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Effect of *Averrhoa carambola L.* (Carambola) fruit juice against ethanol-induced liver injury in mice

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ABSTRACT

Introduction: Alcoholic liver disease (ALD) is a leading cause of morbidity and mortality worldwide, with oxidative stress as the central pathogenic mechanism. The search for nutritional agents capable of modulating this response has focused on fruits such as carambola (*Averrhoa carambola L.*), which is rich in polyphenolic compounds with recognized antioxidant activity.

Objectives: To determine the effect of carambola fruit juice (*Averrhoa carambola L.*) on ethanol-induced liver damage in mice.

Materials and Methods: 35 albino mice were divided into five groups (n=7). The treatment groups received either A. carambola juice (5 and 15 mL/kg) or silymarin (100 mg/kg) for 5 days. All groups, except the negative control, received 5% ethanol ad libitum. The liver index, lipid peroxidation (LPO), antioxidant enzyme activity (SOD and CAT), and the GSH/GSSG ratio were evaluated, complemented by histopathological analysis.

Results: The ethanol control group showed a significant induction of LPO, hepatomegaly, and microvesicular steatosis. In contrast, carambola juice, especially at 15 mL/kg, significantly reduced LPO ($p<0.01$) and increased SOD and CAT activity. A substantial improvement in the GSH/GSSG ratio and an attenuation of cellular degeneration and steatosis were observed in the histological analysis, with an effect comparable to that of silymarin.

Conclusions: Carambola fruit juice, from *Averrhoa carambola L.*, exhibits a hepatoprotective effect against ethanol-induced liver damage in mice.

KEYWORDS

Averrhoa carambola L., hepatoprotection, oxidative stress, ethanol, clinical nutrition, polyphenols (Source: MeSH).

INTRODUCTION

Alcoholic liver disease (ALD) is one of the leading causes of morbidity and mortality worldwide, with excessive ethanol consumption representing its main etiological factor[1]. The clinical spectrum of ALD ranges from simple steatosis to cirrhosis and hepatocellular carcinoma, with pathogenesis converging on profound cellular dysfunction[2].

The principal mechanism of ethanol-induced liver injury involves the excessive generation of reactive oxygen species (ROS), primarily through the microsomal ethanol-oxidizing system (MEOS), mediated by cytochrome P450 2E1 (CYP2E1), and the subsequent formation of the toxic metabolite acetaldehyde[3]. The imbalance between ROS production and endogenous antioxidant defenses leads to an oxidative stress state directly responsible for lipid membrane damage (lipid peroxidation) and protein modification[4].

Oxidative stress substantially impairs hepatocellular antioxidant defenses, depleting reduced glutathione (GSH) stores and inactivating protective enzymes such as superoxide dismutase (SOD) and catalase (CAT)[5]. Mitochondrial

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injury, apoptosis and necrosis, and the inflammatory response that follows contribute to the progression of liver disease[6].

Given the limited availability of effective pharmacological therapies and the need for nutritional approaches, research has increasingly focused on functional and phytochemical-rich foods with potential hepatoprotective properties. The tropical fruit *Averrhoa carambola L.* (carambola) is notable for its high polyphenol content, including flavonoids (such as quercetin) and ascorbic acid, which exhibit potent antioxidant and anti-inflammatory activity in vitro[7].

Previous preclinical studies have shown that aqueous and ethanolic extracts of carambola leaves and stems protect against hepatotoxicity induced by agents such as carbon tetrachloride (CCl_4) and acetaminophen[8,9]. The fruit is recognized for its traditional uses and pharmacological activities[10]. However, there is a notable gap in studies assessing the effects of the **fresh fruit juice**, the form most commonly consumed in the diet, particularly in the context of ethanol-induced toxicity[11].

Therefore, the aim of this study was to evaluate the protective effect of fresh Averrhoa carambola L. fruit juice against ethanol-induced liver injury in mice, to provide scientific evidence supporting its potential application as an adjunctive strategy in clinical nutrition[12].

MATERIALS Y METHODS

Study Design and Ethical Considerations

A true experimental design with post-test and control group was used. The study was conducted in accordance with international guidelines for the care and use of laboratory animals[13]. The experimental protocol was reviewed and approved by the Ethics Committee of the Faculty of Medicine at the Universidad Nacional Mayor de San Marcos (UNMSM)[14].

Animals and Treatment Protocol

A total of 35 male albino mice (Rattus rattus var. albinus), 8 weeks of age and with an average body weight of 25.0 ± 2.0 grams, were used. The animals were acclimatized for one week prior to the beginning of the study and maintained under controlled environmental conditions. The mice were randomly assigned to five groups (n = 7 per group) using simple probabilistic sampling. The experimental treatment was administered for five consecutive days.

The treatment groups (IV and V) received fresh A. carambola juice orally (via gavage) at doses of 5 mL/kg (low dose) and 15 mL/kg (high dose), respectively, while the positive control group (Group III) received Silimarina (100 mg/kg)[15]. Groups I and II received 0.9% NaCl.

Liver damage was induced by providing 5% ethanol in the drinking water *ad libitum* to Groups II, III, IV, and V during the same 5-day period. Juice and Silimarina administration occurred one hour prior to daily ethanol exposure[16].

Juice Preparation

The A. carambola fruits were selected at optimal ripeness from local markets in Lima, Peru, and processed immediately. Fresh juice was obtained by blending and filtration, and the dosing preparations were made daily. The administered dose was determined based on the extrapolated human intake and the concentration of bioactive compounds, ensuring the delivery of a therapeutically relevant dose.

Sample Collection and Determination of the Hepatic Index

At the end of the experimental period, the animals were sacrificed under deep sedation. Final body weight was recorded, and the liver was excised, rinsed, and weighed to determine the Hepatic Index (HI), expressed as the percentage of liver weight relative to total body weight. One portion of the tissue was immediately fixed for histological analysis, while the remaining portion was frozen at −80°C for biochemical determinations.

Biochemical Determinations of Oxidative Stress

Frozen liver tissue was homogenized in cold phosphate buffer to obtain the supernatant, in which the following measurements were performed:

- **Superoxide Dismutase (SOD) and Catalase (CAT) Activity:** The activity of these primary antioxidant enzymes was assessed. SOD activity was measured based on its ability to inhibit nitroblue tetrazolium reduction, while CAT activity was determined by the rate of hydrogen peroxide decomposition[17,18].
- **Lipid peroxidation (LPO):** Levels of thiobarbituric acid reactive substances (TBARS) were measured as a marker of membrane lipid damage, using spectrophotometric analysis[19].
- **Reduced/Oxidized Glutathione Ratio (GSH/GSSG):** An enzymatic assay with DTNB was used to quantify glutathione levels and calculate the GSH/GSSG ratio, a sensitive indicator of the hepatocellular redox state[20].

Histopathology

Liver samples fixed in formaldehyde were processed and embedded in paraffin. Histological sections of 5 µm were obtained and stained with Hematoxylin and Eosin (H&E) for morphological evaluation. A blinded pathologist assessed the severity of steatosis, hydropic degeneration, and in-

flammation using a standardized semiquantitative scoring system.

Statistical Analysis

Data were analyzed using SPSS *software* (version 25.0). Data distribution was assessed using the Shapiro–Wilk test. Comparisons among the five groups were performed using one-way Analysis of Variance (ANOVA). When a significant ANOVA result was obtained, Tukey's *post-hoc* test was applied to identify specific pairwise differences. Results are presented as mean ± SD, and a p value < 0.05 was considered statistically significant.

RESULTS

Liver Index (LI)

The ethanol control group (Group II) showed a significantly higher hepatobiliary index (HI) (5.25 ± 0.35%) than Group I (3.50 ± 0.15%, p<0.001), confirming ethanol-induced hepatomegaly. Treatments with carambola juice attenuated this increase in a dose-dependent manner: the low-dose juice group (Group III) showed an HI of 4.78 ± 0.29% (p<0.05 vs. Group II), while the high-dose juice group (Group V) reduced it to 4.15 ± 0.21% (p<0.01 vs. Group II), a value comparable to the positive control (silymarin, 3.98 ± 0.18%).

Table 1. Liver index by treatment group

GROUPS	Liver index*	
	% LI	% Inhibition
Grupo I	4.55 ± 0.44	—
Grupo II	5.12 ± 0.44	—
Grupo III	4.73 ± 0.34	7.63
Grupo IV	4.58 ± 0.30	10.38
Grupo V	4.30 ± 0.39 (a)	15.95

* Prueba Shapiro-Wilk p<0,05. Prueba ANOVA. Media ± DE.
(a) p<) p<0,01 compared to group II.
* Analysis of variance - Tukey. Mean + SD.

Lipoperoxidation (LPO) / TBARS

TBARS levels, markers of LPO, increased more than threefold in Group II (2.15 ± 0.25 nmol MDA/mg protein) compared to the Negative Control. The juice intervention significantly mitigated this damage: Group V showed a reduction to 0.75 ± 0.11 nmol MDA/mg protein (p < 0.001 vs. Group II), indicating a decrease in oxidative damage and a potent direct antioxidant effect of the juice components.

Table 2. Lipid peroxidation levels in liver tissue by treatment group

GROUPS	Lipoperoxidation*	
	nmol/g tissue	% Inhibition
Grupo I	94.71 ± 22.70(a)	—
Grupo II	166.35 ± 53.40	—
Grupo III	54.84 ± 17.58 (a)	67
Grupo IV	81.78 ± 22.45 (a)	51
Grupo V	62.52 ± 8.80 (a)	62

* Prueba Shapiro-Wilk p<0,05. Prueba ANOVA. Media ± DE.
(a) p<) p<0,01 compared to group II.
* Kruskal Wallis. Median + IRQ.

Actividad de Enzimas Antioxidantes (SOD y CAT)[29]

Exposure to ethanol reduced SOD and CAT activity in Group II, indicating enzyme depletion due to oxidative stress. Carambola juice demonstrated the ability to preserve or restore enzyme activity:

- **SOD:** Group V (10.5 ± 1.2 U/mg protein) exhibited significantly higher activity than Group II (6.1 ± 0.8 U/mg protein, p < 0.01), reaching levels close to normal.
- **CAT:** Group V also maintained CAT activity (15.2 ± 2.1 U/mg protein) compared to the decline observed in Group II (8.8 ± 1.5 U/mg protein, p < 0.05), demonstrating the protection of the antioxidant defense system.

Reduced/Oxidized Glutathione Ratio (GSH/GSSG)

The GSH/GSSG ratio was drastically reduced in Group II (0.55 ± 0.08), reflecting the depletion of GSH essential for cellular detoxification. Juice treatment significantly restored this ratio: Group V reached a value of 1.30 ± 0.15 (p < 0.001 vs. CE), indicating an improvement in hepatic redox status by preserving glutathione reserves.

Histopathological Analysis

Histological sections from Group II showed severe liver damage characterized by diffuse microvesicular steatosis, intense hydropic degeneration, and foci of pericentral necrosis. In contrast, Groups IV and V exhibited remarkable improvement: Group V presented with an almost normal lobular architecture, minimal focal steatosis, and a drastic reduction in cellular degeneration. This histological finding validates the biochemical results, demonstrating that starfruit juice prevents ethanol-induced morphological alteration.

Table 3. Levels of superoxide dismutase and catalase enzyme activities in liver tissue by treatment group

GROUPS	SOD*		CAT **		SOD/CAT*	
	U/mg tissue	% Increase	U/mg tissue	% inhibition	U/mg tissue	% Increase
Grupo I	8.37 ± 1.11	—	0.21 ± 0.06	—	38.22 ± 1.36	—
Grupo II	8.56 ± 0.44	—	0.22 ± 0.03	—	39.66 ± 1.57	—
Grupo III	7.87 ± 0.82	-8.06	0.21 ± 0.05	-4.55	36.33 ± 1.10 (a)	8.41
Grupo IV	6.60 ± 0.35 (a)	-22.97	0.21±0.03	- 4.55	31.97 ± 0.44 (a)	19.39
Grupo V	7.21 ± 0.51 (a)	-15.79	0.22± 0.03	0.00	32.79 ± 2.17 (a)	17.33

(a) p<0,01 compared to group II. * Analysis of variance - Tukey.Mean + SD. ** Kruskal Wallis. Median + IRQ.

Table 4. GSH profile levels in liver tissue by treatment group

GROUPS	GSH*		GSH total**		GSH / GSSG*	
	μmol/g tissue	% Increase	μmol/g tissue	% inhibition	μmol/g tissue	% Increase
Grupo I	561.18 ± 159.12	—	1014.92 ± 240.64	—	1.25 ± 0.33	—
Grupo II	429.99 ± 86.32	—	889.81 ± 149.73	—	0.93 ± 0.08	—
Grupo III	681.27 ± 138.40 (a)	58.44	1194.62 ± 249.47 (a)	34.25	1.35 ± 0.22 (a)	45.53
Grupo IV	328.16 ± 90.80	-23.68	722.66± 113.68	- 18.78	0.84 ± 0.23	-9.89
Grupo V	485.04 ± 63.84	12.80	843.82± 102.05	-5.17	1.37 ± 0.20 (a)	47.15

(a) p<0,01 compared to group II. * Analysis of variance - Tukey. Mean + SD.

DISCUSSION

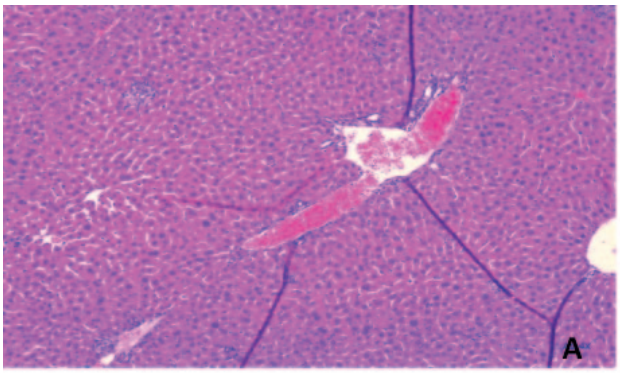
The results confirm the potential of Averrhoa carambola L. juice to exert a significant hepatoprotective effect in the model of acute ethanol-induced toxicity[21]. The mitigation of liver injury is evidenced by the attenuation of hepatomegaly (reduced liver index) and the reversal of biochemical and morphological alterations induced by oxidative stress[22].

The most robust finding is the significant and dose-dependent decrease in lipid peroxidation (LPO). This effect is attributed to the high concentration of scavenging compounds in the juice, mainly flavonoids (quercetin) and proanthocyanidins, which have been shown to efficiently neutralize reactive oxygen species (ROS)[23]. The chemical structure of these compounds enables them to donate electrons and stabilize free radicals generated by ethanol metabolism, thereby interrupting the lipid peroxidation chain reaction[24].

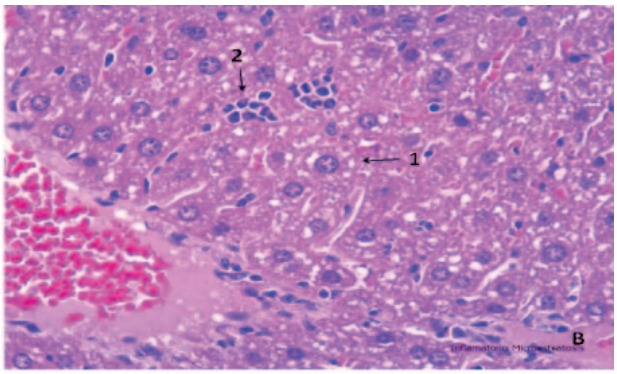
Preservation of the glutathione system, reflected in the maintenance of a favorable GSH/GSSG ratio, is crucial[14]. Glutathione (GSH) is essential for the conjugation of toxic metabolites and the elimination of H_2O_2 via glutathione peroxidase[25]. Our results suggest that the phytochemicals present in the juice, in addition to acting directly as antioxidants, may also allosterically modulate the activity of enzymes involved in the glutathione cycle or prevent the inactivation of glutathione reductase, thereby maintaining the cellular reserve[26].

At the molecular and morphological levels, the reduction of microvesicular steatosis and hepatomegaly in the groups treated with juice (particularly Group V) is a finding of great clinical relevance[14]. It is known that oxidative stress and acetaldehyde activate pro-lipogenic transcription factors and pro-inflammatory signaling pathways such as NF-κB[27]. Compounds such as quercetin, which is abundant in starfruit, are known for their ability to inhibit NF-κB activation and suppress the expression of genes involved in lipid accumulation, which could explain the histological attenuation of steatosis observed[28].

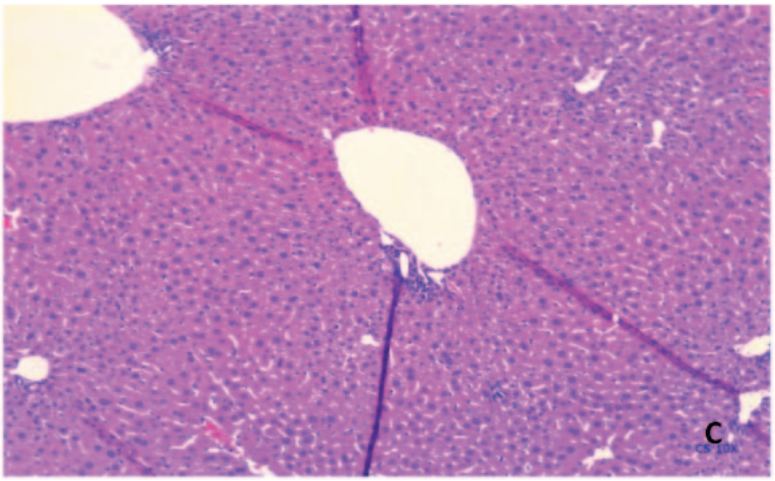
The importance of this study for Clinical Nutrition and Hospital Dietetics lies in the use of the complete food matrix (fresh juice) rather than purified extracts[29]. In the context of



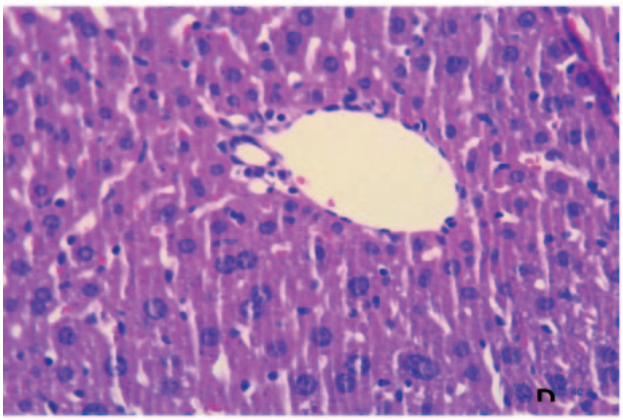
Group I: (A) Columns of hepatocytes with good distribution. Blood vessels with slight congestion. Portal spaces with interstitial edema. Stain: H-E (4X).



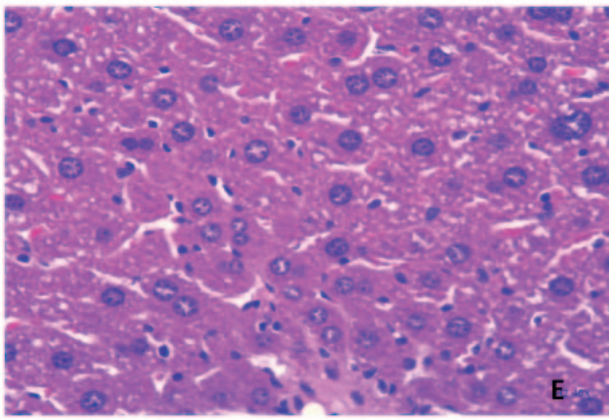
Group II: (B) Group II: (B) Columnar distortion of hepatocytes, hemorrhage and microsteatosis (1). Abundant Kupffer cells and lymphocytic infiltrate (2). Stain: H-E (40X).



Group III: (C) Non-aligned columns of hepatocytes, hemorrhagic areas. Vascular congestion. Abundant Kupffer cells and lymphocytes: Stain: H-E (10X).



Group IV: (D) Well-distributed columns of hepatocytes, mild venous congestion, microsteatosis. Increased Kupffer cells and lymphocytes. Stain: H&E (40X).



Group V: (E) Well-distributed columns of hepatocytes, microsteatosis, severe venous congestion. Kupffer cells and lymphocytes preserved. Stain: H-E (40X).

Figure 1. Photomicrograph of liver tissue in mice

prevention or nutritional co-management of patients at risk of alcoholic liver disease or steatosis, the dietary recommendation of fresh juice consumption represents a practical and sustainable intervention. The dose-dependent results (15 mL/kg being more effective than 5 mL/kg) suggest that the concentration of phytochemicals in the diet is a critical factor in achieving the therapeutic effect[14].

However, this study has limitations. The liver injury model was acute (5 days); a chronic exposure model (8–12 weeks) would be required to evaluate the effect of the juice on the prevention of hepatic fibrosis, a more advanced and clinically relevant stage of alcoholic liver disease (ALD). Furthermore, although the biochemical and histological evidence is strong, future studies should include the measurement of key inflammatory markers (TNF-α, IL-6) and the quantification of the expression of key metabolic enzymes (CYP2E1, ADH) to provide a more comprehensive molecular understanding of the protective mechanism[30].

In summary, the findings demonstrate that A. carambola L. juice is a potent hepatoprotective agent capable of restoring redox homeostasis and preserving hepatic morphological integrity in a model of ethanol-induced damage. This work lays the foundation for exploring the application of this tropical fruit in clinical nutritional therapy[14].

CONCLUSIONS

Oral administration of *Averrhoa carambola L.* fruit juice significantly prevents hepatomegaly and morphological damage, reducing signs of ethanol-induced steatosis and hydropic degeneration in mice.

The main protective mechanism is the modulation of oxidative stress, evidenced by a marked, dose-dependent reduction in lipid peroxidation (TBARS) and the restoration of endogenous antioxidant capacity (GSH/GSSG and SOD/CAT activity).

The hepatoprotective effect is most pronounced with a dose of 15 mL/kg, suggesting that consuming adequate amounts of the fresh juice may be a valuable adjunctive dietary strategy in the context of clinical nutrition for liver health.

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