Biomedical & Pharmacology Journal Vol. **1**(1), 47-50 (2008)

The bioaccumulation of mercury in kidney and liver of wistar rat exposed to methyl mercury

E.K. NWANGWA, C.R. NWOKOCHA, U.E. UZUEGBU* and S.I. OVUAKPORAYE

Department of Physiology and *Department of Medical Biochemistry College of Health Sciences, Delta State University Abraka (Nigeria)

(Received: February 12, 2008; Accepted: April 04, 2008)

ABSRACT

The bioconcentration of mercury in the kidney and liver of rats was studied. Twenty-four wister rats were randomly divided into four experimental groups(n=6). GroupA(control)were given rat chow and drinking water .Group B (given rat chow +10% by mass of *Solanum lycopersicum*(sl). Group C (given rat chow + mercury in water at a concentration of 10ppm . Group D (given rat chow +Hg in 10ppm+10% by mass of sl).

The result shows a statistically significant increase (P<0.05) in the concentration of mercury in the liver and kidney. There was also a statistically significant increase (P<0.05) in liver enzyme-Glutamate Pyruvate Transaminase(GPT) and Glutamate oxaloacetate Transaminase (GOT), which shows the destructive effects of mercury in the organs. The study also shows a statistically significant decrease in the destructive effects as a result of the administration of *Solanium lycopersicum.*

Key words : Bioaccumulation, Mercury poisoning, *Solanum lycopersicum*, Liver enzymes.

INTRODUCTION

With increasing industrialization, there is an increased release of toxicants including heavy metal into the environment, with subsequent effects on the public health.

Methyl mercury (MeHg) intoxication has been a public health problem for many decades (WHO 1990). Mercury is naturally occurring element, existing in multiple forms and in various oxidation states. It is used in a wide variety of products and processes. Exposure to mercury can occur in both occupational and environmental setting, the later primarily dietary exposure (ATSDR, 1989).

The most famous methyl mercury poisoning cases occurred in Minamata and Niigata in Japan in the 1950-1960s, where the consumption of methyl mercury contaminated sea food caused the death or incapacitation of many people (Takeuchi,1998). The neuropathy caused by this is named Minamata disease. Methyl mercury is known to pass through the placental barriers and produce many deleterious effects on the unborn fetus (Mottet, 1987). Some evidence suggests that mercury accumulates on the fetal side of the placenta resulting in higher concentration in the fetus (Viny et al.,1990) and accumulates in the brain(Choi, 1989),this results to cerebral palsy, mental retardation , low birth weight and early sensorimotor dysfunction (Gilbert and Grant-Webster ,1995).

Organic mercury is readily distributed throughout the body but tends to concentrate in the brain and kidneys (ATSDR,1989;Goyer, 1991).Mercury is known to bind to microsomal and mitochondrial enzymes resulting in cell injury and death .Mercury in renal cells localizes in lysosomes (Madsen and Christensen, 1978).

A report from Dutclak et al (1991) indicates that extensive absorption of methyl mercury occurs in the gall bladder and subsequent biliary-hepatic cycling of the compound contributes to its long biologic half life.

Mercury is not destroyed by metabolism, but rather converted to different forms and oxidation state (ATSDR 1989) and involves an oxidation-reduction cycles. The primary route of its excretion is urine and faeces (ATSDR 1989).This may explain the renal damage and failure associated with its toxicity (Yasutake et al., 1990 ;Moreira and Moreira, 2004).

To lower the bioaccumulation of mercury in organs in the body chelating agents are used, a well known example is DMPS(2,3- dimercapto-1-propanesulphurnic acid) .Repeated use slowly lower the level of methyl mercury in the brain but had little effect on inorganic mercury (Pinegree *et al,* 2001)

Solanum lycopersicum commonly called" Tomatoes" is a plant in the solanaceae family. It is native to central and south America .Tomatoes is one of the best sources of lycopene. It is the nutrient that gives it the red color. Chemically, lycopene is a bioflavoniod , closely related to beta carotene. It is one of the most important antioxidant (Ihesie, 2008). It helps strongly in neutralizing free radical and therefore protect cells and tissues from degenerative cancerous change.It also enhances the functional capacity of the liver. It is very useful in the treatment and prevention of prostate, breast, ovarian and cervical cancers (Ihesie, 2008)

Therefore this study is designed to find out the bioaccumulation of mercury in the kidney and liver of wistar rats and possibly the detoxification of the effect by the use of *Solanum lycopersicum.* The result of this study will help in ameliorating the increasing effect of mercury poisoning in our environment.

MATERIAL AND METHODS

Twenty- four rats of the wistar strain were purchased from the animal house unit of College of Health Sciences, Delta State University, Abraka. Nigeria The rats weighed between 150g-180g.They were about 9-10 weeks old. The rats were kept in standard environmental conditions and allowed two weeks to acclimatize. The rats were randomly divided into four experimental groups of six rats in each group.

Group A (control) were given clean drinking water ad libitum and rat chow.

Group B were given rat chow +10% by mass of *Solanium lycopersicum*(sl).

Group C were given rat chow +mercury in water at a concentration of 10ppm.

Group D were given rat chow +mercury in water at a concentration of 10ppm +10% by mass of *Solanium lycopersicum*(sl)

Preparation of tomatoes

Riped tomatoes were bought from a local market in Warri, Delta State. Nigeria. It was rinsed to remove debris. It was milled and moderately dried in an oven to allow for good mixing with the rat chow. The required quantity was calculated and administered orally.

Collection of samples

The rats were sacrificed by decapitation after an overnight fast. The whole blood sample obtained was centrifuged at 1,200xg for 5 minutes at room temperature. The sera were used to analyze the liver enzymes within 1 hour of collection.

The kidney and liver were dissected out trimmed to remove fats and processed for mercury content. Local ethical committee gave approval for the handling and method of sample collection.

Mercury bioaccumulation analysis

The kidneys and liver were separately dried at 60^0 c and pulverized into a uniform particle size prior to analysis. Each component of the dried kidney and liver were weighed out and digested in a conical flask with mixed acid prepared from 2ml HCLO and 20ml HNO_3 (Ney, 1983).

The resultant solution in the conical flask were placed on hot plate with constant stirring before they were transferred into the fume cupboard and allowed to stand over night. On cooling, the mixture was filtered and the filtrate was made up to 100ml in a volumetric flask with deionised water. The

solution were analyzed for mercury (as recommended by American Chemical Society, 1988). All acids and reagents were of analytical grade. All samples were analyzed in triplicate and coefficient of variation was less than 5%

Liver enzyme analysis

The serum was used for the analysis of Alkaline phosphatase(ALP), Glutamate oxaloacetate transaminase(GOT), and Glutamate pyruvate Transaminase (GPT). This was done using atomic absorption mass spectrophotometer as recommended by International Federation of Clinical Chemistry (IFCC).

Statistical analysis

All statistical analysis was done using the computer package SPSS-pc (version 7.5).

RESULTS

The results obtained are as shown in the tables below.

Table 1: Liver Enzymes analysis at the end of the experiment

Parameter	GP A n=6 (control)	GP B n=6 Rat chow +10% mass of sl	GP C n=6 Rat chow +Hg at conc of 10ppm	Gp D n=6 Rat chow +Hg at concof 10ppm +10% mass of Sl
GPT µ/l	2.10± 0.09	2.20 ± 0.50	4.05 ± 0.05*	2.30 ± 0.04
GOT µ/l	1.85± 0.04	1.90 ± 0.29	4.47 ±0.14*	2.70 ± 0.60
ALP µ/l	4.45± 0.14	4.55 ± 0.90	4.60 ± 0.22	4.40 ±0.20

Data are expressed as mean ± SEM

GPT =Glutamate pyruvate transaminase. GOT=Glutamate oxaloactate Transaminase.

ALP=Alkaline phosphatase; sl= Solanum lycopersicum; Hg =mercury; ppm = parts per million.

**statistically significant*.

Table 2: Bioconcentration of mercury in the kidneys and liver

Parameter	GP A n=6 (control)	GP B n=6 Rat chow +10% mass of sl	GP C n=6 Rat chow +Hg at conc of 10ppm	Gp D n=6 Rat chow +Hg at conc of 10ppm +10% mass of Sl
KIDNEYS(mg/kg	0.01± 0.04	0.01 ± 0.01	0.36 ± 0.15*	0.14± 0.11
LIVER (mg/kg)	0.04 ± 0.02	0.03 ± 0.02	0.96± 0.18	0.37 ± 0.21

Data are expressed as mean ± SEM. Hg =mercury; sl =Solanum lycopersicum ; ppm =parts per million

DISCUSSION

Table 1 shows the changes in the liver enzymes level at the end of the study.The result shows a statistically significant (P<0.05) in Glutamate pyruvate Transaminase (GPT) and Glutamate oxaloacetate Transaminase (GOT) in Group C (fed rat chow +Hg at a concentration of 10ppm).There was no statistically significant change in Group D (fed rat chow +Hg at concentration of 10ppm + 10% mass of *Solanum lycopersicum*) There was also no statistically significant difference in Alkaline phosphatase in all the groups. This is consistent with the finding of (UNEP 2002) that reported hepatic impairment. This study therefore shows that a degenerative/necrotic change occurs

with the administration of mercury and this effect could be reduced/ ameliorate by the administration of *Solanium lycopersicum* as shown in Group D.

Table II shows the bioconcentration of mercury in the kidney and liver. The study shows a statistically significant increase ($P<0.05$) in the concentration of mercury in the kidney and liver as shown in Group C and an increase in the values in Group D but not statistically significant. There is a dearth of information on this aspect of the report. But this study shows that mercury accumulates in the organs studied with a progressive distruction of its primary function. The study also shows a decrease in bioaccumulation of mercury as a result of administration of *Solanium lycopersicum*.

CONCLUSION

The study has shown the degenerative effect of mercury toxicity in the organs studied and the ameliorating effect of *Solanum lycopersicum*. However a histopathological study of the organ is recommended for further knowledge on the extent of tissue damage.

ACKNOWLEDGEMENTS

The author wish to acknowledge the staff of Petroleum Training Institute, Effurum for doing some of the analysis in their laboratory, and to Kenneth Mutoh for the preliminary work on this subject matter.

REFERENCES

1. Agency for Toxic Substances and Disease Registry (ATSDR) Toxicological (1989).
2. Choi, BH. The effect of methyl mercury on the developing brain. *Progress in Neurobiology*, **32**: 447-470(1989)
3. Dutczak, W., Clarkson, TW., and Ballatori, N., Biliary-hepatic recycling of a xenobiotic: gall bladder absorption of methyl mercury. *Amer. J.Physiol.* **260**: 273-288(1991).
4. Gilbert, SG., Grant-Webster, K. Neurobehavioural effects of development in methyl mercury exposure.Environ Health perspect 103 (supp 16): 135-142 (1995).
5. Goyer R. Toxic effect of metals. In; Amdor, M (eds)Doull J. et al., Cognitive performance of children prenatally exposed to safe level of methyl mercury. *Environ.Res* **77**: 165-175 (1991) .
6. Ihesie, G. Food that fight and prevent cancer. *Vangard* **13**: (609) :Pp 47 (2008).
7. Kingston,HM., and Jassie, LB : Introduction to microwave sample preparation. American Chemical Soceity Washington DC 35-51 (1988).
8. Madsen, KMM., and Christensen, E.F: Effects of mercury on lysosomal protein digestion in the kidney proximal tubule. *Invest* **38**:165-171 (1978).
9. Mottet,NK.,Shaw, CM., Bubacher, TM. Health risk from increase in methyl mercury exposure. *Environ. Health perspect*. **63**: 133-140 (1985).
10. Ney, J.I., and Van Hussel, JH., Sourcs of variability in accumulation of heavy metals by fish in a road side stream.*Arch.Environ. Contam. Toxicol.* **12**: 701-706 (1983).
11. Pinegree, SD., Simmonds, PL., Wood, JS. Effect of 2,3-dimercapto-1-propane sulfonic acid (DMPS) on tissue and urine mercury levels following prolonged methyl- mercury exposure in rats. *Toxicol. Sci.* **61**(2): 224-233 (2001).
12. Takeuchi, T., (1968) In: Minamata disease (M.Kutsama eds) Kumamoto univ. press Kummato, Japan Pp141-228.(1968).
13. UNEP. Global mercury assessment, United Nations, Effect of inorganic mercury on in vitro placental nutrients transfer and oxygen consumption. *Reprod.Toxicol* **6**: 69-75.(2002)
14. Viny, MJ., Takahashi,Y.,Orsheider et al.,:The Transport of elemental mercury into fetal tissues. *Biology of neonates.* **21**:239-244.(1972)
15. World Health Organisation (WHO). Methyl mercury, *Environ.Health crit.* **101**: 144(1990).