Original Article

Liver Histopathology of Trypanosoma-Infected Albino Rats when Treated with *Zingiber Officinale* and *Curcuma Longa*

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Abstract

This study evaluated the hepatoprotective effects of *Zingiber officinale* and *Curcuma longa* on liver histopathology in *Trypanosoma brucei brucei*-infected albino rats. A total of 25 rats were randomly assigned into five groups: uninfected control, infected untreated control, and three infected groups treated with ginger, turmeric, and a combination of both, respectively. Following infection and treatment, liver tissues were harvested, processed, and examined histologically using standard staining procedures. Data were analyzed qualitatively based on microscopic observations. Histopathological findings revealed normal hepatic architecture in the uninfected control group, while the infected untreated group showed severe vascular congestion, hepatocellular degeneration, and inflammatory infiltration. The ginger-treated group exhibited persistent pathological changes similar to the untreated group, indicating limited hepatoprotective effect. In contrast, the turmeric-treated group showed marked restoration of hepatic architecture with minimal inflammation, suggesting significant hepatoprotective and anti-inflammatory activity. The combined treatment group demonstrated moderate improvement but did not achieve the level of recovery observed with turmeric alone. These findings indicate that *Curcuma longa* possesses superior hepatoprotective potential against trypanosome-induced liver damage compared to *Zingiber officinale*.

Keywords: *Zingiber officinale*, *Curcuma longa*, liver histopathology, albino rats, *Trypanosoma brucei brucei*

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Introduction

The liver is the largest internal organ and a central metabolic hub, playing indispensable roles in nutrient metabolism, detoxification, immune surveillance, and biosynthesis of essential biomolecules. Its functional versatility is underpinned by a highly specialized and intricately organized histological architecture that enables efficient coordination between vascular, biliary, and cellular components[1]. Histological evaluation of the liver therefore provides critical insights into both physiological homeostasis and pathological alterations, making it a cornerstone of experimental and clinical hepatology[2]. At the microscopic level, the liver is composed of parenchymal and non-parenchymal elements arranged into functional units commonly described as hepatic lobules, portal lobules, or acini[3]. The hepatic parenchyma is predominantly formed by hepatocytes, which constitute approximately 80% of the liver mass and exhibit extensive metabolic capacity due to their rich cytoplasmic organelle content[3]. These cells are organized into plates radiating from the central vein, forming a highly ordered structure that facilitates efficient exchange of metabolites, nutrients, and waste products[4].

Trypanosomiases can be significant diseases of domestic and wild mammals, affecting millions of livestock in Africa, the Americas and Asia, with humans mostly in the tropics, like Africa and Latin America[5]. These pathogenic trypanosomes mainly inhabit the host's blood and lymph, but do occur sometimes in the cerebrospinal fluid (CSF) and other host tissues, and some of them have intracellular stages[6]. The parasite causes tissue damage by utilization of metabolites, excretion of toxic substances, mechanical damage to the host's tissue and immune mediated injuries and put strain on such organs as the liver[7].

Recent advances in liver histology have increasingly emphasized the dynamic interplay between cellular architecture and function, particularly in the context of disease[1]. Alterations in sinusoidal structure, endothelial function, and hepatocyte organization have been linked to a wide spectrum of liver pathologies, including fibrosis, cirrhosis, and metabolic liver diseases[8]. Histological assessment remains essential for understanding these pathological processes, as structural disruptions often precede clinical manifestations and biochemical changes[2]. Furthermore, emerging technologies such as three-dimensional tissue modeling and advanced imaging have enhanced our understanding of liver microanatomy and its relationship to physiological and pathological states[9 10].

Two important plants that could be of great boosting effect on the liver –ginger (*Zingiber officinale*) and turmeric (*Curcuma longa*). Both raw turmeric and ginger are naturally loaded with antioxidants and nutrients. These properties facilitate the healing of several ailments and common health issues by improving different organ function. Since they possess these properties, they serve to reduce inflammation, support immune system, and improve blood flow and digestion, as well as maintain a good body weight and organ function[11 12].

The implications of this study for public health are particularly significant in regions where trypanosomiasis remains endemic and healthcare resources are limited. In many parts of sub-Saharan Africa, including rural communities in Nigeria, the burden of parasitic infections is compounded by limited access to early diagnostic tools such as histopathology, leading to delayed detection of liver damage and poor clinical outcomes[13]. Understanding liver histoarchitecture in the context of trypanosome infection can enhance early disease recognition and inform targeted interventions. Moreover, the exploration of accessible, plant-based therapeutic agents like ginger and turmeric offers a cost-effective, culturally acceptable strategy for mitigating hepatic injury and improving overall health[14]. Integrating such evidence into public health frameworks could support preventive healthcare, reduce disease burden, and strengthen resilience in low-resource settings where conventional treatments may be scarce or unaffordable.

Materials and Methods

Study Area

This research study was conducted at the Zoology Research Centre in the Faculty of Biosciences, Nnamdi Azikiwe University, Awka, Anambra State, and lasted for a period of five (5) weeks.

Ethical Clearance

The study adhered to ethical guidelines for the ethical treatment of animals during capture, handling, and sample collection. Necessary permits were obtained from the Animal Research Committee of Nnamdi Azikiwe University, Awka. The certificate with ref. no.; NAU/AREC/2024/ 010 5.

Trypanosome Parasite Procurement and Management: *T. brucei brucei*parasites were obtained with permission from the trypanosome bank of NITR, head office, Kaduna. The parasites were then inoculated into two rats and transported down to Nnamdi Azikiwe University, Awka in a transportation box measuring 40 x 20 x 20 cm3 to ensure adequate ventilation.

Trypanosome Inoculation: One millilitre (1 ml) of *T. brucei brucei*-infected blood was taken from a donor rat and diluted with normal saline. The diluted blood containing approximately 1.25 x 10^6 parasites was used to inoculate the experimental animals from groups B – E via the intraperitoneal route[12]. Parasitaemia in the experimental animals was microscopically estimated on day three post-infection (PI), and the test confirmed wriggling movement of approximately above 1.25 x 10^6 Trypanosoma parasites[15 11].

Animals Procurement and Experimental Design: A total of 25 male albino Wistar rats at about 5 – 6 weeks old weighing between 60 – 70 grams obtained from the Faculty of Veterinary Sciences, University of Nigeria, Nsukka were used for the study. The rats were transported to the research station in a transportation box measuring 40 x 20 x 20 cm3 to ensure adequate ventilation. The rats were acclimatized for two weeks before the experiment began and randomly housed in 5 stainless steel metabolic cages laid out in a complete randomized design (CRD) of five treatments and fed with commercial food (Vital Feed Broiler Starter, 18.00 ± 0.50 g/100 g crude protein, and 2106.00 kcal/kg metabolizable energy, Vital Feed, Grand Cereals Limited, Jos, Plateau State, Nigeria) and water two times daily (9am and 5pm)[11].

The experimental rats were of homogenous sizes and randomly stocked into one cage (24 x 24 cm2) per treatment at the rate of five rats per cage. The treatments were labelled A – E as follows; Group A, positive control (uninfected rats, fed 1000 g of chick mash), Group B, negative control (infected rats, fed 1000 g of chick mash), Group C (infected rats, fed with 10 g of *Z. officinale* mixed in 1000 g of chick mash), Group D (infected rats, fed with 10 g of *C. longa* mixed in 1000 g of chick mash), and Group E (infected rats, fed with 5 g of *Z. officinale* and 5 g of *C. longa* mixed in 1000 g of chick mash). The ratio of supplement to feed was according to[16], who reported that the inclusion of 2% ginger powder, a polyphenolic compound, specifically 6-gingerol, enhances iron absorption in humans (as confirmed in the experimental Wistar rats), mitigate iron deficiency anaemia, and protect against iron-induced oxidative damage in various tissues. 2% inclusion per feed translates to 20 g/1000 g; making 10 g/1000 g very attainable.

Histological Analysis: Following necropsy, liver tissues were collected from representative animals in the control and experimental groups and immediately fixed in **10% neutral buffered formalin** for 24–48 hours. Histological processing was carried out at **IFEs Histopathology Laboratory, Awka, Anambra State**, using standard paraffin-embedding techniques. Briefly, tissues were dehydrated through graded ethanol, cleared in xylene, infiltrated with molten paraffin wax, and embedded in paraffin blocks. Sections of **4–5 µm** thickness were prepared using a rotary microtome, mounted on glass slides, and stained routinely with **hematoxylin and eosin (H&E)**. The stained sections were examined under a light microscope to evaluate hepatic

architecture, cellular integrity, and any histopathological lesions or tissue alterations using established histopathological criteria[17].

Semi-Quantitative Histopathological Scoring (Modified Ishak System): To enhance objectivity and reproducibility, liver histopathological changes were evaluated using a modified Ishak scoring system adapted for experimental animal studies. Hematoxylin and eosin (H&E)-stained liver sections were examined under a light microscope at magnifications of ×100 and ×400.The assessment focused on key indices of hepatic injury, including hepatocellular degeneration (vacuolation/ballooning), necrosis, inflammatory cell infiltration, sinusoidal congestion, and disruption of hepatic architecture. Each feature was graded on a scale of 0-4 based on severity and extent of involvement:

0 = No lesion (normal histology)

1 = Minimal change (focal involvement, <25% of tissue)

2 = Mild change (25-50% of tissue affected)

3 = Moderate change (50-75% of tissue affected)

4 = Severe change (>75% of tissue affected)

In addition, where applicable, fibrosis was evaluated separately using an adapted staging component of the Ishak system:

0 = No fibrosis

1 = Fibrous expansion of some portal areas

2 = Fibrous expansion of most portal areas

3 = Bridging fibrosis (portal-portal or portal-central)

4 = Cirrhosis or advanced fibrosis

For each specimen, at least five randomly selected, non-overlapping microscopic fields were analyzed, and the mean score for each parameter was calculated. A total histopathological score was obtained by summing the individual lesion scores for each animal.

Data Analysis: The data collected on the histopathology analysed via a qualitative (descriptive) where careful **visual examination of tissue sections** under a microscope was carried out; identification and description of **morphological features were examined and c**ompared with tissue structure between **control and treated groups. Also** a quantitative analysis where all slides were coded and assessed in a blinded manner by an experienced histopathologist to reduce observer bias. Data were expressed as mean ± standard error of the mean (SEM) for each experimental group and subjected to appropriate statistical analysis to determine intergroup differences. Tuckey post hoc test was used to determine the significant difference between groups.

Result

The liver section from Group A exhibited normal hepatic architecture, characterized by a well-defined central vein, radiating hepatic cords, and intact sinusoidal spaces. Hepatocytes appeared uniform, polygonal, and well-organized, with no evidence of cellular degeneration, necrosis, or inflammatory infiltration in either the hepatic lobules or portal triads. The absence of portal or lobular inflammation indicates preserved hepatic function and confirms the suitability of this group as a baseline control. Overall, this histological presentation is consistent with a healthy, non-injured liver, reflecting normal metabolic and detoxification capacity.

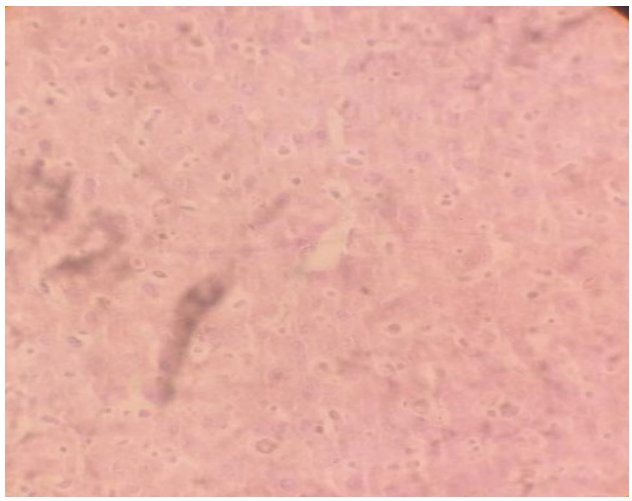

Plate 1: Histopathology of group A liver

Table 1. Semi-Quantitative Histopathological Scoring of Liver Sections in Group A – Modified Ishak System

Histopathological Parameter	Description of Lesion	Score
Vascular Congestion	No evidence of congestion; normal central vein and sinusoidal spaces	0
Inflammatory Cell Infiltration	No inflammatory cells observed in lobular or portal regions	0
Hepatocellular Degeneration	Hepatocytes uniform, polygonal, and well-organized	0
Hepatocellular Necrosis	No necrotic changes observed	0
Architectural Disruption	Intact hepatic cords and normal lobular arrangement	0
Cumulative Injury Score	**Overall absence of hepatic damage**	0

Liver sections from Group B demonstrated marked pathological alterations, including severe vascular congestion, particularly within the central veins and surrounding sinusoids. The hepatic lobules showed extensive inflammatory cell infiltration, with hepatocytes appearing swollen, distorted, and in some areas indistinguishable due to cellular damage. Inflammation extending into the portal areas suggests a diffuse inflammatory response, likely driven by infection-induced oxidative stress and immune activation. These features indicate acute hepatic injury, compromised microcirculation, and potential impairment of hepatocellular function. The extensive deviation from the normal architecture observed in Group A underscores the damaging impact of infection in the absence of therapeutic intervention.

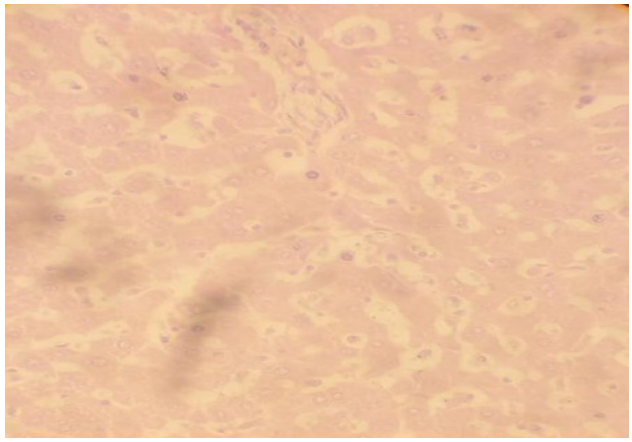

Plate 2: Histopathology of group B liver

Table 2. Semi-Quantitative Histopathological Scoring of Liver Sections in Group B – Modified Ishak System

Histopathological Parameter	Description of Lesion	Score
Vascular Congestion	Severe congestion of central veins and sinusoids, widely distributed	4
Inflammatory Cell Infiltration	Diffuse infiltration extending from lobular regions into portal areas	4
Hepatocellular Degeneration	Marked swelling, distortion, and loss of hepatocyte integrity	3.5
Hepatocellular Necrosis	Areas of cell loss with indistinguishable hepatocytes	3
Architectural Disruption	Severe distortion of hepatic lobules and loss of normal arrangement	4
Cumulative Injury Score	**Overall severity of hepatic damage**	18.5

The liver section from Group C revealed persistent pathological features, including vascular congestion and inflammatory changes within both the central vein and hepatic lobules. Hepatocytes appeared disorganized and poorly defined, suggesting ongoing cellular degeneration and incomplete tissue recovery. Although treatment with *Zingiber officinale* was administered, the histological architecture remained closely comparable to that of the untreated infected group (Group B), indicating that the ginger treatment alone provided limited hepatoprotective or anti-inflammatory effect under the conditions of this study. The continued presence of inflammation suggests insufficient mitigation of infection-induced hepatic injury.

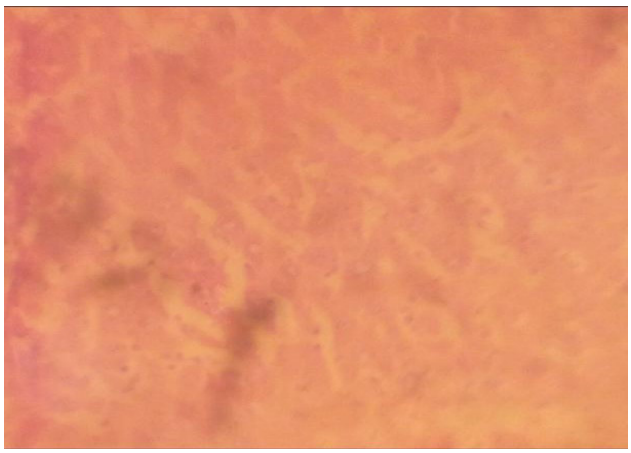

Plate 3: Histopathology of group C liver

Table 3. Semi-Quantitative Histopathological Scoring of Liver Sections in Group C – Modified Ishak System

Histopathological Parameter	Description of Lesion	Score
Vascular Congestion	Persistent congestion in central vein and sinusoids	3.5
Inflammatory Cell Infiltration	Ongoing inflammatory changes in lobular and vascular regions	3.5
Hepatocellular Degeneration	Disorganized, poorly defined hepatocytes indicating cellular degeneration	3
Hepatocellular Necrosis	Evidence of tissue injury with partial cellular loss	2.5
Architectural Disruption	Distortion of hepatic arrangement, though slightly less than Group B	3
Cumulative Injury Score	**Moderate to severe hepatic damage**	**15.5**

In contrast, liver sections from Group D showed substantial histological improvement. The hepatic architecture closely resembled that of the normal control, with a clearly defined central vein, preserved hepatocyte arrangement, and largely intact sinusoidal spaces. Only minimal inflammatory changes were observed in the portal region, with no significant lobular inflammation or hepatocellular degeneration. This near-normal appearance indicates that *Curcuma longa* exerted a pronounced hepatoprotective and anti-inflammatory effect, likely through modulation of oxidative stress, inflammatory mediators, and cellular repair mechanisms. The findings suggest effective restoration of hepatic structural integrity compared to Groups B and C.

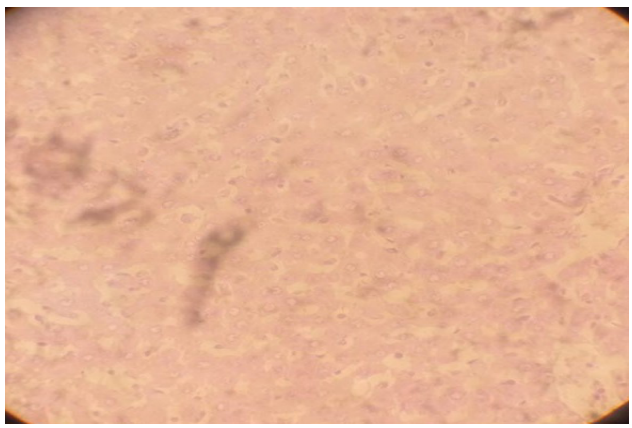

Plate 4: Histopathology of group D liver

Table 4. Semi-Quantitative Histopathological Scoring of Liver Sections in Group D – Modified Ishak System

Histopathological Parameter	Description of Lesion	Score
Vascular Congestion	No significant congestion; normal vascular architecture	0.5
Inflammatory Cell Infiltration	Minimal inflammatory cells confined mainly to portal areas	1
Hepatocellular Degeneration	Hepatocytes largely normal with preserved morphology	0.5
Hepatocellular Necrosis	No evidence of necrosis observed	0
Architectural Disruption	Hepatic lobular structure closely resembles normal control	0.5
Cumulative Injury Score	**Minimal hepatic injury / near-normal histology**	**2.5**

Liver sections from Group E showed a mixed histopathological outcome. While the central vein appeared relatively clear, there were still inflammatory vessels and areas of tissue damage evident within the hepatic parenchyma. The persistence of inflammation and structural disruption indicates that the combined treatment did not achieve the level of hepatic recovery observed with *C. longa* alone. Notably, the histological features in this group remained more similar to the untreated infected group (Group B) than to the normal control, suggesting a possible lack of synergistic hepatoprotective interactionbetween *Z. officinale* and *C. longa* in this experimental context. This outcome may reflect dose interactions, pharmacodynamic interference, or insufficient anti-inflammatory potency of the combined regimen.

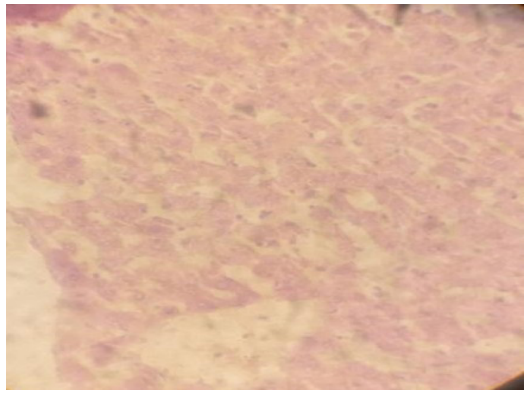

Plate 5: Histopathology of group C liver

Table 5. Semi-Quantitative Histopathological Scoring of Liver Sections in Group E – Modified Ishak System

Histopathological Parameter	Description of Lesion	Score
Vascular Congestion	Mild to moderate vascular alterations; central vein relatively clear but some congestion present	2.5
Inflammatory Cell Infiltration	Persistent inflammatory vessels and parenchymal infiltration	3
Hepatocellular Degeneration	Areas of tissue damage with partial hepatocyte degeneration	2.5
Hepatocellular Necrosis	Focal necrotic changes present	2
Architectural Disruption	Partial disruption of hepatic architecture; not fully restored	2.5
Cumulative Injury Score	**Moderate hepatic injury; closer to infected untreated group than control**	**12.5**

ANOVA Analysis of Semi-Quantitative Histopathological Scoring of Liver Sections in all groups – Modified Ishak System

One-way ANOVA revealed that treatment significantly influenced the severity of liver damage. Based on Tukey post-hoc analysis, Group D was the most efficacious treatment, reducing cumulative injury scores to levels statistically comparable with healthy controls. Group E conferred significant but partial protection, while Group Cproduced the least amelioration. These findings provide histopathological evidence supporting the hepatoprotective potential of turmeric, with Group D showing the greatest therapeutic promise.

Table 6. ANOVA Analysis of Semi-Quantitative Histopathological Scoring of Liver Sections in all groups – Modified Ishak System

Group	Histopathological Parameters	Damage score
Group A	5	0.00 ± 0.00^{a}
Group B	5	3.70 ± 0.20^{c}
Group C	5	3.10 ± 0.19^{bc}
Group D	5	0.50 ± 0.16^{a}
Group E	5	2.50 ± 0.16^{b}

Similar superscripts are not significantly different at $p>0.05$

Discussion

The present study combined qualitative histological observations with semi-quantitative scoring to evaluate hepatic alterations in *Trypanosoma brucei brucei*-infected rats and the effects of phytotherapeutic interventions. The untreated infected group (Group B) exhibited severe hepatic injury, characterized by marked vascular congestion, extensive inflammatory infiltration, hepatocellular degeneration, and architectural distortion. These findings were supported by a high cumulative injury score (18.5), confirming significant liver damage associated with trypanosome infection. This observation aligns with previous reports that trypanosome infections induce hepatic injury through oxidative stress, inflammatory responses, and metabolic disruption.

The semi-quantitative scoring further revealed that treatment with *Zingiber officinale* (Group C) resulted in only a modest reduction in injury severity (score: 15.5), with persistent congestion, inflammation, and hepatocellular damage. Histologically, this group closely resembled the untreated infected group, suggesting limited hepatoprotective effect under the experimental conditions. This contrasts with studies such as[18,19], which reported near-normal hepatic architecture following ginger treatment. Differences in dosage, duration, or disease model may account for this discrepancy. In contrast, *Curcuma longa*-treated rats (Group D) demonstrated substantial histological improvement, reflected by near-normal hepatic architecture and a markedly reduced cumulative injury score (2.5), comparable to the control group. Minimal inflammatory infiltration and preserved hepatocyte morphology were observed, indicating a reduction in tissue damage. These findings are consistent with previous studies[20] reporting the tissue-protecting and anti-inflammatory properties of turmeric, potentially mediated through antioxidant activity and modulation of inflammatory pathways. The combined treatment group (Group E) showed moderate improvement (score: 12.5), with reduced but persistent inflammation and structural disruption. Although some recovery was evident compared to the untreated group, the level of improvement did not reach that observed in the turmeric-treated group. This suggests that the combination of *Z. officinale* and *C. longa* did not produce an additive or synergistic effect under the conditions of this study; although combination of other phytogenics in another study[21] produced positive result.

Statistical analysis using one-way ANOVA and Tukey post hoc test confirmed significant differences in cumulative injury scores among groups, with Group D showing significantly lower scores compared to Groups B, C, and E ($p < 0.05$), and

values comparable to the control group. These results provide quantitative support for the histological observations and reinforce the relative differences in treatment effects. Overall, the integration of semi-quantitative scoring with histological assessment improved the objectivity of the findings and allowed for clearer differentiation of treatment outcomes across experimental groups.

Conclusion

The study concludes that treatment with *Curcuma longa* was associated with lower histopathological injury scores and preservation of hepatic architecture relative to infected untreated animals, while *Zingiber officinale* showed comparatively limited improvement under the conditions of this study. The combined treatment produced moderate effects but did not demonstrate enhanced outcomes compared to *C. longa* alone. These findings indicate differential responses to the tested plant treatments; however, further studies are required to clarify underlying mechanisms, optimize dosing, and evaluate broader therapeutic relevance.

Conflict of interest: Nil

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References

1. Xu X, Su J, Zhu R, Li K, Zhao X, Fan J, Mao F. From morphology to single-cell molecules: high-resolution 3D histology in biomedicine. Mol Cancer. 2025 Mar 3;24(1):63.
2. Choi JH. Histological and molecular evaluation of liver biopsies: A practical and updated review. Int J Mol Sci. 2025 Aug 10;26(16):7729.
3. Hassani M. Liver structure, function and its interrelationships with other organs: a review. Int J Dent Med Sci Res. 2022;4(1):88-92.
4. Nagy P, Thorgeirsson SS, Grisham JW. Organizational principles of the liver. In: The Liver: Biology and Pathobiology. 2020 Feb 12. p. 1-3.
5. Pérez-Molina JA, Molina I. Chagas disease. Lancet. 2018 Jan 6;391(10115):82-94.
6. Desquesnes M, Gonzatti M, Sazmand A, Thévenon S, Bossard G, Boulangé A, Gimonneau G, Truc P, Herder S, Ravel S, Sereno D. A review on the diagnosis of animal trypanosomoses. Parasites Vectors. 2022 Feb 19;15(1):64.
7. Ufele AN, Njoku OO. Effect of Vitamin C on the packed cell volume of trypanosome infected rats. Zoologist (The). 2009;7.
8. Gracia-Sancho J, Caparrós E, Fernández-Iglesias A, Francés R. Role of liver sinusoidal endothelial cells in liver diseases. Nat Rev Gastroenterol Hepatol. 2021 Jun;18(6):411-31.
9. Segovia-Miranda F, Morales-Navarrete H, Kücken M, Moser V, Seifert S, Repnik U, Rost F, Brosch M, Hendricks A, Hinz S, Röcken C. Three-dimensional spatially resolved geometrical and functional models of human liver tissue reveal new aspects of NAFLD progression. Nat Med. 2019 Dec;25(12):1885-93.
10. Wang Q, Liu J, Yin W, Wang A, Zheng J, Wang Y, Dong J. Microscale tissue engineering of liver lobule models: advancements and applications. Front BioengBiotechnol. 2023 Dec 8;11:1303053.
11. Ndinyelum OM, Ufele-Obiesie AN. Effects of *Zingiber officinale* and *Curcuma longa* on the liver function of albino rats infected by *Trypanosoma brucei brucei*. Anim Res Int. 2024;21(3):5819-27.
12. Ufele-Obiesie AN, Ndinyelum OM. Some haematological changes in *Trypanosoma*-infected albino rats when treated with *Zingiber officinale* and *Curcuma longa*. Int J ContempMicrobiol. 2025 Jan 1;11(1).
13. Ayeh-Kumi PF, Tetteh-Quarcoo PB, Aryee IN, Baddoo PNA, Ocansey JT, Otoboah MK. Navigating the parasitic landscape: insights into infection patterns and public health strategies in West Africa. Trop Med Infect Dis. 2025;10(5):125.
14. Tekade RK, Saraf S, editors. Biomolecular and Safety Considerations of Phytopharmaceuticals. Boca Raton: CRC Press; 2026.
15. Quintana JF, Chandrasegaran P, Sinton MC, Briggs EM, Otto TD, Heslop R, Bentley-Abbot C, Loney C, de Lecea L, Mabbott NA, MacLeod A. Single cell and spatial transcriptomic analyses reveal microglia-plasma cell crosstalk in the brain during *Trypanosoma brucei* infection. Nat Commun. 2022 Sep 30;13(1):5752.
16. Aniekwensi PO, Peter AK, Okore NK, Wahab YA, Aladapo OB, Alawode MO, et al. Haemoprotective effects of dry ginger powder (*Zingiber officinale*) on Wistar rats fed with iron treated water. Asian J Res Med Pharm Sci. 2024;13(2):79-90.

17. Mayhew WW, Wright SJ. Seasonal changes in testicular histology of three species of the lizard genus *Uma*. J Morphol. 1970 Feb;130(2):163-85.

18. Al-Muswie RT, Al-rekab EA, Al-Asadi NA. Histological effect of *Curcuma longa* and *Zingiber officinale* on the liver damage after treatment with hydrogen peroxide in rats (*Rattus norvegicus*). UnivThi-Qar J. 2018;13(1):113-126.

19. Sakina R, Norman N, Paviethira P, Halim S. Effects of herbal medicine (turmeric, cinnamon, ginger) on histopathology and gene expression in diabetic induced rats: a qualitative meta-analysis. Pharm Biosci J. 2022 May 21:01-8.

20. Fischer E, Otu OEC, Agbor CA, Raymond AEM, Umoh EA, Effiong OE, Jumbo SB, Asuquo JO, Ogon BE, Anwana NE. Testicular histomorphometric parameters, reticular fibres expression and antioxidant markers assessment on testicular tissue of methamphetamine-exposed Wistar rats treated with *Curcuma longa*. F1000Research. 2026;15:57.

21. Rume PO, Preye TM, Iwuoha BC, Okeke JJ, Wahedi JA. Effect of *Cymbopogon citratus* (Lemon Grass) extract and diminazene aceturate (Berenil) on *Trypanosoma brucei brucei* in vivo. South Asian J Parasitol. 2022 Apr 20;5(3):98-105.