales and Schales method described Schales and Schales, (1971).

Statistical Analysis: The results obtained from the laboratory investigation were expressed and mean±SD. Statistical significance was determined using one way analysis of variance (ANOVA) while values of $P<0.05$ was considered to be statistically significant.

RESULTS

Table 1 presented the body weight changes in wistar rats fed with Potash for 21 days. In this study it was observed that the mean weight of both test groups and control increased before the administration of the substance, this observation as was not statistically significant ($P>0.05$). Similarly, there was significant reduction ($P<0.05$) in the body weight following the administration of potash

Table 2 presents the renal profile of wistar rats fed with graded doses of Potash for 21 days. The results obtained in this study shows that there was significant increase ($P<0.05$) in the levels of serum Sodium, Potassium, Chloride, Urea and creatinine in the treated animals when compare to the values in the control. These statistical significant differences in the mean values of these parameters were dosage dependent. Also, there was no statistical significant difference ($P>0.05$) in the uric acid levels.

DISCUSSION

This study investigated the effect of potash on weight and Renal function of Albino wistar rats. The results obtained showed that the administration of potash induced a significant decrease in the weight of wistar rats fed orally for 21 days when compared with the control groups. This reduction is dosage dependent. This finding is in agreement with the study carried out by Kurokawa *et al*., (1990) in which they reported that the reduction in body weight could be as a result of the chemical nature of potash and probably as a result of decreased feed intake resulting from the undesirable taste of potash. This finding is also supported by the work done by Okalie and Ikewuchi (2004) who reported a significant reduction in body weight of Rabbits that received potassium bromate.

TABLE 1: Body weight changes of rats fed graded doses of potash at various intervals.

Stages of Weight measurement	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)
WBE	116.7±20.4[a]	120.8±33.2[a]	125.0 ± 22.4[a]	142.5 ± 19.7[a]
WAE	145.0±20.0[a]	137.5±26.2[a,b]	120.8±18.8[a]	100.0 ±31.6[b]

KEY: Values are mean ± Standard deviationValues in a row with different superscripts are significantly different at P≤0.05. **WBE:** Weight before experiment. **WBE:** Weight after experiment

TABLE 2: RENAL PROFILE LEVELS IN WISTAR RATS FED WITH GRADED DOSES OF POTASH

PARAMETERS	Group A (n=6)	Group B (n=6)	Group C (n=6)	Group D (n=6)
Sodium (mmol/l)	125.3± 2.6[a]	134.8±1.9[b]	152.5 ± 2.4[b]	155.3 ± 1.9[b]
Potassium (mmol/l)	1.30 ± 0.2[a]	1.8±0.6[b]	3.8±0.3[b]	4.9 ± 0.1[b]
Chloride (mmol/l)	79.3±4.6[a]	85.7±1.6[b]	92.2±2.5[b]	91.0±0.9[b]
Urea (mg/dl)	32.3±6.2[a]	45.0±14.7[b]	54.2±6.5[b]	63.2±10.8[b]
Uric Acid (mg/dl)	7.9±1.1[a]	7.5±0.3[a]	7.7±0.5[a]	7.8±0.8[a]
Creatinine (mg/dl)	0.5± 0.2[a]	0.8±0.1[b]	1.6±0.3[b]	2.9±0.2[b]

Values are mean ± Standard deviation; Values in a row with different superscripts are significantly different at P≤0.05

In this study, it was observed that there was an increase in the levels of serum urea, creatinine, potassium, sodium and chloride as well as normal levels of uric acid when compared with the control group. This increase was statistically significant (P<0.05). These observations were in agreement with Khan *et al*., (2003) who also reported markedly elevated plasma urea and creatinine levels in rats fed orally with potash. Also, this report correlates with the study done by Khan *et al*., (2003) who stated that 125mgkg^{-1} body weight of potassium bromated given intra peritoneally to rats resulted in marked elevation Blood urea nitrogen and creatinine. Similar findings were reported by Watanabe *et al*. (2001).

Also, Urea and Creatinine are good indices of renal function. Therefore elevated levels of these parameters are indicative of nephrotoxicity. This correlates with the study carried out by Dioka *et al*., (2004); Boogaard *et al*., (2005); Hernandez-Seraato *et al*., (2006) on the nephrotoxicity effect of certain mixtures of hydrocarbon, gasoline vapours, lead, insects and pesticides in human and experimental animals.

This study, revealed a clear indication that potash contains chemical substances with nephrotoxic potentials. The specific chemical constituents and mechanisms responsible for nephrotoxic effect reported in this study to be associated with oral exposure to potash in not very clear. However, it may be assumed that reactive metabolites of potash constituents could have interacted with the renal tissues to cause derangements in glomerular functions.

An elevation in circulating serum creatinine, urea, potassium, sodium and chloride has been reported to be strongly associated with the development of hypertension and renal disease (Mazzali *et al*., 2001; Hernandez-Serrato *et al*. 2006; Patil *et al*., 2007). All these literature reports correlates the results of the findings in this present study, thereby documented a hypothetical evidence that exposure to potash may be a risk factor for the development of hypertension and renal disorders.

Furthermore, the significant increase in the concentration of creatinine in the serum reported in this study might have resulted from its decrease excretion which in turn is related to renal insufficiency. The concentration of creatinine in the blood is known to correlate inversely with the volume of glomerular filtration. Hence, creatinine is considered to be among the useful markers of the filtration function of kidneys, particularly creatinine is excreted only through the kidney (Appelton, 1995; Birkner *et al*., 2000; Grucka-Mamcza *et al*., 2005). Increase serum urea

concentration also explains the impaired renal function implicated in rats treated with potash. The high serum urea levels may result from a decrease in the rate of urea secretion in the urine which may likely result from renal insufficiency and shut down (Dioka *et al*, 2004, Hernandez-Serrato *et al*., 2006; Patil *et al*., 2007). According to Appelton (1995), increase in serum urea and creatinine concentration is a reflection of impaired renal function. However, the parameters reported in this study are indicators of renal function.

Potash is hence considered toxic to the kidney owing to the various changes in the renal function parameters assessed. Therefore, at higher doses, potash is nephrotoxic and can induce renal damages. Conclusively, potash may have been the cause of the various deleterious distortions which occurred in the kidney and a necessary reduction in its consumption is essential.

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REFERENCES

Appelton, J. (1995): Changes in plasma electrolytes and metabolites of rat following acute exposure to sodium fluoride as strontium chloride. *Arch. Oral. Biol*., 40: 265-268.

Birkner, E.G., Mamczar, Z., Machoy, R.T. and Polaniak R. (2000): Disturbance of protein metabolism in rats after acute poisoning with sodium fluoride. Fluoride, 33: 182-186.

Boogaard, P.J., Rocchi, P.S.J. and Sittert, N.J.V. (2005): Effect of exposure to low concentrations of fluorinated hydrocarbons on the kidney and liver of industrial workers. *B.J. Ind. Med.,* 50: 331-339.

Cheesbrough, M. (2005): Clinical chemistry test. In:District laboratory practice in tropicalcountries. Cambridge University press. Low price edition. 2nd ed. 333-337.

Dioka, C.E., Orisakwe, O.E. Adeniyi F.A . and Meludu, S.C. (2004): Liver and Renal function in Artisans occupationally exposed to lead in mechanic village in Nnewi, Nigeria. *Int. J.Environ. Res.,* Public Health, 1: 21-25.

Grucka-Mamczar, E., Birkner, J. Zaleyskafiolka, J. and Machoy, Z. (2005): Disturbances of kidney function in rats with fluoride-induced hyperglycemia after acute poisoning by sodium fluoride. *Flouride*, 38: 48-51.

Guyton, A.C. and Hall, J. (2006): Metabolic functions of the liver. Text Book of Medical Physiology. W.B sanders company Philadelphia. Pp. 861-864.

Hernandez-Serrato, M.I., Fortoul, T. I., Rojas-Martinez, R., Mendoza-Alvarado, L. R. and Canales-Trevino et al.(2006): Lead blood concentrations and renal function evaluation: Study in an exposed Mexican population. *Environ* . Res., 100: 227-233.

Jasinski, S. M. (2008): "Potash". Available at http://minerals.usgs.gov/minerals/pubs/commodity/potash/.

Khan, N., Sharma, S. and Sultan, S. (2003): *Nigella Sativa* (Black cumim) ameliorates potassium bromated induced early events of carcinogens- dimension of oxidative stress. *Hum. Exp. Toxicol*., 22: 193-203.

Kurokawa, Y., Mackawa, A., Takahashi, N. and Hayeshi, Y. (1990): Toxicity and carcinogenicity of potassium bromate, a new renal carcinogen. *Environ Health Perspect.,* 87: 309-355.

Mazzali, M., Hughes, J., Kim, Y., Jefferson, A. and Kang (2001): Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. Hypertension, 38: 1101-1106.

Moore, K.L. (2003): Congenital malformations due to environmental factors; Developing Humans W.B. Saunders Co. Ltd Philadelphia 2nd Ed. Chap 8. P. 173-183.

Natural Resources Canada (NRCAN) (2008): Preliminary estimate of the mineral production of Canada, by province, 2008. http://mmsd.mms.nrcan.gc.ca/stat-stat/prod -prod/PDF/2008p.pdf (accessed March 2009).

Okalie, N. P. and J.C. Ikwuchi, (2004): Cataractogenic Potential of Bromate-medicated oxidative stress in rabbits. *J. Med.Sci*., 4: 158-163.

Patil, A.J., Bhagwat, V.R., Dongre, N.N., Ambekar, J.G. and Das, K .K.(2007): Occupationallesd exposure in battery manufacturing workers, silver jewlry workers, and spray paintersin western Maharashtra(India): Effect on liver and kidney function. *J. Basic Clin. Physio. Pharmacol,* 18: 87-100.

Skoog, D.A., West, D. M., Holler, F. J. and Crouch, S. R. (2005). *Analytical Chemistry: An Introduction, 7th ed.* Chapter 23, pp. 594 631.

Sims, G.K. (2006): Using the berthelot method for nitrite and nitrate analysis soil sci. soc. *Am.J.* 70(3): 1038.

Watanabe, S., Yosshimura, Y. and Fukui, T. (2001): Contribution of glutathione peroxidase and nitric oxide to potassium bromate-induced oxidative stress and kidney damage in mice. *J. Health. Sci*., 47: 565-570.

AUTHORS CONTRIBUTION

This study involved animal handling, sample collection, sample analysis, statistical analysis, and research reporting (manuscript preparation and review). All the authors played significant roles and no conflict of interest is declared.