World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

Check for updates

Mercury retention after *Panax ginseng* treatment against mercuric chloride intoxication in hepato-haemato indices in albino rats

Kanhiya Mahour *

Department of Zoology, Experimental Laboratory, R. P. P. G. College Kamalganj, Farrukhabad (U.P.)-209724, Affiliated to CSJM University, Kanpur-282002 (U.P.), India.

World Journal of Biology Pharmacy and Health Sciences, 2023, 13(02), 345–349

Publication history: Received on 16 January 2023; revised on 25February 2023; accepted on 28 February 2023

Article DOI: https://doi.org/10.30574/wjbphs.2023.13.2.0113

Abstract

Mercuric chloride was introduced per Os in albino rats (*Rattus norvegicus*) as per the LD_{50} (9.26 mg/kg b.w.). The assessment of mercuric chloride toxicity was done both after acute (0.926 mg/kg b.w.) and sub-acute (0.033 mg/kg b.w.) per Os treatment, while *Panax ginseng* was also introduced (10 mg/kg b.w.) per Os in the albino rats separately. Mercuric chloride treatment significantly increases mercury retention in liver and blood serum along with increase in liver weight, while *Panax ginseng* alone caused significant decrease in liver weight and mercury retention in liver and blood. Increase in mercury retention in blood serum and liver is due to the reactivity between –SH protein of blood serum and liver with oxidized form of mercury (Hg^{++}), while decrease in *Panax ginseng* treatment is due to antioxidant activity of *Panax ginseng* the ginsenosides. Moreover, mercuric chloride followed by *Panax ginseng* and *Panax ginseng* followed by mercuric chloride treatment revealed significant modulation for raised mercury retention and liver weight towards normal level along with blood serum and liver changes. The results suggest a modulating role of *Panax ginseng* extract against raised mercury concentration in blood serum and liver under stress of mercuric chloride.

Keywords: Mercuric chloride; *Panax ginseng*; Serum; Hepatic; Enzyme; Serum

1. Introduction

The liver and blood are the two important vital organs for assessing effects of xenobiotic substances. Mercuric chloride, widely used in industrial purposes, poses a threat to the mankind an account of its level which gets in the environment exceeded as mercury is such a heavy metal which has been found to be injurious in all three forms, elemental, inorganic and organic.

Many metabolic disorders in man are accompanied by alterations in the concentration of one or more trace elements in some body fluid, especially blood serum or plasma [1].

Mercuric chloride is an inorganic compound that has been used in agriculture as fungicides, in medicine as tropical antiseptic and disinfectant and in chemistry as an intermediary in the production of other mercury compounds.

Industrial use of mercury and its toxic effects on human as well as animal systems are well documented [2], Neurotoxic, nephrotoxic and reproductive effects of mercury are known since long time and documented clinically and experimentally [3-7].

Hence, this study was undertaken to evaluate mercury concentration in liver and blood serum with mitigate role of *Panax ginseng* against mercuric chloride intoxication in albino rat.

*Corresponding author: Kanhiya Mahour

2. Material and methods

2.1. Collection of extract

The plant root powder was gifted by Prof. A. Kumar, Cancer & Radiation Biology Laboratory, department Of Zoology, Rajasthan University, Jaipur (Rajasthan).

2.2. Experimental animals

Rattus norvegicus weighing approximately (120-130) gm of both the sexes were procured from inbreed colony. The rearing of animals as well as experiment was approved by the ethics committee of department of Zoology, Dr. B. R. A. University, Agra (U.P.) India. The animals were fed with a standard balanced diet and water was provided *ad libitum.*

2.3. Experimental compound

Experimental compound (mercuric chloride) was obtained from Bayer India Ltd. Bombay while other chemicals were procured from Sigma chemical, Germany and SRL, Mumbai.

2.4. Experiment group

The acute oral LD_{50} was determined on albino rats. The data were analyzed by probit analysis for LD_{50} determination (Table-1).

Table 1 Toxicity evaluation of Mercuric Chloride in albino rat, *Rattus norvegicus* Specifying fiducial limits

Experimental individual	Compound	Regression equation	LD50 (mg/kgb.w.)	Variance	Fiducial limits
Rattus norvegicus	Mercuric Chloride	Y=5.146+3.410 (x-1.009)	9.26 mg	0.006	m1=(±)0.972 m2=(±)0.960

Animals were divided into 5 groups of 5 rats each. Group I received 1 ml of distilled water and 10 µl tween-20, group II received *Panax ginseng* (10 mg/kg b. wt), group III received mercuric chloride after LD_{50} (9.26 mg/kg b. wt.) determination for acute (0.926 mg/kg b. wt.) and sub-acute (0.330 mg/kg b. wt.) sets, group IV received *Panax ginseng* followed by mercuric chloride while, group V received mercuric chloride followed by *Panax ginseng* (Table-2, 3 and 4).

Table 2 liver weights in gms/100 gm of body weight (Gms) after successive treatment of various substances

Treatment duration	Treatment sets	Control	Mercuric chloride treated	*Panax ginseng* treated	Mercuric chloride followed by *Panax ginseng*	*Panax ginseng* followed by mercuric chloride
6 hrs	Acute	3.30±0.051*	3.38±0.008^{b}	3.19±0.005^{c}	3.34±0.003^{a}	3.29±0.003^{a}
12 hrs		3.21±0.02*	3.21±0.005^{a}	3.20±0.01^{c}	3.19±0.00^{a}	3.17±0.01^{a}
24 hrs		3.19±0.02*	3.28±0.008^{c}	3.10±0.008^{c}	3.21±0.01^{a}	3.18±0.00^{a}
7 days	Sub acute	2.52±0.02*	2.85±0.261d	3.21±0.00^{c}	3.22±0.01^{a}	3.17±0.01^{a}
14 days		3.02±0.18*	3.12±0.008^{d}	2.96±0.008^{c}	3.30±0.003^{b}	3.00±0.008^{a}
28 days		3.06±0.16*	3.58±0.01^{d}	2.13±0.008^{c}	3.30±0.003^{a}	3.02±0.008^{b}

Abbreviation used: *=Mean ± S.Em. , Student's'; a=>0.05; b=<0.05; c=<0.01; d=<0.001;

Table 3 Mercury levels (ng/mg tissue) in liver of *Rattus norvegicus* after acute and sub-acute treatment (Table-3)

Treatment	Sets	Treatment duration (Mean± S.Em.)					
		6 hrs	12 hrs	24 hrs	7 days	14 days	28 days
Mercuric chloride	Control	0.0163±0.003*	0.0153±0.003*	0.0163±0.003*	0.0157±0.007*	0.0163±0.007*	0.0177±0.007*
	Treated	0.2953±0.003[d]	0.3013±0.007[d]	0.3223±0.001[d]	0.3670±0.001[d]	0.9113±0.007[d]	0.9963±0.009[d]
Panax ginseng	Control	0.0157±0.003*	0.0150±0.000*	0.0163±0.003*	0.0163±0.009*	0.0170±0.006*	0.0180±0.006*
	Treated	0.0147±0.003[a]	0.0147±0.003[a]	0.0187±0.003[a]	0.0147±0.003[a]	0.0137±0.009[a]	0.0117±0.003[a]
Mercuric chloride followed by *Panax ginseng*	Control	0.0163±0.003*	0.0153±0.003*	0.0167±0.007*	0.0160±0.006*	0.0167±0.003*	0.0177±0.007*
	Treated	0.1713±0.009[a]	0.1763±0.007[a]	0.1852±0.015[a]	0.1790±0.012[a]	0.1783±0.009[a]	0.1407±0.012[a]
Panax ginseng followed by mercuric chloride	Control	0.0177± 0.007*	0.0167±0.012*	0.0170±0.001*	0.0163±0.013*	0.0183±0.007*	0.0177±0.007*
	Treated	0.0307±0.007[c]	0.0373±0.012[d]	0.0420±0.012[d]	0.0377±0.015[d]	0.0293±0.009[d]	0.0313±0.009[d]

Table 4 Mercury levels (pg/ml) in blood serum of *Rattus norvegicus* after acute and sub-acute treatment (Table-4)

Treatment	Sets	Treatment duration (Mean± S.Em.)					
		6 hrs	12 hrs	24 hrs	7 days	14 days	28 days
Mercuric chloride	Control	0.0163±0.003*	0.0153±0.003*	0.0163±0.003*	0.0157±0.007*	0.0163±0.007*	0.0177±0.007*
	Treated	0.2953±0.003[d]	0.3013±0.007[d]	0.3223±0.001[d]	0.3670±0.001[d]	0.9113±0.007[d]	0.9963±0.009[d]
Panax ginseng	Control	0.0157±0.003*	0.0150±0.000*	0.0163±0.003*	0.0163±0.009*	0.0170±0.006*	0.0180±0.006*
	Treated	0.0147±0.003[a]	0.0147±0.003[a]	0.0187±0.003[a]	0.0147±0.003[a]	0.0137±0.009[a]	0.0117±0.003[a]
Mercuric chloride followed by *Panax ginseng*	Control	0.0163±0.003*	0.0153±0.003*	0.0167±0.007*	0.0160±0.006*	0.0167±0.003*	0.0177±0.007*
	Treated	0.1713±0.009[a]	0.1763±0.007[a]	0.1852±0.015[a]	0.1790±0.012[a]	0.1783±0.009[a]	0.1407±0.012[a]
Panax ginseng followed by mercuric chloride	Control	0.0177± 0.007*	0.0167±0.012*	0.0170±0.001*	0.0163±0.013*	0.0183±0.007*	0.0177±0.007*
	Treated	0.0307±0.007[c]	0.0373±0.012[d]	0.0420±0.012[d]	0.0377±0.015[d]	0.0293±0.009[d]	0.0313±0.009[d]

2.5. Estimation of mercury level

The rats were sacrificed and liver was excised out immediately. Mercury levels were estimated by acid digestion method [8]. A known amount of tissue was weighted and digested in a mixture of perchloric acid, conc. nitric acid and sulphuric acid. The digested sample was cooled, transferred to volumetric flask and diluted to a required volume. Digested sample was analyzed by making use of the cold vapour atomic absorption spectrometry technique, while blood serum mercury concentration was estimated from 20 time's diluted serum with AAS. Body and liver weight were also recorded.

Statistically significant values between experimental and control values were calculated according to Fisher's student's' test [9, 10].

3. Results and discussion

3.1. LD50

LD_{50} of mercuric chloride calculated in the present investigation has been 9.26 mg/kg b.w. and is in accordance to Mark *et al.* [11] who observed LD_{50} as 8.26 mg/kg b. w. against mice.

3.2. Hepatic weight

Liver weight were significantly ($p<0.001$) increased in acute and sub-acute mercuric chloride treated groups with respect to 100 gms of body weight. It was significantly ($p<0.01$) decreased in *Panax ginseng* treated groups. It was increased non-significantly in mercuric chloride followed by *Panax ginseng* treated sets, decreased non- significantly in *Panax ginseng* followed by mercuric chloride treated set. (Table-2)

Increased liver weight in mercuric chloride treated groups may be due to hepatic fibrogenesis, it was decreased in *Panax ginseng* treated group probably due to their antioxidant potential. In mercuric chloride followed by *Panax ginseng* and *Panax ginseng* followed by mercuric chloride sets, liver weight was decreased in comparison to the mercuric chloride treated set may probably be due to release of free radicals of mercuric chloride. These free radicals behave like electron accepter (lone pair of electron).

3.3. Hepatic mercury concentration

Mercury concentration in liver were significantly increased in acute and sub-acute mercuric chloride treated groups with respect to control, it was decrease none significantly in *Panax ginseng* treated groups. It was decreased in mercuric chloride followed by *Panax ginseng* sets; significantly decrease in *Panax ginseng* followed by mercuric chloride treated sets (Table-3).

Increased mercury concentration in liver in mercuric chloride treated set is due to binding capacity of mercury with –SH groups of protein [3, 5, 6, 12]. It was decreased in *Panax ginseng* treated group probably due to their antioxidant potential [7, 13]. In mercuric chloride followed by *Panax ginseng* and *Panax ginseng* followed by mercuric chloride sets, mercury concentration was decreased significant in *Panax ginseng* followed by mercuric chloride treated set should be due to release of Hg^{++} of mercuric chloride. These Hg^{++} bind with free radicals of *Panax ginseng* (electron accepter) and minimized mercury toxicity [7].

3.4. Blood serum mercury concentration

Mercury concentration in blood serum was also increased significantly in acute and sub- acute mercuric chloride treated groups with respect to control. It was decrease significantly in *Panax ginseng* treated groups. It was decrease non-significant in mercuric chloride followed by *Panax ginseng* sets however, significant decreased in *Panax ginseng* followed by mercuric chloride set (Table-4).

Increased mercury concentration in serum in mercuric chloride treated set is due to readily binding capacity to RBCs of blood [5, 6, 14, 15, 16], while in all others groups it should be *vide supra*.

4. Conclusion

Comparing all data of liver weight, mercury concentration in liver and blood serum suggested that concentration of mercury minimized by *Panax ginseng* in all respective sets expect mercuric chloride treated set. Hence *Panax ginseng* is having modulating effect in liver and blood serum against mercuric chloride intoxication in albino rats.

Compliance with ethical standards

Acknowledgments

The author is very thankful to Late Prof. P. N. Saxena, former Dean of School of Life Sciences, Khandari campus, Dr. B.R. Ambedkar University Agra U. P. and Prof. Ashok Kumar Ex Vice chancellor of Kanpur and Gorakhpur University for valuable suggestions and support.

Statement of ethical approval

The experimental rats received human care in compliance with the *Guide for the Care and Use of Laboratory Animals* prepared by the National Academy of Sciences and published by the National Institute of Health.

References

[1] McCall J T, Glodstein, N P and Smith L H. Implication of trace metals in human diseases. Fed. Thoc. 1971, 30:1011

[2] WHO. Environmental Health Criteria 118: Inorganic Mercury Environmental Aspects. World Health Organization, Geneva, 1991.

[3] Rao, M V. Mercury and its effects on mammalian systems- A critical review. Ind. J. Envi. Biol. Toxicol., 1997, 7:3-11.

[4] Gayatri R. P. Studies on the influence of some therapeutic agents on mercuric chloride toxicity in histophsiology of the mouse vital organs. Ph. D. thesis, Gujarat University, Ahmedabad, 1998.

[5] Bablola O O, Okomji R E, Atoyebi J O, Senneya T F, Raimi, M M, Ejim-Eze E E, Adeniran O A, Akinsiku O T, Areda J O, John O O and Odebunmi S O. Distribution of lead in selected organs and tissues of albino rats exposed to acute lead toxicity. Scientific research and essay, 2014, 5(9):845-848.

[6] Lakshmi, B V S, Sudhakar M and Nireesha G. Modification of mercury induced biochemical alterations by Triticumaestium Linn in rats. Ind. J. Physiol. Pharmacol. , 2014, 58(4):423-436.

[7] Saxena, P N, Goyal R and Bhushan B. Modulation of mercuric chloride nephrotoxicity in albino rat by flower extract of *Tagetes erecta*. J. Pharmacognosy and phytochem, 2020, 9(1):17-26.

[8] Subramanian, R, Muhantha, A and Panda S. Manual for estimation of selenium and mercury in biological materials. Jawaharlal Nehru University, New Delhi, 1995.

[9] Fisher, R A and F Yates. Statistical methods for research workers, 12th ed. Pp.365 Oliver and Boyd. Edinburgh, 1950.

[10] Finney D J. Probitt analysis. Cambridge University press, pp. 303 (1971).

[11] Mark M J, Jean E S and Anthony D W. Pretreatment and heavy metal LD_{50} values. Toxicol. Appl. Pharmacol. (1979) 49:41-44.

[12] Flora G J S, Mature R, Sandhu N and Dua K K. Time dependant preventive effects of simultaneous administration of selenium and zinc during mercury exposure in rats. Ind. J. Pharmacol. (1994) 26: 209-212.

[13] Sakakibara K, Shibata Y, Higashi T, Sanada S and Shoji J. Effect of ginseng saponin on cholesterol metabolism. I. The level and the synthesis of serum and liver cholesterol in rats treated with ginsenosides. Chem. Pharm. Bull. (1975) 23:1009-1016.

[14] Rao A N. Trace element estimation. J. Health. Allied. Sci. (2005) 4(1):1-9.

[15] Rao M V, Patel J S and Mehta A R. Effect of ascorbate on methyl mercury induced toxicity in guinea pigs. J. Anim. Morphol. Physiol. (1994) 41: 39-41.

[16] Singh K, Khanna A K and Chander R. Hepatoprotective activity of ellagic acid against carbon tetrachloride induced hepatotoxicity in rats. Ind. J. Exp. Biol. (1999) 37: 1025-1026.

[17] Nasr SE, Moustafa MHA, El-Azeen WA, Boughdady A and Yousry MM. A histological study of the effect of *Panax ginseng* versus Finasteride on induced benign prostatic hyperplasia in rats with potentiality of spontaneous recovery. Egyption Journal of Histology, 2020, 44(3):587-599. doi: 10.3390/biom10030385

[18] Abdel Dayem DAM, Mouhmamd AS, Ali AH, El-Tahawy NFG. Ginseng ameliorates effect of prolonged use of omeprazole in rat renal cortex though decrease inflammation, fibrosis and apoptosis. bioRxiv. doi.org/10.1101/2020.01.22.915587

[19] Sung-Min nam, Sun-Hye Choi, Hee-Jung Cho, Jin SeokSeo, Minsuk Choi, Sang soepNahm, DyungJoon Chang and Seung-Yeol Nah. Ginseng Gintonin attenuates lead induced rat cerebellar impairments during gestation and lactation. Biomolecules, 2020, 10(3); 385-394. doi: 10.3390/biom10030385