Journal of Applied Veterinary Sciences, 10 (2): 137 - 143 (April, 2025).
ISSN: Online: 2090-3308, Print: 1687-4072
Journal homepage : https://javs.journals.ekb.eg

Histopathological Study of The Acute and Chronic Toxic Effects of Dimethyl Mercury on Liver and Kidney of Male Albino Rats

Semaa Ahmad Baker[1*], Semaa Sami Al-Modaress[2] and Karam Hashim Al-Mallah[3]
[1]Department of Biology, College of Education for Pure Sciences, University of Mosul, Iraq
[2]Department of Biology, College of Basic Education, University of Mosul, Iraq
[3]Department of Pathology and Poultry disease, College of Veterinary Medicine, University of Mosul, Iraq
***Corresponding Author:** Semaa Ahmad baker, E-Mail : **semaaabakir123@uomosul.edu.iq**

ABSTRACT

For the purpose of evaluating the harmful effect of di-methyl mercury, the well-known pollutant of the aquatic ecosystem, on liver and kidney of mammals with its both short- and long-term toxicity models, the goal was achieved by using twenty-four mature albino male rats, aged between 10 and 12 weeks, which were divided into 3 groups (8 rats for each). These included a control group, group 1 for chronic toxic effects (treated with a 2.5 mg/kg daily dose of di-methyl mercury for 30 days), and group 2 for acute toxic effects (treated with a 5 mg/kg daily dose of di-methyl mercury for 15 days). Four animals of each group were euthanized, and the others were kept to be sacrificed after 30 days as a recovery period. The sections were prepared from liver and kidneys, stained and examined to record and to grade the intensity of histopathological changes. Liver sections elucidated mild to moderate pathological changes in both treated groups, including vacuolar degeneration of hepatocytes, foci of coagulative necrosis, and inflammatory cell deposition mainly composed of lymphocytes, plasma cells and macrophages in peri-portal and Peri-central and peri-sinusoidal areas. Also, the vascular reaction, as hyperemic portal arteries, congested sinusoids and central veins, was present with Kupffer cell proliferation. Cholangitis or cholangiohepatitis were observed in 2.5 and 5 mg methyl mercury-treated groups as a solitary case at the end of exposure or after the recovery period. The examination of the kidney sections explored adverse changes manifested by vascular congestion, cloudy swelling of the renal tubular epithelium, protein cast in the renal tubules, interstitial nephritis and multifocal hemorrhage at the cortex and medulla; these changes were apparent at both acute and chronic models of toxicity. It was clear that the recovery period was not enough to exclude or reverse the toxic effects of methyl mercury because the same changes were present at the end of the recovery period.

Keywords: Albino Rat, Dimethyl mercury, Histopathology, Kidney, Liver.

Original Article:
DOI: 10.21608/javs.2025.364375.1544
Received : 27 February, 2025.
Accepted: 26 March, 2025.
Published in April, 2025.

J. Appl. Vet. Sci., 10(2): 137-143.

INTRODUCTION

It is well known that Methyl mercury is the most toxic substance between compounds containing mercury, it formed when the inorganic mercury solved in the aquatic environment of lakes, rivers and oceans. It is usually condensed through passing food chain reaching to the human consumer **(Kim *et al.*, 2006).** The mercury converts to methyl mercury by the aquatic bacteria and transmitted through the aquatic animals **(Lee *et al.*, 2006)**. The rate of absorbance of inorganic mercury is not exceeding 2%-38% while the organic mercury being completely absorbed **(Abernethy *et al.*, 2010).**

Mercury in nature is available in either an organic or in organic compounds. The organic compounds can be classified to allyl mercury and alkyl mercury, and the later includes the methyl and ethyl mercury compounds and the methyl mercury considered the most dangerous on the public health than ethyl mercury that is commonly used in vaccines **(Kim *et al.*, 2006).** It is mentioned by **Kjellstrom *et al.*, (1982)** that Minamata disease in japan is recored to be caused by consuming sea food including fish and oysters that is polluted with either monomethyl or dimethyl mercury. a famous acute toxic episode was appeared in Japan through fifties and in Iraq through seventies of the last century with methyl mercury which resulted in a heavy neurologic disturbance in the exposed local citizens **(Vicente *et al.*, 2004).** Although such toxic crises were never be recorded after that but the heavy usage of methyl mercury in the mining and industrial activities raises concerns about the hazards on human and animal health **(Castoldi *et al.*, 2008).**

When the polluted feeds being consumed the methyl mercury are separated by the gastric acids and binds to cysteine in duodenum to be absorbed completely and pass through the portal vein to the circulation and bind to the hemoglobin in red blood cells, also accumulated in the central nervous system and causes neuronal disorder **(Lee *et al.*, 2006).** It is also recorded by **Cheuk and Wong, (2006)** that methyl mercury may exert its toxic effects through absorbance by lungs through inhalation. In a study on the retina of fish *Danio rerio*, it was noted that there are abundant mercury deposits in the photoreceptor cell layer (the outer and inner parts of the photoreceptor cell layer), in the inner and outer nuclear layers, in the plexiform layer, and very rarely in the ganglion cell layer **(Mela *et al.*, 2010).** In another study, swelling of mitochondria expansion of the plasma reticulum of cardiac cells and degeneration of cardiac muscle fibers were observed **(Su and Chen,1979).**

For all that, the present study was held to evaluate the possible toxicity of Methyl mercury in both acute and chronic forms on the histologic features of liver and kidney in rats and to estimate the possibility to heal from toxicity and restoring the normal histologic features after recovery period.

MATERIALS AND METHODS

Animals

Twenty-four mature albino male rats, aged between 10 and 12 weeks of age and weighing 250-275 gm, were gained from the animal house, College of veterinary medicine, University of Mosul, where the experiment was performed under the ethical approval license number UM. VET.2023.143.

Experimental design

The animals were divided into 3 groups (8 rats each); these included a control group drenched with 2 ml of distilled water daily, group 1 for chronic toxic effects treated with a 2.5 mg/kg daily dose of di-methyl mercury for 30 days **(Ajibade *et al.*, 2019)** and group 2 for acute toxic effects treated with a 5 mg/kg daily dose of di-methyl mercury for 15 days **(Fossato da Silva *et al.*, 2011).**

Four animals were euthanized by chloroform and cervical dislocation from each treated group at the end of the acute and chronic exposure groups. The other four animals from each group were kept without treatment for 30 days as a recovery period.

Histopathological examination

The euthanized rats were dissected and grossly examined for any abnormalities in liver and kidneys; samples from organs were harvested and preserved using 10% neutral buffered formalin. After a sufficient period of fixation, the samples were processed by dehydration, clearing, embedding, sectioning, staining and mounting **(Salah *et al.*, 2024)** then the tissue sections were examined for any abnormalities by using a light microscope with photographing using the digital AmScope camera.

Scoring system

The sections for each organ were examined with the routine histopathologic examination to record all the observed lesions. The visual determination of ordinal data from 4 digits (0, 1, 2, 3) for each lesion was prepared from scanning and photographing all the prepared sections for each organ separately, and a photo catalogue was set from 4 images, each one representing a single microscopic field with the same magnification. This catalogue included a score of 0 for a field with an undetectable lesion, a score of 1 for the field with mild presence, a score of 2 for the moderate presence, and a score of 3 for the severe presence of the specific lesion. Then 5 microscopic fields were randomly selected and examined at each section and scored for the specific lesion by examiner eye match with the previously prepared catalogue to get the score of the field as ordinal data (**Hussein *et al.*, 2023**).

The statistical analysis

The recorded data were analyzed by applying one-way analysis of variance and Duncan's test to detect differences between groups at the ($P \leq 0.05$) level of significance by using the SPSS program, version 19.

RESULTS

Liver sections

The microscopic examination of the liver sections revealed moderate to severe pathologic changes compared with the normal histological view of the sections from the control group. Those changes in the methyl mercury-treated group may reflect the state of toxicity. It is clearly noticed that most sections at both 2.5 mg and 5 mg methyl mercury treated groups revealed moderate to severe vacuolar degeneration of hepatocytes characterized by cell swelling with large fat droplets in cytoplasm, which were more severe at the peri-portal and peri-central zones of hepatocytes; this progressed to variable-size foci of coagulative necrosis at many sections.

These changes were accompanied by the inflammatory reaction manifested by inflammatory cell deposition. Mainly composed of lymphocytes, plasma cells and macrophages at peri-portal, Peri-central and peri-sinusoidal areas, also the vascular reaction as hyperemic portal arteries, congested sinusoids and central veins were present. Kupffer cell proliferation or hyperplasia was noticed at peri-sinusoidal spaces. Cholangitis, or inflammation of the bile ducts, was observed in the 2.5 and 5 mg methyl mercury-treated groups in solitary cases at the end of exposure or after the recovery period. It was clear that the recovery period was not enough to exclude or reverse the toxic effects

of methyl mercury because the same changes were present after the recovery period, even though they were less intense than during the exposure period (**Table 1 and Figs. 1, 2, 3, 4).**

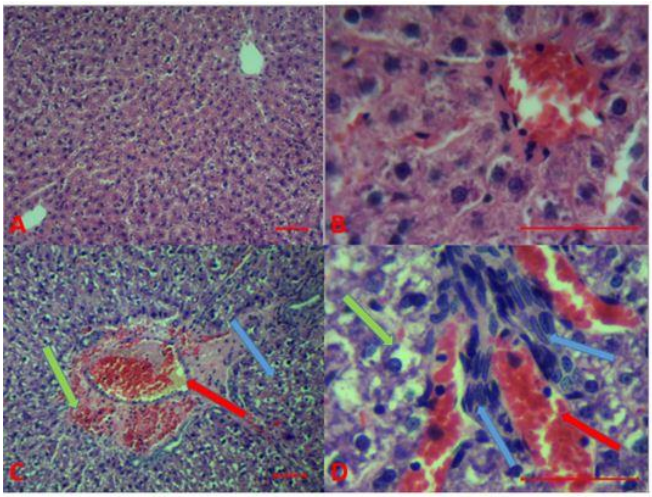

Fig. 1: Sections in liver of rats showing: A: Normal view of the hepatic tissue from control group. B: Moderate central vein and sinusoidal congestion from 2.5 Mg Methyl mercury treated group at the 30 days of experiment .C: Sever central vein congestion (Red arrow), a focus of peri-central hemorrhage (Green arrow) and Vacuolar degeneration of the hepatic cells (Blue arrow) at the 15th day of experiment in 5 mg methyl mercury treated group .D: Sever sinusoidal congestion (Red arrow), a moderate Kuepfer cell proliferation (Blue arrow) and vacuolar degeneration of hepatocytes (Green arrow) at 2.5 Mg methyl mercury treated group at 30 days of experiment. Staining H&E. Scale bar=100 µm for all images.

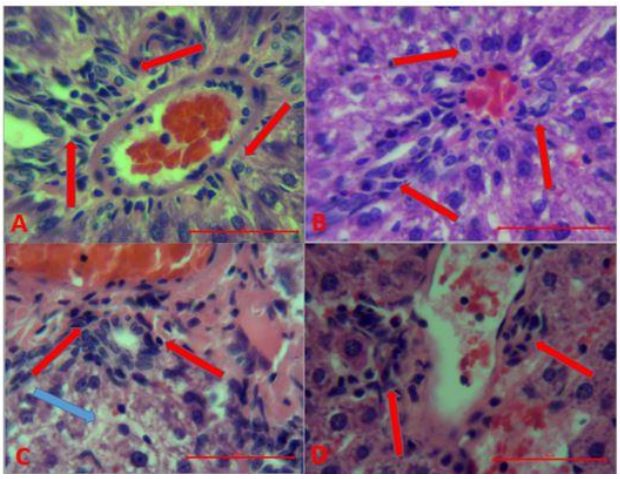

Fig. 2: Section in liver of rats showing peri-central or peri-sinusoidal inflammatory cells infiltrations composed mainly of lymphocytes, plasma cells and macrophages (Red arrows). 2.5 Mg Methyl mercury treated group. A: At the 30th day of experiment. B: At the end of the recovery period. 5 Mg Methyl mercury treated group. C: At the 15th day of experiment. B: At the end of the recovery period. Staining H&E. Scale bar=100 µm for all images.

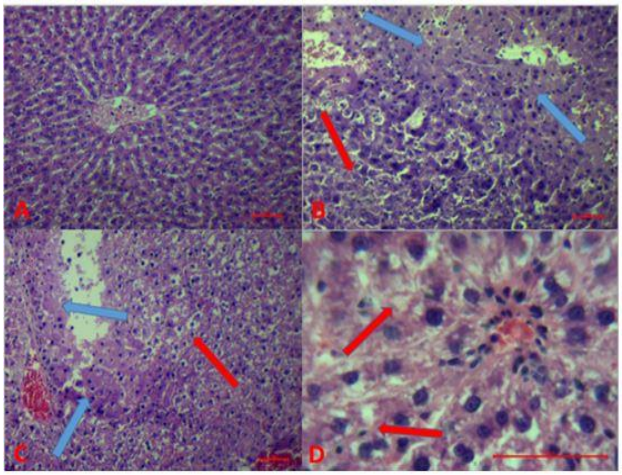

Fig. 3: Sections in liver of rats showing: A: Normal histological view of liver from control group. B: Sever vacuolar degeneration (Red arrow) and large focus of coagulative necrosis (Blue arrow) at 2.5 Mg Methyl mercury treated group at the day 30 of the experiment. C: Sever vacuolar degeneration (Red arrow) and large focus of coagulative necrosis (Blue arrow) at 5 Mg Methyl mercury treated group at the day 15 of the experiment. D: Sever to moderate vacuolar degeneration in hepatocytes at the end of the recovery period at 5 Mg Methyl mercury treated group. Staining H&E. Scale bar=100 µm for all images.

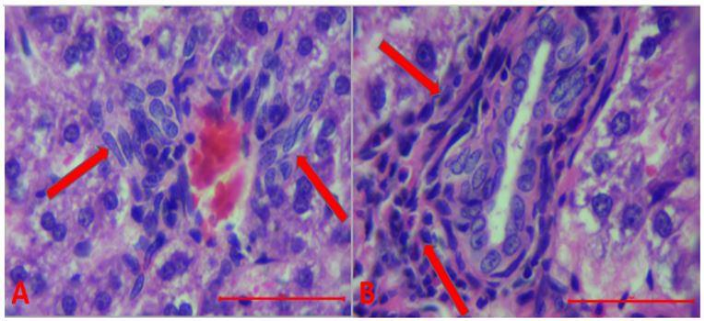

Fig. 4: Sections in liver of rats showing: A: Proliferation of Kuepfer cells around hepatic sinusoids (Red arrow). B: Cholangitis or inflammation of bile ducts (Red arrow) at 2.5 Mg Methyl mercury treated group at the day 30 of the experiment. Staining H&E. Scale bar=100 µm for all images.

Kidney sections

The examination of the kidney sections showed development of several pathological changes at the di-methyl mercury treated groups reflects vascular and metabolic disorders accompanied by inflammatory reaction , noticed at both treated groups with 2.5 and 5 mg di-methyl mercury with variable degree of intensity at the end of exposure period, leaving animals to recover appears to be not sufficient to reverse the adverse effects of the exposure period and the lesions still apparent in renal tissue with similar intensities, those lesions were expressed as hyperemic renal arterioles accompanied by perivascular edema and focal hemorrhages, the harmful effects on the tubular renal epithelium appeared as cloudy swelling and mostly associated with mononuclear inflammatory infiltrations as interstitial nephritis and even in glomeruli as glomerulonephritis. **(Table 2 and Figs. 5, 6, 7).**

Table 1: Histopathological changes appeared in the liver of rats exposed to dimethyl mercury with the score of severity.

Lesion / Group	Central /sinusoidal congestion	Pericentral /perisinusoidal inflammatory infiltrations	Periportal Inflammatory infiltrations	Hepatic vacuolar degeneration	Kuepfer cells proliferation	Hepatocytic coagulative necrosis	Cholangitis
Group 1 Control	0.50 ± 0.22^{B}	0.20 ± 0.13^{B}	0.40 ± 0.16^{B}	0.20 ± 1.33^{B}	0.50 ± 0.16^{B}	0.00 ± 0.00^{C}	0.00 ± 0.00^{A}
Group 2 2.5 M 30 Days	2.0 ± 0.25^{A}	1.80 ± 0.29^{A}	1.60 ± 0.33^{A}	1.40 ± 1.63^{A}	1.50 ± 0.26^{A}	1.00 ± 0.33^{AB}	0.40 ± 0.22^{A}
Group 3 2.5 M Recovery	$1.2\pm0.29A^{B}$	1.30 ± 0.30^{A}	1.30 ± 0.33^{AB}	1.30 ± 0.30^{AB}	2.00 ± 0.25^{A}	0.80 ± 0.20^{AB}	0.40 ± 0.26^{A}
Group 4 5 M 2 Weeks	$1.3\pm0.36A^{B}$	1.10 ± 0.31^{A}	1.20 ± 0.38^{AB}	1.60 ± 0.22^{A}	1.50 ± 0.37^{A}	1.20 ± 0.35^{A}	0.20 ± 0.20^{A}
Group 5 5 M Recovery	1.2 ± 0.39^{AB}	1.10 ± 0.31^{A}	0.90 ± 0.34^{AB}	1.50 ± 0.30^{A}	1.50 ± 0.30^{A}	0.40 ± 0.22^{BC}	0.10 ± 0.10^{A}

*The values expressed as Mean± Standard error.
**The letters compared vertically were A for superiority followed by B and C

Table 2: Histopathological changes appeared in the kidney of rats exposed to dimethyl mercury with the score of severity.

Lesion / Group	Renal vascular congestion	Cloudy swelling of renal tubular epithelium	Albumin urea /cast	Interstitial nephritis / inflammatory infiltrations	Cortical or Medullary hemorrhage
Group 1(Control)	0.40 ± 0.16^{B}	0.30 ± 0.15^{C}	0.10 ± 0.10^{C}	0.20 ± 0.13^{B}	0.20 ± 0.20^{B}
Group 2 (2.5 M 30 Days)	2.40 ± 0.26^{A}	2.30 ± 0.21^{A}	1.20 ± 0.32^{A}	1.70 ± 0.21^{A}	1.30 ± 0.26^{A}
Group 3 (2.5 M Recovery)	0.80 ± 0.24^{B}	1.20 ± 0.24^{B}	0.30 ± 0.15^{BC}	1.40 ± 0.30^{A}	0.40 ± 0.16^{B}
Group 4 (5 M2 Weeks)	2.30 ± 0.21^{A}	2.30 ± 0.21^{A}	1.0 ± 0.33^{A}	1.70 ± 0.26^{A}	1.60 ± 0.40^{A}
Group 5 (5 M Recovery)	1.00 ± 0.21^{B}	0.80 ± 0.24^{BC}	0.50 ± 0.22^{ABC}	1.30 ± 0.21^{A}	0.50 ± 0.22^{B}

*The values expressed as Mean± Standard error.
**The letters compared vertically were A for superiority followed by B and C.

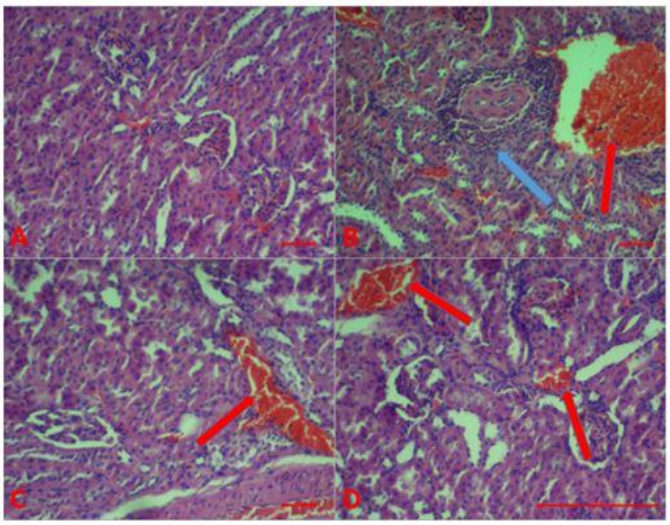

Fig. 5: kidney sections of rats showing: A: Normal section of renal tissue with normal glomeruli, normal tubular epithelium and normal circulation from control group. B: Hyperemic renal arteriole (Red arrow) with perivascular inflammatory infiltrations (Blue arrow) from 2.5 Mg methyl mercury treated group at the 30th day of experiment.C: Congested renal capillaries (Red arrows) at 5Mg methyl mercury treated group at the 15th day of experiment. D: Congested renal capillaries (Red arrows) at 5Mg methyl mercury treated group at the end of the recovery period. Staining H&E. Scale bar=100 µm for all images.

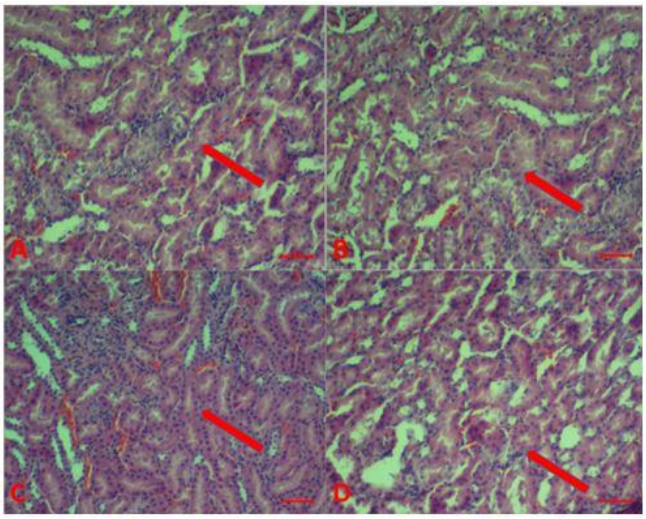

Fig. 6: kidney sections of rats showing: A :Cloudy swelling of the renal tubular cells at 2.5 Mg methyl mercury treated group (Red arrows) at 30th day of experiment. B: Cloudy swelling of the renal tubular cells (Red arrows) at 5 Mg methyl mercury treated group at 15th day of experiment. C: Cloudy swelling of renal tubular epithelium with hyaline casts with in renal tubules (Red arrows) at the end of the recovery period of 2.5 Mg methyl mercury treated group. D: Cloudy swelling with micro hemorrhages (Red arrows) at the end of the recovery period at 5 Mg methyl mercury treated group. Staining H&E. Scale bar=100 µm for all images.

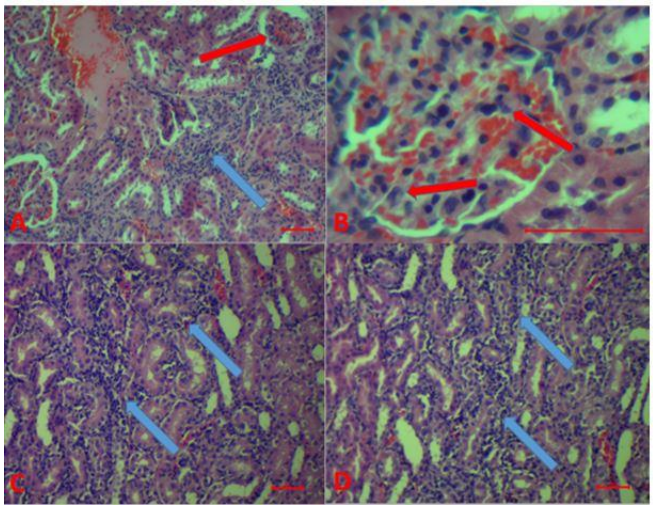

Fig. 7: kidney sections of rats showing: A: Glomerulitis (Red arrows) and interstitial nephritis (Blue arrows) at 2.5 Mg methyl mercury treated group at the 30th day of experiment. B: Magnified view of Glomerulitis (Red arrows) at 2.5 Mg methyl mercury treated group at the 30th day of experiment. C: Sever interstitial nephritis (Blue arrow) at 5 Mg methyl mercury treated group at the 15th day of experiment. D: Sever interstitial nephritis (Blue arrow) at 5 Mg methyl mercury treated group at the end of the experiment period. Staining H&E. Scale bar=100 µm for all images.

DISCUSSION

The results revealed that both acute and chronic oral doses of dimethyl mercury induced notable lesions in the liver and kidney. These findings are similar to the statements of many researchers who found harmful biological effects on the CNS, heart, lungs, kidneys and immune organs in people with high blood mercury concentration **(Myers *et al*., 2007).** Other researchers highlighted a harmful influence of methyl mercury as a water pollutant on aquatic creatures, aquatic birds, mammals and other predators seeking them **(Singh and Maurya, 2023).** In a study performed by **Mela *et al*., (2007)** who stated that adding 0.075 µg of methyl mercury per gram of wet weight of feeds of *Hoplias malabaricus* fish caused a serious renal and hepatic injury, including disruption of the rough endoplasmic reticulum and mitochondria with alteration of the nucleus shape of hepatocytes.

Coagulative necrosis at 2.5 mg and 5 mg of methyl mercury-treated rats in the present study agreed with the results of both **Simenova (1999)** and **Rabitto *et al*., (2005)** who mentioned hepatic necrosis as a part of the toxicopathic effects of methyl mercury; these also align with the results of **Nwangwa *et al*., (2015)** who noticed an active biochemical accumulation of methyl mercury in liver and kidney of Wister rats and a significant elevation of serum glutamate oxaloacetate transaminase and pyruvate oxaloacetate transaminase

when exposed to the methyl mercury in drinking water at a 10 ppm concentration, which reflects the hepatic damage. As stated by **Manhan, (1991)**, hepatic necrosis is induced through enzymatic inhibition and disruption of membranes and disturbances in the synthesis of protein and the metabolism of carbohydrates. The detoxification mechanism of liver for methyl mercury will cause oxidative stress and elevation of the reactive oxygen species, leading to the degenerative effects and necrosis of the hepatocytes **(Limke *et al*., 2004).**

The presence of the high affinity of the methyl mercury to bind with the sulfhydryl group of the glutathione gives the rest an important role to reverse the cytotoxic effects of the methyl mercury; this fact was supported by the trial of **Yasutake and Hirayama, (1994)** who challenged the C57Bl female mice with a 160 µmol/kg dose of methyl mercury against GSH levels in liver and kidney and recorded 16% and 20% reduction in the liver and blood, respectively.

The histopathological findings have a similarity to what was mentioned by **Zurida Zulkipli *et al*., (2021)** who stated a variable lesion included hepatic lipidosis, vacuolar swelling and necrosis of hepatocytes with bile duct hyperplasia; also, they noticed glomerulonephritis, interstitial nephritis, severe tubular necrosis and hyalinization in the fish exposed to methyl mercury.

CONCLUSION

The oral administration of dimethyl mercury to the rat led to harmful and toxic effects on liver and kidney in rats by both acute and chronic models of exposure.

Acknowledgment

The authors sincerely thank the management of the animal house and the department of pathology and poultry diseases at the college of the veterinary medicine/ University of Mosul. For their support.

Conflict of interest:

The authors declare no similarity in the objectives and research method with other articles.

REFERENCES:

ABERNETHY, D.R., DESTEFANO, A.J., CECIL, T.L., ZAIDI, K., WILLIAMS, R.L., and USP METAL IMPURITIES ADVISORY PANEL, 2010. Metal impurities in food and drugs. Pharmaceutical research, 27, pp.750-755.

AJIBADE, A.J., ESHO, J.O., KEHINDE, B.D., and ADELEYE, O.O., 2019. Histological and biochemical effects of mercury chloride on the kidney of adult Wistar rats. J Pharm Pharmacol, 1, pp.21-7. https://doi.org/10.36349/easjpp.2019.v01i01.006

CASTOLDI, A.F., JOHANSSON, C., ONISHCHENKO, N., COCCINI, T., RODA, E., VAHTER, M., CECCATELLI, S., and MANZO, L., 2008. Human developmental neurotoxicity of methylmercury: impact of variables and risk modifiers. Regulatory Toxicology and Pharmacology, 51(2), pp.201-214. https://doi.org/10.1016/j.yrtph.2008.01.016.

CHEUK, D. K., and WONG, V., 2006. Attention-deficit hyperactivity disorder and blood mercury level: a case-control study in Chinese children. Neuropediatrics. 37(4), PP. 234-240. https://doi.org/10.1055/s-2006-924577

DA SILVA, D.A.F., TEIXEIRA, C.T., SCARANO, W.R., FAVARETO, A.P.A., FERNANDEZ, C.D., GROTTO, D., BARBOSA JR, F., and KEMPINAS, W.D.G., 2011. Effects of methylmercury on male reproductive functions in Wistar rats. Reproductive Toxicology, 31(4), pp.431-439. https://doi.org/10.1016/j.reprotox.2011.01.002

KIM, S., KIM, G. B., PARK, K. H., KANG, T. S., LEE, J. H., and NAM, S. H., 2006. A study on exposure and health effect of mercury (II) Incheon: National Institute of Environmental Research, pp. 1–115.

KJELLSTROM, T.E., REEVES, R.L., and MITCHELL, J.W., 1982. Comparison of mercury in hair with fish eating habits of children in Auckland. Community health studies, 6(1), pp.57-63. https://doi.org/10.1111/j.1753-6405.1982.tb00351.x

LEE, C., CHO, S.D., CHANG, D.S., SHIN, D.H., OH, D.H., WHANG, I.K., KWON, K.S., WOO, G.J., CHUN, H.S., OH, S.S., and KIM, G.H., 2006. Food safety guidelines for consumer. Safe Food, 1(4), pp.31-43.

LIMKE, T.L., HEIDEMANN, S.R., and ATCHISON, W.D., 2004. Disruption of intraneuronal divalent cation regulation by methylmercury: are specific targets involved in altered neuronal development and cytotoxicity in methylmercury poisoning? Neurotoxicology, 25(5), pp.741-760.

MAHMOOD, A. HUSSEIN, MAHA T. AL-SAFFAR ad KARAM H.AL-MALLAH, 2023. Biochemical and histopathological assessment of zinc acetate-induced nephrotoxicity in male albino rats. Journal of Applied Veterinary Sciences, 8 (4): pp. 37-42. https://dx.doi.org/10.21608/javs.2023.218887.1248

MANAHAN, S. E. 1991. Water Pollution Environment Chemistry, first ed. Lewis Publishers, London.

MELA, M., RANDI, M.A.F., VENTURA, D.F., CARVALHO, C.E.V., PELLETIER, E., and RIBEIRO, C.O., 2007. Effects of dietary methylmercury on liver and kidney histology in the neotropical fish Hoplias malabaricus. Ecotoxicology and Environmental Safety, 68(3), pp.426-435. https://doi.org/10.1016/j.ecoenv.2006.11.013

MELA, M., CAMBIER, S., MESMER-DUDONS, N., LEGEAY, A., GRÖTZNER, S.R., DE OLIVEIRA RIBEIRO, C.A., VENTURA, D.F., and MASSABUAU, J.C., 2010. Methylmercury localization in Danio rerio retina after trophic and subchronic exposure: a basis for neurotoxicology. Neurotoxicology, 31(5), pp.448-453. https://doi.org/10.1016/j.neuro.2010.04.009.

MYERS, G.J., DAVIDSON, P.W., and STRAIN, J.J., 2007. Nutrient and methyl mercury exposure from consuming fish. The Journal of nutrition, 137(12), pp.2805-2808. https://doi.org/10.1093 /jn/137.12.2805.

NWANGWA, E.K., NWOKOCHA, C.R., UZUEGBU, U.E., and OVUAKPORAYE, S.I., 2015. The bioaccumulation of mercury in kidney and liver of wistar rat exposed to methyl mercury. Biomedical and Pharmacology Journal, 1(1), pp.47-50. https://api.semanticscholar.org/CorpusID:59467386

RABITTO, I.S., COSTA, J.A., DE ASSIS, H.S., PELLETIER, E., AKAISHI, F.M., ANJOS, A., RANDI, M.A.F., and RIBEIRO, C.O., 2005. Effects of dietary Pb (II) and tributyltin on neotropical fish, Hoplias malabaricus: histopathological and biochemical findings. Ecotoxicology and environmental safety, 60(2), pp.147-156.

SALAH, B.A., AL-FATHI, M.Y., and SHINDALA, M.K., 2024. Histological liver, kidney, and brain changes induced by pregabalin drug in albino rats. Iraqi J Vet Sci, 38(3), pp.555-64. https://doi.org/10.33899/ijvs.2024.145830.3398.

SIMENOVA, A.D. 1999. Behavioural, haematological and histological studies on acute toxicity mercury on Salmo gaisdneri Richardson and tilapia rendalli Boulenger. J. Fish Biol, 10, pp.575-582. http://dx.doi.org/10.1111/j.1095-8649.1977.tb04090.x

SINGHM, B., and MAURYA, N. K., 2023. Mercury Toxicity: Its Toxic Effect on Fish and Human Health, Vigyan Varta an International E-Magazine for Science Enthusiasts.4(7), pp. 32-35. https://www.researchgate.net/publication/372288739.

SU, J. Y., and CHEN, W., 1979. The effect of methylmercury on isolated cardiac tissues. Am J Pathol. 95(3), pp. 753–764**.**

VICENTE, É., BOER, M., NETTO, C., FOCHESATTO, C., DALMAZ, C., SIQUEIRA, I.R., and GONÇALVES, C.A., 2004. Hippocampal antioxidant system in neonates from methylmercury-intoxicated rats. Neurotoxicology and teratology, 26(6), pp.817-823. https://doi.org/10.1016/j.ntt.2004.08.003

YASUTAKE, A., and HIRAYAMA, K., 1994. Acute effects of methylmercury on hepatic and renal glutathione metabolisms in mice. Archives of toxicology, 68(8), pp.512-516.

ZULKIPLI, S.Z., LIEW, H.J., ANDO, M., LIM, L.S., WANG, M., SUNG, Y.Y., and MOK, W.J., 2021. A review of mercury pathological effects on organs specific of fishes. Environmental Pollutants and Bioavailability, 33(1), pp.76-87. https://doi.org/10.1080/26395940.2021.1920468.

How to cite this article:

Semaa Ahmad Baker, Semaa Sami Al-Modaress and Karam Hashim Al-Mallah, 2025. Histopathological Study of The Acute and Chronic Toxic Effects of Dimethyl Mercury on Liver and Kidney of Male Albino Rats. Journal of Applied Veterinary Sciences, 10 (2): 136-143. **DOI: 10.21608/javs.2025.364375.1544**