https://doi.org/10.47108/jidhealth.Vol9.Iss4.460
Mwangengwa LM, Medardus JJ, Journal of Ideas in Health (2026); 9(4):1452-1457
Journal of Ideas in Health

Prolonged low-dose atrazine exposure in drinking water: effects on liver, testis, and spermatozoa in albino rats

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Abstract

Background: Water atrazine contaminants can harm the tissues of animals at high levels, but effects of chronic low dose exposure remain unclear. This study aims to evaluate the impact of chronic low-dose atrazine exposure on the liver and testis in Wistar rats.

Methods: This was a controlled in vivo experimental animal study carried out between February and May 2026 at the Small Animal Research Unit (SARU) within the College of Veterinary Medicine and Biomedical Sciences at Sokoine University of Agriculture (SUA). The experiment involving 15 male Wistar rats allocated into three parallel groups: two atrazine-exposure groups receiving atrazine at 0.013 µg/L (drinking water level) and 0.172 µg/L (river level), and control distilled water. Treatment was by gastric gavage for 120 days. Following exposure, body weights were recorded, and animals were sacrificed under ether anesthesia. The liver and testes were excised, weighed, and processed for histological examination. Spermatozoa were analyzed from caudal epididymis.

Results: Exposure to 0.013 µg/L dose did not significantly affect body weight which increased at 0.172 µg/L dose. Both doses resulted in a significant reduction in liver and testicular weights. Histological analysis revealed insignificant pathological changes, with only mild central vein congestion and limited hepatocellular degeneration in the liver. Testicular tissues were largely normal, with minor germinal epithelial erosion in some seminiferous tubules. Spermatozoa parameters showed slight, statistically insignificant reductions.

Conclusion: Prolonged low-dose atrazine exposure did not induce significant adverse effects, although minor tissue alterations were observed.

Keywords: Atrazine, Drinking water, Liver, Low dosage, Sperm cells, Testis, Tanzania

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How to cite: Mwangengwa LM, Medardus JJ. Prolonged low-dose atrazine exposure in drinking water: effects on liver, testis, and spermatozoa in albino rats. J Ideas Health. 2026 August 31;9(4):1452-1457. doi: 10.47108/jidhealth.Vol9.Iss4.460

Article Info: (**Original Research**)
Received: 22 June 2026
Revised: 25 July 2026
Accepted: 21 August 2026
Published: 31 August 2026

Journal Home Page: https://www.jidhealth.com

e ISSN: 2645-9248

Background

Atrazine is a widely used herbicide for controlling weeds and enhancing crop productivity in modern agriculture [1]. Its use is particularly common in the production of food crops such as maize, sorghum, sugarcane, and pineapple [2–5], as well as in forestry and non-food crop systems [6]. Despite its extensive application, atrazine use is highly regulated or banned in several countries due to concerns regarding its potential environmental and health risks [7,8]. Following application, atrazine enters the soil, where it undergoes degradation through processes such as photolysis and microbial activity. These mechanisms involve biochemical transformations, including N-dealkylation and hydrolysis of the chloro substituent, resulting in intermediate by-products [4,9,10]. The rate of atrazine degradation is influenced by environmental factors such as soil temperature, moisture, and pH [11,12]. Notably, atrazine can persist in soil and surface water for extended periods, with reported stability exceeding 100 days at 20 °C [13]. Furthermore, its degradation decreases with increasing soil depth, facilitating leaching and accumulation in groundwater [14,15]. Environmental and public health concerns related to atrazine arise from the potential toxicity of both the parent compound and its degradation products, which may adversely affect ecosystem integrity, biodiversity, and food safety [16,17]. In Tanzania, atrazine residues have been detected in water sources used by humans and animals. For example, a study in the coastal region reported concentrations of 0.013 µg/L in drinking water, 0.016 µg/L in pond water, and 0.172 µg/L in river water [18]. Although these concentrations fall within World Health Organization (WHO) guideline limits and are unlikely to pose acute toxicity risks [19,20], the long-term health

implications of chronic exposure remain poorly understood. Evidence from experimental studies indicates that high doses of atrazine can induce pathological alterations in the liver [21–23]. However, the effects of prolonged exposure to low concentrations on hepatic function particularly given the liver's critical role in detoxification and metabolism remain insufficiently understood. Additionally, atrazine has been identified as an endocrine-disrupting compound [24,25]. At high doses, it has been associated with adverse effects on testicular structure, sperm count and motility, and epididymal enzyme activity. Whether similar effects may arise from chronic low-dose exposure is yet to be established. Accordingly, this study aimed to evaluate the effects of prolonged exposure to atrazine at concentrations comparable to those detected in Tanzanian water bodies on liver and testicular histopathology, as well as sperm parameters, using the white albino rat as an experimental model.

Methods

Study area

The study was conducted over four consecutive months, from February to May 2026, at Sokoine University of Agriculture (SUA) in Morogoro Municipality. The study area is located between longitudes 35.6° and 39.5° E and latitudes 5.7° and 10° S, on the lower slopes of the Uluguru Mountains, at approximately 1,600 feet above sea level. The experiments were carried out at the Small Animal Research Unit (SARU), within the College of Veterinary Medicine and Biomedical Sciences at SUA [26].

Study esign

The study employed controlled experimental animal study (in vivo) design based on the protocol described by [27,32], with minor modifications to suit the study objectives. The animals used for this study were male Wistar rats aged 6 months.

Sample size

A total of fifteen Wistar male rats were used in the study. The sample size adhered to ethical principles aimed at reducing animal use in research with necessary adjustments for this experiment [28,32].

Preparation of different atrazine concentrations

The stock solution of atrazine (185 g/L) was serially diluted to obtain working concentrations of 0.172 µg/L and 0.013 µg/L. Initially, the stock concentration was converted into consistent units (185 g/L = 1.85×10^8 µg/L) to facilitate accurate dilution calculations. Based on the target concentrations, the overall dilution factors were calculated to be approximately 1.08×10^9 for 0.172 µg/L and 1.42×10^{10} for 0.013 µg/L. Given that such a large dilution factor is impractical to achieve in a single step, a series of sequential dilutions was employed. Serial dilutions were performed beginning with a 1:100 dilution, followed by successive 1:10 dilutions. Each set of dilutions yielded a cumulative factor of 1:1000, and this process was repeated three times to achieve an overall dilution factor of 10^9, yielding an intermediate atrazine concentration of 0.185 µg/L. This intermediate solution was subsequently adjusted to obtain the desired working concentration of 0.172 µg/L and 0.013 µg/L. All dilutions were prepared using distilled water obtained from the College of Veterinary Medicine and Biomedical Sciences at SUA.

Experimental setup

The experiment consisted of three parallel groups of Wistar rats: two test groups and one control group. One treatment group received atrazine at a concentration of 0.013µg/L, representing levels found in drinking water, while the second group received 0.172 µg/L, similar to levels reported in river water [29]. Control group received distilled water. Feeding was done by gastric gavage using a needle designed for that purpose. More details on the experimental setup are displayed in Table 1. Atrazine exposure: Atrazine solutions were prepared at concentrations corresponding to 0.172 µg/L and 0.013 µg/L. The prepared solutions were administered to animals once daily by gastric gavage at a volume of 2 ml/animal/day. The administered atrazine dose was calculated based on the concentration of atrazine in the gavage solution, the volume administered, and the average initial body weight (104.6 g) of rats using the equation: Dose (µg/kg body weight/day) = [atrazine concentration (µg/mL) × administered volume (mL/day)] / body weight (kg). The resulting doses were 0.00329 µg/kg and 0.0002486 µg/kg body weight/day for the concentration of 0.013µg/L, representing levels found in drinking water, and 0.172 µg/L, similar to levels reported in river water [29].

Table 1. Experimental setup for a 120-day study evaluating the effects of prolonged exposure to low-dose atrazine in rats

Groups of adults male rats	Duration of treatment	Parameters
Negative control (n = 5) on distilled water.	**120 days**	Body weight, liver and testis weight, and histology, sperm parameters
Atrazine-treated group (n = 5); atrazine at a dose of 0.0002486 µg/kg body.	**120 days**	Body weight, liver and testis weight, and histology, sperm parameters
Atrazine-treated group (n = 5); atrazine at a dose of 0.00329 µg/kg body weight/day.	**120 days**	Body weight, liver and testis weight, and histology, sperm parameters

Data Collection Procedure

Measurement of the weight of the body, the liver, and the testis

Body weight was recorded before the initiation of treatment and again after 120 days of exposure using a calibrated digital weighing balance. At the end of the experimental period, the animals were euthanized under ether anesthesia. The abdominal cavity was then carefully opened, and the liver and testicular tissues were dissected and weighed using a digital analytical balance.

Assessment of Sperm Cell Parameters

Sperm cell motility

The caudal epididymis of each rat was dissected and homogenized in 1 mL of warm (37 0C) normal saline in a small beaker using a dissection scissor to make a sperm cell suspension. For progressive sperm motility assessment, a protocol by [30] was employed. A drop of sperm suspension was placed on a microscope slide, and a bright-field microscope

equipped with a camera (Olympus BH-2 with a Pro205A camera) was used to record five-second videos from five different fields of view on each slide. The videos were later assessed by three independent individuals to determine the proportion of progressively motile spermatozoa relative to the total number of sperm cells in each microscope slide field across study groups.

Sperm cells' vitality

Sperm cell viability was determined using a staining solution composed of nigrosine and eosin [31]. A drop of the sperm cell suspension was added on a microscope slide, followed by one drop of the staining solution, and mixed with a toothpick before being examined with a bright-field microscope at ×200 magnification. The proportion of live sperm cells with white to pinkish heads, and dead sperm cells, identified by reddish heads was calculated based on a total of 200 sperm cells counted across five different fields.

Sperm cells morphology

Sperm cell morphology was assessed using a staining solution composed of eosin and nigrosine (1:1 ratio). One drop of the sperm cell suspension was added on a microscope slide, followed by one drop of the staining solution, and mixed with a toothpick before being examined with a bright-field microscope at ×1000 magnification. Spermatozoa with normal morphology were identified as those with an intact, curved head, a straight neck and mid-piece, a straight long tail, and a normal acrosome. All other sperm cells were classified as morphologically abnormal.

Sperm cell counts

Sperm cell concentration was determined using a protocol described by [32]. Briefly, a total of 1 µl of sperm suspension was diluted at a ratio of 1:20 by adding 19 µl of 0.9% saline solution. The coverslip was placed in the Neubauer chamber firmly. After vigorous shaking, a 10 µl of the diluted semen was placed in a Neubauer hemocytometer and allowed to settle for 3 minutes. Sperm cells were then counted under a microscope at ×100 magnification in the five central squares of the chambers. Sperm cell concentration (cells/mL) was calculated as the total number of cells counted in the five central squares multiplied by the dilution factor and 10,000.

Histological assessment of the liver and testis

After weighing, liver and testicular tissues were fixed in 10% formalin.

After 48 hours of fixation, the tissues were dehydrated sequentially in 70%, 90%, 95%, and 95% ethanol. They were then cleared in xylene, embedded in paraffin wax, and sectioned at 5 µm thicknesses using a rotary microtome. The sections were stained with hematoxylin-eosin.

Stained sections were examined under a bright-field microscope at x100 and x200 magnifications [33]. Eight sections were prepared from each liver and testicular tissue sample for histopathological assessment. All sections were independently examined by two experienced pathologists. Histopathological changes were assessed systematically based on the presence and severity of observed lesions, including cellular degeneration, necrosis, congestion, inflammatory changes, and other tissue abnormalities.

Statistical analysis

The collected data were analyzed using one-way analysis of variance (ANOVA), followed by Tukey's HSD post hoc test for multiple comparisons. Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 25.0. Differences between group means were considered statistically significant at $p < 0.05$.

Results

This study evaluated the effects of prolonged exposure to low-dose atrazine contamination in drinking water on body weight, organ weights (liver and testes), and spermatozoa parameters using a rat model. The study revealed that prolonged exposure to low-dose atrazine did not cause a reduction in body weight in rats compared to the negative control. Specifically, rats treated with 0.013 µg/L atrazine exhibited body weights comparable to controls, whereas those exposed to 0.172 µg/L showed a modest increase (Table 2). In contrast, the weights of both liver and testes were significantly reduced in rats exposed for 120 days to 0.013 µg/L and 0.172 µg/L atrazine compared with the control group (Table 2). The histological examination of the liver revealed slight structural changes in the tissues in atrazine treated rats compared with the negative control (Figure 1). The liver section of the negative control rats showed normal hepatocytes arranged in cords radiating from the normal central vein and entrapping normal blood sinusoids between the cords (Figure 1). Rats under chronic exposure to concentrations of 0.013 µg/L and 0.172 µg/L of atrazine revealed mild congestion in the central vein and possessed a mixture of normal and a few degenerating hepatocytes showing cytoplasmic vacuolation and pyknotic nuclei in the liver tissues (Figure 1). The testicular tissues also showed slight histopathological changes in the seminiferous tubules in the atrazine-treated rats compared with the negative control (Figure 2). The negative control rats showed normal seminiferous tubules with intact germinal epithelium composed of several layers of spermatogenic cells (Figure 2). In contrast, the testes of atrazine treated rats revealed a mixture of normal seminiferous tubules with intact germinal epithelium and others exhibiting mild germinal epithelium erosion, beginning from the center of the tubules (Figure 2).

Table 2. Body, liver and testicular weights of rats (n= 15) following 120 days exposure to low-dose atrazine.

Parameter (g)	Negative control	Atrazine-treated (0.013 µg/L)	Atrazine-treated 0.172 µg/L	P-value
Final Body weight (Mean ± SEM)	103.3 ± 1.94	100.94 ± 4.29	125.01 ± 9.87	0.039
Weight of the liver (Mean ± SEM)	4.160 ± 0.11	3.68 ± 0.04	3.28 ± 0.06	<0.001
Weight of the Testis (Mean ± SEM)	1.98 ± 0.14	1.75 ± 0.15	1.29 ± 0.08	0.007

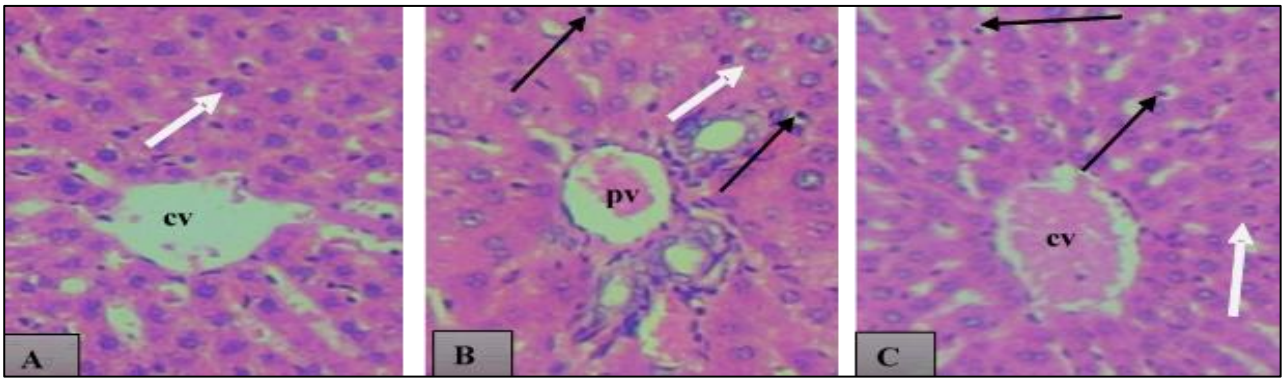

Figure 1. Representative hematoxylin and eosin (H&E) stained histological sections of rat liver tissue after 120 days of treatment (×200 magnification). **Section A** illustrates the hepatic architecture of the negative control group (distilled water), showing normal hepatocytes (white arrow) arranged in well-organized cords radiating from a structurally intact central vein, with intervening normal blood sinusoids. **Section B and C** represent liver sections from rats exposed to atrazine at concentrations of 0.013 µg/L and 0.172 µg/L, respectively. These sections demonstrate mild histopathological alterations, including congestion of the central vein and a mixed population of hepatocytes comprising morphologically normal cells (white arrows) alongside degenerating hepatocytes characterized by cytoplasmic vacuolation and pyknotic nuclei (black arrows).

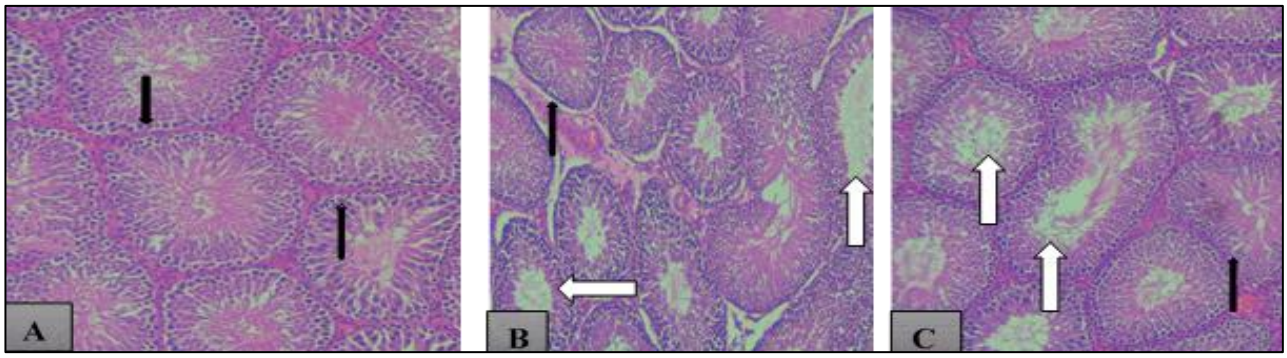

Figure 2. Representative hematoxylin and eosin (H&E) stained histological sections of rat testicular tissue following 120 days of treatment, examined at ×200 magnification. Section A depicts the testicular architecture of the negative control group administered distilled water, showing normal seminiferous tubules (black arrow) with an intact germinal epithelium composed of multiple orderly layers of spermatogenic cells. Sections B and C represent testicular sections from rats exposed to atrazine at concentrations of 0.013 µg/L and 0.172 µg/L, respectively. These sections demonstrate a heterogeneous pattern comprising both morphologically normal seminiferous tubules with preserved germinal epithelium (black arrows) and tubules exhibiting mild germinal epithelial erosion originating centrally (white arrows).

Assessment of sperm cell parameters revealed no statistically significant differences between atrazine-treated rats and the negative control group. Although the proportion of dead spermatozoa was slightly higher in both the 0.013 µg/L and 0.172 µg/L treatment groups, the proportion of viable spermatozoa was correspondingly slightly reduced compared to controls. Similarly, the percentage of spermatozoa with normal morphology showed a minor decline in atrazine-exposed rats, while no significant differences were observed in the prevalence of morphologically abnormal sperm cells (Table 3). Progressive sperm motility was also slightly higher in the control group relative to the treated groups. Furthermore, total sperm counts were modestly, but not significantly, reduced in rats exposed to 0.013 µg/L and 0.172 µg/L atrazine compared to the negative control.

Table 3. Sperm cell parameters of rats (n=15) 120 days of exposure to low-dose atrazine.

Sperm parameter	Negative control	Atrazine- treated (0.013 µg/L)	Atrazine- treated 0.172 µg/L	P-value
Dead spermatozoa (%)	22.59±6.26	48.28±10.758	47.16±10.82	0.148
Live spermatozoa (%)	69.45±3.29	51.58±10.775	52.90±10.72	0.402
Progressive motile spermatozoa (%)	50±11.18	45±9.354	40±10.00	0.789
Sperm cell counts (×10^{6} spermatozoa/mL)	91.80±8.29	61±6.272	75.40±9.081	0.056
Normal-shaped spermatozoa (%)	63.50±9.97	46.8±13.130	60.826±11.408	0.613
Abnormally shaped spermatozoa (%)	35.5±10.05	53.2±13.131	39.12±11.437	0.613

Discussion

This study evaluated the effects of prolonged exposure to low-dose atrazine contamination in drinking water on liver and testicular weights, histopathological architecture, and sperm cell parameters in a rat model. A significant increase in body weight was observed in rats exposed to 0.172 µg/L atrazine compared with those receiving 0.013 µg/L and the negative control. In contrast, a significant reduction in both liver and testicular weights was observed in rats exposed to 0.013 µg/L and 0.172 µg/L compared with the control group. Histopathological examination revealed only mild morphological alterations in hepatic and testicular tissues compared with the negative control.

In the liver, these changes were characterized by mild congestion of the central vein and a mixture of morphologically normal hepatocytes alongside degenerating cells exhibiting cytoplasmic vacuolation and pyknotic nuclei. Similarly, testicular sections showed predominantly intact seminiferous tubules with preserved germinal epithelium, although occasional tubules exhibited mild germinal epithelial erosion originating centrally. Comparable findings have been documented in previous studies [21,22]. For instance, [22] reported non-significant alterations in body weight in quails exposed to atrazine at 50, 250, and 500 mg/kg. In contrast, [21] observed significant reductions in liver weight and altered gene expression profiles related to lipid metabolism and androgen biosynthesis following atrazine exposure (5 mg/kg body weight/day), accompanied by hepatic steatosis. Similarly, disruptions in seminiferous tubular architecture consistent with endocrine disruption have been reported at higher doses [23]. In the present study, atrazine concentrations of 0.013 µg/L and 0.172 µg/L did not produce statistically significant changes in sperm parameters, including motility, viability, morphology, or total sperm count, when compared with controls. These findings differ from those of [34,35], who reported significant impairments in sperm quality following exposure to substantially higher atrazine doses. The discrepancies are most likely attributable to differences in exposure concentration and duration. Overall, the mild histopathological changes observed suggest that even low-level chronic atrazine exposure may induce subtle tissue responses in sensitive organs; however, extrapolation to human health outcomes should be made cautiously due to species-specific differences and exposure conditions. The main limitation of this study was the small sample size of five animals per experimental group. The numbers were minimized in accordance with ethical considerations; therefore, the findings should be considered preliminary and interpreted with caution. Future studies should use relatively larger sample size for confirmation of the observed effects.

Conclusion

Prolonged exposure to low-dose atrazine was not associated with statistically significant alterations in liver and testicular histology or sperm parameters under the conditions investigated. However, further studies with larger sample sizes are needed to determine whether prolonged exposure to low-dose atrazine may cause mild hepatic or reproductive effects and to better establish their relevance to human environmental exposure.

Abbreviation

SUA: Sokoine University of Agriculture; WHO; World Health Organization; SARU: Small Animal Research Unit; H & E: Hematoxylin and eosin stains; cv: Central vein; pv: Portal vain; 0C: Degrees Celsius; χ2: Chi-square; µg/L: Microgram per liter; mg/kg: Milligram per kilogram.

Declaration

Acknowledgment

Authors acknowledge the valuable assistance of Mr. Jafari Madaraka, a laboratory technician in the Department of Veterinary Anatomy and Pathology at SUA, for his assistance in laboratory work.

Funding

Authors received no financial support to conduct this study.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request by emailing usamwange@sua.ac.tz.

Authors' contributions

All authors contributed equally in designing the study, collecting the data, performing the statistical analyses, interpreting of the data, critically revising the manuscript, and approving the final version. All authors read and approved the final manuscript.

Ethics approval and consent to participate

We conducted the research in accordance with the Declaration of Helsinki. This study was reviewed and approved by the Research Ethics Review Committee of Sokoine University of Agriculture (clearance number DPRTC/R/186/64) on January 20, 2026, prior to the commencement of the research. All animal welfare guidelines were followed during animal handling, restraint, and experimentation. Animals were housed in plastic cages with a wired top cover that allowed adequate spacing and ventilation. The rats were acclimatized to standard environmental conditions of 25-30 °C, relative humidity of 65-75%, and a 12-hour light-dark cycle. Animals were fed broiler mash and provided with water ad libitum. At the end of the experiment, the animals were sacrificed under general anesthesia using ethyl ether. All experiments were conducted at the Small Animals Research Laboratory, which is specifically designed for small animal experimentation.

Consent for publication

Not applicable

Competing interest

The authors declare that they have no competing interests.

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