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Original Article

Investigating the effects of equeous fruit extract of *Tamarindus indica* on mercuric chloride-induced liver toxicity in adult Wistar rats

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Abstract

BACKGROUND AND AIM: Mercury is a highly toxic metal that poses substantial risks to human health due to its widespread environmental presence and industrial use. Exposure to mercury occurs through various routes, leading to tissue damage mostly liver. The aim of this study was to investigate the effects of aqueous fruit extract of *Tamarindus indica* on Mercuric chloride-induced liver damage in adult Wistar rats.
METHODOLOGY: A total of 42 adult male Wistar rats were divided into six groups with seven rats per group and administered 250mg/kg and 500mg/kg doses of *Tamarindus indica* extracts and 1mg/kg of mercury chloride. Liver tissues were harvested after administration of the extracts for 60 days and mercury chloride administration for 30 days. The harvested liver tissues were fixed with 10% formal saline for about 24hrs and processed into microscopic slides, and subjected to microscopic examination. Biochemical assays were conducted also to investigate the levels of liver enzymes.
RESULTS: Rats in group B treated with mercury chloride showed significant decreases in body weight and increases in liver weight, indicative of the inflammation of the hepatocytes. Elevated levels of liver enzymes and histological changes further indicated liver damage induced by mercury chloride. However, rats treated with *Tamarindus indica* extracts showed increase on body weight change, liver weight, liver enzymes, and histological alterations, suggesting potential hepatoprotective properties.
CONCLUSION: *Tamarindus indica* demonstrates potential therapeutic benefits in attenuating mercury chloride-induced liver damage, highlighting its role as a natural hepatoprotective agent.

Keywords:
Mercury chloride; *Tamarindus indica;* Liver damage; Hepatoprotective; Wistar rats

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INTRODUCTION

There is a growing awareness that human activities are playing a significant role in the pollution of the environment with toxic and cancer-causing metallic compounds. Multiple pieces of evidence indicate that these metals, as they accumulate, are contaminating water sources and the food chain with their compounds. As a result, the pollution of the environment with metal compounds from industrial sources is becoming a pressing issue. Unlike the majority of organic pollutants, heavy metals do not undergo degradation; instead, they build up in the environment and the food chain, as reported by Jagadeesan *et al*. (2007). Particularly, mercury has been established as highly toxic to humans, and its uses in various industries has experienced rapid growth in recent times (Budnik and Casteleyn, 2019).

Mercury is widespread both as an environmental pollutant and an industrial contaminant. It leads to vast tissue alterations and is responsible for inducing peripheral neuropathy in both experimental animals and humans, (Şener *et al*., 2007).

Mercury poisoning can occur through inhalation from pollution, ingestion from contaminated foods, or skin absorption through cosmetics, (World Health Organization in 2017). The extensive impacts of mercury associated with industries on human and animal biological systems have been extensively documented by the World Health Organization. General exposure to mercury is further exacerbated by consuming contaminated food and drinking contaminated water (Clarkson and Magos, 2006). Although there have been significant reductions in recent years in environmental and occupational exposures to mercury, this metal remains a threat to human health from various sources such as air, water, and food (Brkljačić *et al*., 2004). Past studies have conclusively shown that mercury chloride specifically

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causes damage to the histology of the liver and ultrastructural aspects. This was substantiated through the observation of periportal fatty degeneration and cell necrosis, as reported by Agha *et al*. (2014).

At present, significant portions of the population are facing exposure to relatively high levels of mercury due to high usage of agricultural pesticides and fluorescent light bulbs. The liver, being second only to the skin in size among the body's organs, occupies a pivotal role as the central chemical processing hub responsible for metabolizing chemical substances. Consequently, it functions as a primary location for mercury metabolism and accumulation, often leading to various forms of liver damage, including hepatitis (Agha *et al*., 2014).

The World Health Organization (WHO) recognizes the crucial role of traditional and complementary medicines (TCM) within the global healthcare system (Burton *et al*., 2015). In Africa, a significant percentage, estimated at over 80% of the population, relies on medicinal plant species to fulfill their fundamental healthcare needs (Ngbolua *et al*., 2011). Medicinal plants have been utilized for generations to alleviate symptoms and manage diseases, playing a critical role in healthcare systems where a substantial portion of the global population employs herbs as medicinal remedies (Sofowora *et al*., 2013).

In modern times, researchers are showing an increasing interest in investigating the pharmacological effects and potential mechanisms of various medicinal plants, utilizing both *in vitro* and *in vivo* models, as reported in the study by Anyasor *et al*. (2019). Within the realm of pharmaceuticals, plants that have been traditionally employed in ethnomedicine for extended periods stand out as valuable sources of active phytoconstituents, offering medicinal or health benefits for various ailments and conditions (Aye *et al*., 2019).

Tamarindus indica, a plant with a long history of use, serves as a prime example of such plants. Tamarind fruit is rich in substances like ascorbic acid and β-carotene, which have been established as potent antioxidants (Farombi *et al*., 2002). Pharmacological investigations into this plant have uncovered its antibacterial, antidiabetic, antifungal, anti-inflammatory, antimalarial, and antioxidant properties (Jha *et al*., 2005). Numerous reviews have explored the anticancer and hepato-protective characteristics of *Tamarindus indica* (Jha *et al*., 2005). This study is aimed at investigating the effects of aqueous fruits extract of *Tamarindus indica* on Mercuric chloride-induced liver damage in adults Wistar Rats.

MATERIALS AND METHODS

Collection and identification of plant material

Tamarindus.indica fruits were harvested in Buni Yadi Village in Gujba Local Government Area of Yobe State. It was identified in Department of Plant Biology and Biotechnology (PBB), University of Benin, Benin City (herbarium number UBH-M102). Mercuric chloride was manufactured by Anosantec Laboratories USA (Batch no. D7p6).

Preparation of aqueous extracts of *Tamarindus indica*

Tamarindus indica was thoroughly washed with running water to remove dirts and debris, air-dried for 2 weeks and then pulverized to powdered form. The powdered form obtained was weighed and soaked in distilled water for 24 hours. The solution was then filtered. The residues were discarded while the filtrate was freeze dried with freeze drying machine and refrigerated.

Tamarindus indica was thoroughly washed underneath clean tap water. It was then allowed to air dry, grinded into powder form (Pulverized), and sieved. After pulverization, the plant was weighed and the weight was 12,100g. Excursive extraction was done on the pulverized plant material by soaking it in ethanol reagent for 72 hours. To ensure proper extraction, the solution was occasionally stirred. At the end of the third day, the solution was carefully filtered through two layers of cheesecloth with thick cotton wool and a handkerchief placed in it. This process was repeated four times. Finally, the filtrate was allowed to pass through a filter paper to eliminate the residues. At the end of the process, the filtrate was dried using a freeze-dryer and the dried extract was weighed. A total yield of 310g was obtained after freeze drying and was kept in a refrigerator in an airtight container until it was time to use it (Liapis and Bruttini, 2020).

Experimental animals

Forty-two (42) adult male Wistar Rats weighing between 150 and 180 grams were used for this experiment. These rats were bred in the Animal House of the Department of Anatomy, School of Basic Medical Sciences, University of Benin, Benin City. They were randomly distributed into 6(six) groups with seven rats per group.

Group A served as control, Group B was administered 1mg/kg of mercury chloride, Groups C and D received 250mg/kg and 500mg/kg of aqueous extract of *Tamarindus indica* respectively, while groups E and F received 250mg/kg and 500mg/kg of *Tamarindus indica* followed by 1mg/kg of mercury chloride respectively. After acclimatization, animals were administered with *Tamarindus indica* and mercury chloride using a gavage with an orogastric tube attached. (Adebayo *et al*., 2006). Twenty-four hours after the end of the experimental periods, the animals were weighed, and sacrificed with chloroform.

Sacrifice of animals

During the time of the experiment, the rats were weighed weekly to monitor their weight change as the experiment progressed.

During sacrifice, the final weights of the rats were taken using compact electric weighing scale calibrated in grams. Cotton wools were soaked with chloroform of about 30ml in an enclosed container was used to anaesthetized the animals. After anaesthetizing, the rat was placed on supine position on the dissection trolley and ventral abdomino- thoracic incision was made on the rat to expose the viscerae. Thereafter blood samples

were collected through inferior vena cava using 5mls syringes, centrifuged and the serum was stored in sterile plain bottles for hormone analysis Thereafter, testes were harvested, weighed and fixed with Bouin's fluid in a universal bottle for histological studies.

Tissue collection, processing and staining, histopathology

At the end of the eight-week administration, rats were humanely sacrificed using chloroform anesthesia. Briefly, cotton wool soaked with 30 ml of chloroform was placed in an enclosed container. Rats were then introduced into the container for 2-5 seconds to induce anesthesia. Following anesthesia, rats were placed in a supine position on a dissection table. An abdomino-thoracic incision was made to expose the abdominal viscera. Blood samples were collected through venous and cardiac puncture using 5-ml syringes. The blood samples were transferred into plain bottles and centrifuged to obtain serum for liver function tests. The liver was harvested, weighed, and fixed in 10% buffered formalin for 24 hours. The tissues were then processed for histological analysis using Haematoxylin and Eosin (H&E) staining. Histological sections were examined using an Olympus research microscope attached to a Leica CC50 digital camera. Photomicrographs were taken at a magnification of 400x.

Biochemical assays

The estimation of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) is conducted using the Reitman and Frankel method (1957), where the respective enzyme activities are determined by incubating serum with specific reagents and measuring the absorbance of the resulting colorimetric complexes at 546 nm after 30 minutes and subsequent addition of sodium hydroxide, while alkaline phosphatase (ALP) is measured following Englehardt's method (1970) by monitoring the yellow color formation from p-nitrophenyl phosphate at 405 nm, and total bilirubin is quantified using the Jendrassik and Grof method (1938) through its reaction with diazotized sulphanilic acid, measuring absorbance at 578 nm, along with albumin estimation following Doumas et al. (1971), where binding with bromocresol green at 578 nm allows for proportional quantification based on absorbance readings.

Statistical analysis

GraphPad Prism version 9.0 (GraphPad Software Inc.) for Windows was used to analyze the data obtained in the study. Results were expressed as Mean ± SEM (standard error of mean). Differences among the means were determined by one-way analysis of variance (ANOVA). Values were considered statistically significant if P value was less than 0.05 ($p < 0.05$). Least Significant Difference (LSD)*Post Hoc* test was used to determine where the significance lay.

RESULTS

Table 1 shows results for body weight and liver weight. From the table, there was no statistically significant difference ($P>0.05$) in initial body weight across all the groups. The final body weight was significantly reduced ($P<0.05$) in Mercury Chloride-only-treated group compared to the control. However, there was no statistically significant difference ($P>0.05$) in final body weight of Mercury chloride group treated with 250mg/kg of *Tamarindus indica* extract compared to Mercury chloride only treated group but there was a statistically significant increase ($P<0.05$) in final body weight of Mercury chloride group treated with 500mg/kg of *Tamarindus indica* extract compared to Mercury chloride only treated group. There was no statistically significant difference ($P>0.05$) in final body weight of the groups treated with 250mg/kg and 500mg/kg of *Tamarindus indica* extract compared with the control group.

Comparing initial and final weights within each group, there was a statistically significant increase ($P<0.05$) in body weight in the control group, 250mg/kg of *Tamarindus indica* extract only treated group and 500mg/kg of *Tamarindus indica* extract only treated group; there was a statistically significant decrease ($P<0.05$) in body weight in the Mercury chloride only treated group. However, there was a statistically significant increase ($P<0.05$) between initial and final body weight in 250mg/kg of *Tamarindus indica* extract plus Mercury chloride and 500mg/kg of *Tamarindus indica* extract treated plus Mercury Chloride groups respectively. This is also reflected in the body weight change (final body weight minus initial body weight) where there was no statistically significant difference ($P>0.05$) in body weight change of the groups treated with 250mg/kg and 500mg/kg doses of *Tamarindus indica* extract compared with the control group but there was weight loss in the Mercury chloride only group, 250mg/kg dose plus Mercury chloride group and 500mg/kg dose plus Mercury chloride group which was statistically significant ($P<0.05$) compared to the control group.

There was no statistically significant difference ($P>0.05$) in liver weight of the groups treated with 250mg/kg and 500mg/kg doses of *Tamarindus indica* compared with the control group but there was statistically significant ($P<0.05$) increase in liver weight in the Mercury chloride only group compared to the control group. However, there was a statistically significant increase ($P<0.05$) in the liver weight in the 250mg/kg and 500mg/kg doses of *Tamarindus indica* compared to the Mercury chloride only group. There was no statistically significant difference ($P>0.05$) in testiculosomatic of the groups treated with low dose of the extract, low dose plus CdCl and high dose plus CdCl compared with the control group.

Table 2 shows results for liver function test. From the table, there was a statistically significant increase ($P<0.05$) in Aspartate transferase (AST), Alanine transferase (ALT), Alkaline Phosphate (ALP), Total bilirubin, Conjugated bilirubin and Albumin in the Mercury chloride only treated group and the 500mg/kg of *Tamarindus indica* only treated group as compared to control. However, there was a statistically significant decrease ($P<0.05$) in Aspartate transferase (AST), Alanine transferase (ALT), Alkaline Phosphate (ALP), Total bilirubin, Conjugated bilirubin and Albumin

in the 250mg/kg and the 500mg/kg of *Tamarindus indica* only treated group as compared to the Mercury chloride only treated group.

Histological finidings

The control group of rat liver tissue exhibited normal anatomical features characterized by intact hepatocytes, sinusoids, bile ducts, hepatic arteries, portal veins, and central veins (Plate 1).

In contrast, the rat liver exposed to mercury chloride displayed significant pathological changes, including zonal necrosis, vasodilatation, vascular ulceration and congestion, periportal infiltrates of inflammatory cells, and bile duct epitheliosis (Plate 2). These findings indicate that mercury chloride has a detrimental effect on liver tissue, leading to extensive damage and inflammation, which could impair liver function severely.

The administration of 250mg/kg of tamarind to a separate group of rats resulted in liver tissue that displayed normal architecture, much like the control group (Plate 3). This suggests that at this dosage, tamarind does not adversely affect the liver and may even be protective, preserving the normal histological features commonly associated with healthy liver function.

Similarly, the rat liver subjected to 500mg/kg of tamarind also presented with normal anatomical structures, including healthy hepatocytes, bile ducts, and portal veins (Plate 4). However, there was an indication of periportal lymphocyte mobilization, suggesting a mild and localized immune response without damaging effects on liver architecture. This indicates that tamarind at this higher dosage can still maintain liver health while possibly stimulating some level of immune activity.

In rats administered both mercury chloride and 250mg/kg of tamarind, the liver architecture was observed to appear normal, with intact hepatocytes, bile ducts, and portal veins, along with a noted periportal lymphocyte mobilization (Plate 5). This implies that tamarind may have a protective effect against the toxic effects of mercury chloride, allowing for the preservation of typical liver structure and some degree of immune response despite the initial injury caused by mercury exposure.

Finally, the rat liver exposed to both mercury chloride and 500mg/kg of tamarind showed hepatocytes alongside focal portal vascular ulceration and periportal lymphocyte mobilization (Plate 6). While there is evidence of some liver damage, the presence of tamarind appears to mediate the extent of the injury when compared to mercury chloride treatment alone. This suggests a partial protective role of tamarind at this higher dosage, indicating that while some damage persists, overall liver architecture is better preserved relative to the control solely subjected to mercury chloride.

Table 1. Body and liver weights of control and *Tamarindus indica* treated mercury chloride-induced rats

Tests/groups	Group A	Group B	Group C	Group D	Group E	Group F	p-value
Initial weight (g)	168.7±6.766	185.3±9.701	166.7±2.404	168.7±5.969	135.3±1.764	157.7±5.978	0.0325
Final weight (g)	206.3±2.404*	168.7±17.40*	183.7±2.728*	187.7±6.692*	164.7±3.712*	171.7±3.180*	0.0285
Body weight change (g)	42.00±0.5774	-16.33±8.090*	17.00±2.517*	19.00±1.000*	11.33±2.404#	14.00±2.082#	<0.0001
Liver weight (g)	5.833±0.120	8.000±0.416*	5.600±0.153	5.367±0.153	5.500±0.253#	6.067±0.067#	0.0001

* $p< 0.05$ compared with the control group; # $p< 0.05$ compared with the Mercury chloride-alone group.

Table 2. Liver function tests markers of controls and *Tamarindus indica*-treated mercury chloride-induced rats

Tests/groups	Group A	Group B	Group C	Group D	Group E	Group F	*P*-value
Ast (u/l)	81.87±0.121	158.1±0.174*	71.82±1.359	137.1±1.265#	119.5±11.44#	110.6±5.443#	0.0001
Alt (u/l)	137.1±0.202	264.8±0.292*	120.3±2.277	299.7±2.118#	200.1±19.17#	185.3±9.118#	0.0001
Alp (iu/l)	250.0±0.369	482.7±0.532*	219.3±4.151	418.7±3.861#	364.9±34.95#	337.8±16.62#	0.0001
Total bilirubin (mg/dl)	469.7±0.693	907.0±0.999*	412.0±7.799	786.7±7.255#	685.5±65.66#	634.6±31.23#	0.0001
Conjugate bilirubin (mg/dl)	343.0±0.506	662.3±0.729*	300.9±5.695	574.5±5.298#	500.6±47.95#	463.5±22.80#	0.0001
Albumin (g/L)	635.6±0.938	1227±1.351*	55.7±10.55	1065±9.817#	927.6±88,85#	858.8±42.26#	0.0001

* $p< 0.05$ compared with the control group; # $p< 0.05$ compared with the Mercury chloride-alone group.

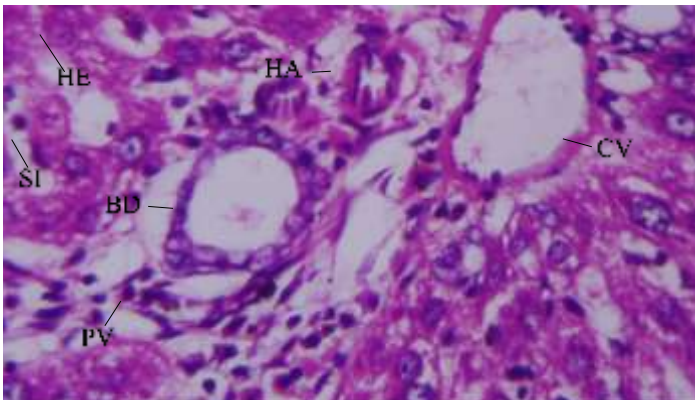

Plate 1: Rat liver. Control. Composed of normal tissue architecture: hepatocytes (HE), sinusoids (SI), bile ducts (BD), hepatic artery (HA), portal vein (PV), Central Vein (CV): H and E 400x

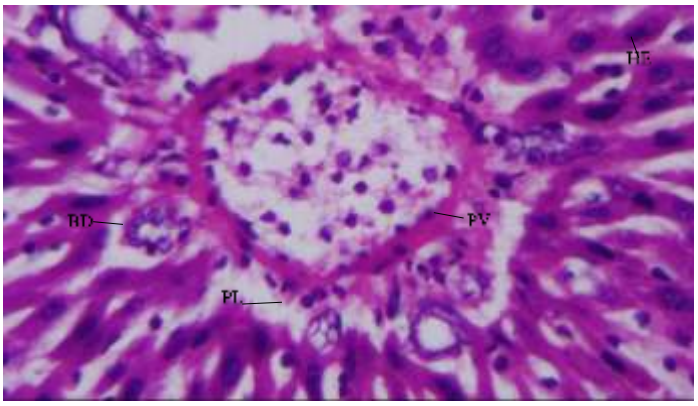

Plate 4: Rat liver administered 500mg Tamarind only showing normal architecture: hepatocytes (HE), bile ducts (BD), portal vein (PV), periportal lymphocyte mobilization (PL): H and E 400x

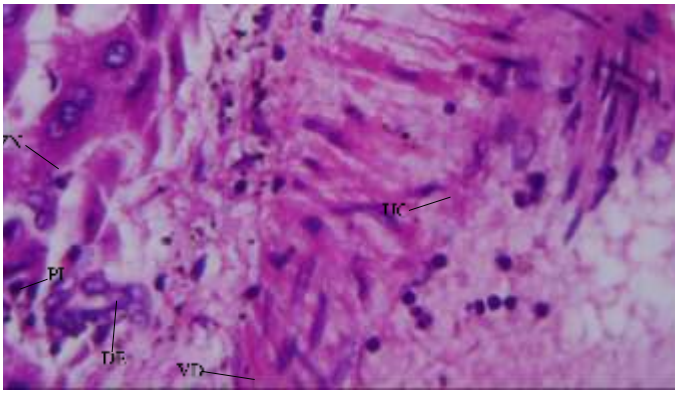

Plate 2: Rat liver administered Mercury Chloride only showing: zonal necrosis (ZN), vasodilatation (VD), vascular ulceration and congestion (UC), periportal infiltrates of inflammatory cells (PI), bile duct epitheliosis (DE): H and E 400x

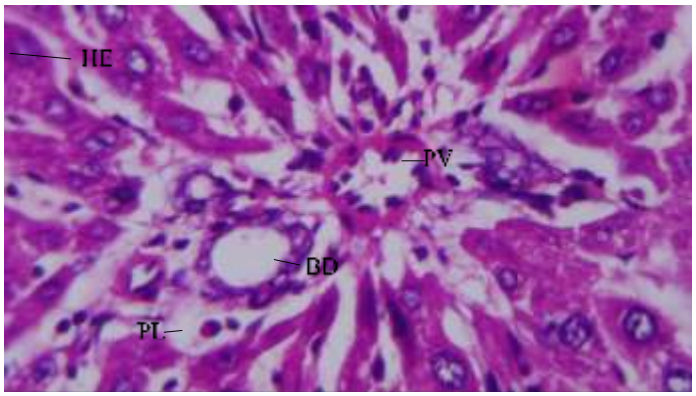

Plate 5: Rat liver administered Mercury Chloride + 250mg Tamarind showing normal architecture: hepatocytes (HE), bile ducts (BD), portal vein (PV), periportal lymphocyte mobilization (PL): H and E 400x

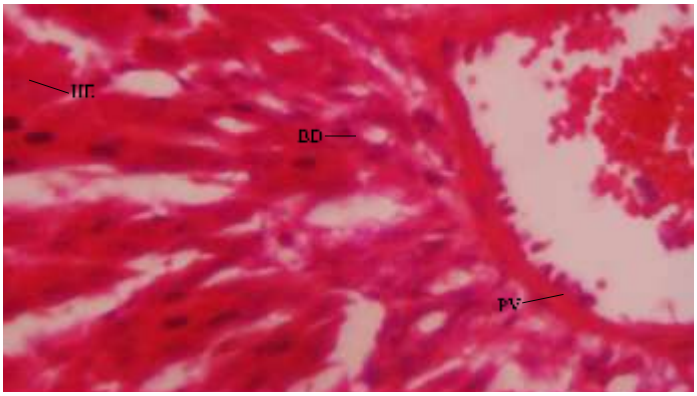

Plate 3: Rat liver administered 250mg Tamarind only showing normal architecture: hepatocytes (HE), bile duct (BD), portal vein (CV): HandE 400x

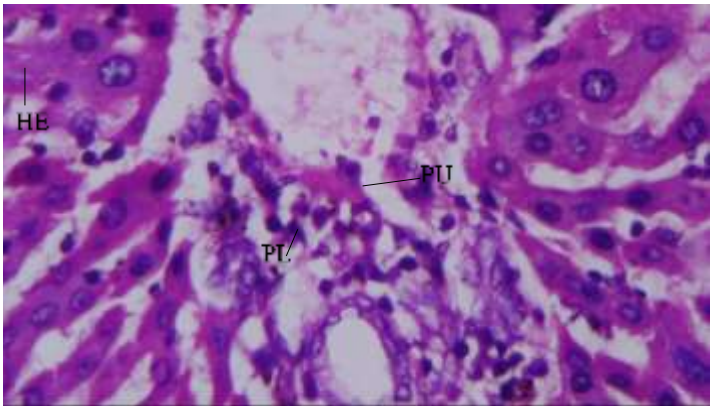

Plate 6: Rat liver administered Mercury Chloride + 500mg Tamarind showing: hepatocytes (HE), focal portal vascular ulceration (PU), periportal lymphocyte mobilization (PL): HandE 400x

DISCUSSION

Rats treated with mercury chloride only exhibited a statistically significant decrease in body weight change compared to the control group (Group A). This decrease was observed at the end of the administration period. The reduction in body weight suggests that mercury chloride negatively impacts weight regulation in rats (Caglayan *et al*., 2019). This result corroborated with result obtained by Hussain *et al*., (1997). Rats treated with *Tamarindus indica* at doses of 250mg/kg (Group C) and 500mg/kg (Group D) also showed a statistically significant decrease in body weight change when compared to the control group. These effects were evident during the treatment period. *Tamarindus indica*, despite its potential health benefits, appears to contribute to weight loss in rats. Similar finding was reported by Kuru, (2014) indicating that *Tamarindus indica* possesses weight reduction and hypolipidemic properties (Dhingra *et al*., 2011) Interestingly, rats treated with 250mg/kg (Group E) and 500mg/kg (Group F) of *Tamarindus indica* before mercury chloride administration exhibited a statistically significant increase in body weight change. This increase was observed when compared to the group receiving mercury chloride treatment alone. Pre-treatment with *Tamarindus indica* seems to mitigate the weight-reducing effects of mercury chloride. *Tamarindus indica* contains bioactive compounds such as hydroxycitric acid (HCA), which has been studied for its impact on appetite (Kamar *et al*., 2022). HCA may influence the activity of enzymes involved in lipid metabolism and appetite control (Han *et al*., 2016). By modulating neurotransmitters (such as serotonin), HCA could affect satiety and food intake (Bano and Akhter, 2020). *Tamarindus indica* may interact with metabolic pathways related to glucose utilization, lipid oxidation, and energy expenditure (Kuddus *et al*., 2020). Compounds in *Tamarindus indica* might enhance insulin sensitivity or alter glucose transporters, affecting overall energy balance (Costa *et al*., 2022). *Tamarindus indica* may impact gut microbiota composition (Li *et al*., 2020). Gut microbes play a role in energy extraction from food and overall metabolism. Alterations in gut microbiota could affect weight regulation (Blaut, 2014).

Liver weight was significantly increased in the group that received mercury chloride treatment only. Elevated liver weight in the mercury chloride-only group suggests potential hepatotoxicity. Mercury chloride is known to accumulate in liver tissues (Joshi *et al*., 2014). It can disrupt cellular membranes, impair mitochondrial function, and induce oxidative stress (Ahmad and Mahmood, 2019). Increased liver weight may result from inflammation, fluid retention, or cellular hypertrophy (Ahmad *et al*., 2021). Rats treated with various doses of *Tamarindus indica* before administration of mercury chloride exhibited significant reduction in the liver weight, this appears to be a mitigation of the increase in liver weight observed in te mercury chloride only treated group thus suggesting that *Tamarindus indica* may possess' protective effects against hepatotoxicity (Liman and Atawodi, 2015). *Tamarindus indica* may enhance detoxification pathways, reduce oxidative stress, or modulate inflammatory responses. It could influence liver enzymes involved in metabolism and regeneration (Guneidy *et al*., 2023).

Liver enzymes including AST, ALT, ALP, TB, CB and Albumin showed significant increase in the animals treated with mercury chloride when compared with the control. Similar result was obtained from literature by Joshi *et al*., 2014 after similar research was carried out by him and his team. An increase in these liver enzymes suggest significant hepatic damage. This increase could be due to hemolysis due to its presence in the red blood cells. The elevations could also be due to an insult from the free radical injury to the hepatocyte (Atiba *et al*., 2016). The free radical injury in our study was evidenced by the corresponding increase in MDA, a product of free radical injury.

The control group represents the normal tissue architecture of the rat liver. This baseline condition allows us to understand the liver's natural function without any specific intervention. No external factors were applied, so the liver maintains its healthy state. Rat Liver Administered Mercury Chloride showed signs of Zonal Necrosis (ZN) which are localized cell death in specific zones, Vasodilatation (VD) which is widening of blood vessels, potentially affecting blood flow, Vascular Ulceration and Congestion (UC) which are damage to blood vessels leading to congestion. Periportal Infiltrates of Inflammatory Cells (PI) which potentially suggest Immune response to injury. Bile Duct Epitheliosis (DE) thus suggesting Changes in bile duct lining. These changes implies that Mercury Chloride exposure induces liver damage (Goa *et al*., 2021). This likely involves oxidative stress, inflammation, and vascular disruption (Takahashi and Shimihata, 2019). This result was in corroboration with similar finding by Nabil *et al*. (2020). However, rat Liver Administered 250mg/kg and 500mg/kg of Tamarind only maintains normal liver architecture suggesting that tamarind may protect against Mercury Chloride-induced damage. This could be due to the ability of Tamarind's antioxidants to counteract toxic effects (Sandesh *et al*., 2014). Futhermore, rat Liver Administered Mercury Chloride + 250mg Tamarind showed that the Liver maintains normal architecture suggesting that Tamarind mitigates Mercury Chloride damage. This may be due to *Tamarindus indica*'s antioxidants and anti-inflammatory properties, which play a role (Borquaye *et al*., 2020). But rat Liver Administered Mercury Chloride + 500mg Tamarind showed Focal Portal Vascular Ulceration (PU): Localized vessel damage. Periportal Lymphocyte Mobilization (PL): Immune response persists. This suggest that Tamarind at 500mg/kg partially protects against Mercury Chloride-induced injury. This could be due to continued immune modulation by Tamarind (Heloneida *et al*., 2021).

Conclusion: Overall, these findings suggest that *Tamarindus indica* may offer therapeutic potential in mitigating mercury chloride-induced liver damage, highlighting its potential as a natural hepatoprotective agent.

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