ORIGINAL ARTICLE

CONTENTS

Effect of *Ocimum basilicum* herbs extract on pro-inflammatory cytokines in ethanol-induced liver damage in rats

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ABSTRACT

Aim: The aim of this study is to evaluate the hepatoprotective and immunomodulatory effects of *Ocimum basilicum* extract on pro-inflammatory cytokines, specifically TNF-α, IL-6, and IL-1β, in a rat model of ethanol-induced liver damage.

Materials and Methods: A total of 120 male rats were divided into four groups: a control group, an ethanol-induced liver damage group, a low-dose basil treatment group, and a high-dose basil treatment group. Pro-inflammatory cytokines (TNF-α, IL-6, IL-1β) were measured using ELISA.

Results: The ethanol exposure group showed significantly elevated TNF-α (548.8 ± 83.78 pg/mL), IL-6 (410.3 ± 167.7 pg/mL), and IL-1β (373.1 ± 127.7 pg/mL) compared to the control group. Basil supplementation, particularly at high doses (100 mg/kg), effectively reduced these cytokines, with TNF-α (316.4 ± 57.37 pg/mL) and IL-6 (133.9 ± 86.76 pg/mL) levels approaching control values. IL-1β was also significantly reduced (152.3 ± 79.37 pg/mL) but remained slightly elevated.

Conclusions: This study showed that *Ocimum basilicum* extract has potent hepato protective and anti-inflammatory properties, making it a promising natural therapeutic agent for alcohol-induced liver damage.

KEY WORDS: liver, TNF-α, *Ocimum basilicum*, cytokines, rats, bioactive compounds

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INTRODUCTION

Basil (*Ocimum spp.*) is known as a fragrant herb belonging to the Lamiaceae family; it is widely familiar for its role in medicinal and therapeutic uses [1]. Basil is currently grown all over the world for its fragrant leaves and extracts that are high in phytochemicals. Basil have numerous health benefits, and often known as holy basil because its species widely used in traditional medical systems, especially Ayurveda [2].

Basil is herbs considering as reservoir of bioactive compounds such as ursolic acid, eugenol, apigenin, rosmarinic acid and caryophyllene which have been known to be responsible for the diverse pharmacological and medicinal activities. Basil has been well for possessing strong anti-oxidant, anti-inflammatory, hepato-protective, anti-microbial and immunological activities [3]. All these activities of basil make it a subject of significant interest in modern pharmacological and medical research. Due to its rich phytochemical composition and extensive therapeutics, basil continues to be an area of intense focus of research within the field of herbal medicine science, pharmacology, and integrative-health.

Basil herb is relevant to the health of the liver function since it inhibits inflammation, prevents oxidative stress and detoxifying hepatocytes from negative effect of toxins, infections and medications [4]. It is confirmation of its efficacy as an adjunctive traditional therapeutic agent for liver damage and inflammatory disorders of a systemic nature via its potential to modulate inflammatory mediators, e.g., TNF-α (tumor necrosis factor alpha), IL-6 (interleukin-6), and IL-1β (interleukin-1 beta) [5].

Apart from this, Basil herb is a useful plant that improves the immune system of the body since it has the potential to increase the immune system. It keeps the immune system balanced, reduces the chances of over-inflammation and its role in recovery from infections and inflammatory conditions by controlling immunological response [6]. There are several reasons for damage to the liver such as alcohol intake, toxins, infection and autoimmune disorders. Perhaps most common is alcohol induced liver damage that consists

of inflammation, oxidative stress and dysfunctional liver function [7]. Due to its significant function of detoxification, the liver is highly susceptible to damage due to the accumulation of inflammatory mediators and toxic substances .

The immune-response has a main role in liver injury. Tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β) are examples of pro-inflammatory cytokines that are raised and contribute to chronic-inflammation, which damages tissue and accelerates the development of cirrhosis, fibrosis or liver failure [8-10].

AIM

The aim of this study is to evaluate the hepatoprotective and immunomodulatory effects of *Ocimum basilicum* extract on pro-inflammatory cytokines, specifically TNF-α, IL-6, and IL-1β, in a rat model of ethanol-induced liver damage.

MATERIALS AND METHODS

PREPARATION OF BASIL EXTRACTION

Fresh basil leaves are typically used which are supply form local farm in Samawah city, Leaves are rinsed thoroughly with distilled water to remove impurities. Then they are drying in a shaded, well-ventilated area at room temperature then grinded into a fine powder using a blender [11].

For the extraction of bioactive compounds from basil, 200 g of powdered basil was placed in a flask with 300 mL of ethanol (70%) was added. The mixture was heated in 70°C with stirring continuously for 4 hours to facilitate the extraction of phytochemicals from the basil powder. After the extraction, the mixture was filtered to separate the solid plant material from the liquid extract using standard filter paper.

The product was concentrated using a rotary evaporator vacuum under reduced pressure at a temperature of 40–50°C to remove the ethanol-solvent. It was washed with "diethyl ether" and the final extract was stored in dark containers in a cool, dry place, and refrigeration [12].

ANIMAL GROUPS

A total of 120 male rats, weighing approximately 250 g, were randomly divided into four groups, with 30 rats per group. These groups were as follows:

Group 1 Healthy, Untreated Control (UC): This group remained untreated and served as the baseline for comparison.

Group 2 Ethanol Treatment (EtOH): Rats in this group were given 10-20% ethanol in their drinking-water to induce liver damage and inflammation.

Group 3 Low Basil Dose + Ethanol (LB): Rats were treated with a low dose of basil-extract (50 mg/kg body weight/day) along with 10-20% ethanol in their drinking water.

Group 4 High Basil Dose + Ethanol (HB): Rats received a high dose of basil extract 100 mg/kg body weight/day) along with 10-20% ethanol in their drinking-water.

The rats received the treatment over the course of four weeks, during which period their behavior, body weight, and any indications of distress were observed. After the treatment period ended, the rats were euthanized, and blood was collected for immunological tests.

EVALUATION OF CYTOKINES

The pro-inflammatory cytokines (Tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β)) levels were measured to study the immunomodulatory activity of basil extract in "ethanol-induced liver injury". The blood was collected from every research group by heart puncture under anaesthesia at the end of the experiment. The serum was isolated by centrifugation at 3,000 rpm for 10 minutes and stored at -80°C until analysis [13]. Enzyme-linked immunosorbent assay (ELISA) kits were used to measure the levels of TNF-α, IL-6, and IL-1β. To guarantee the accuracy of the data, each experiment was repeated three times. Cytokine levels were used as markers of systemic inflammation, and they were measured in pg/mL.

STATISTICAL ANALYSIS

Data of cytokines were analyzed by one-way ANOVA and then post hoc comparisons were made by "Tukey's test". Data were presented as mean ± standard error (SE), and significance was considered at $p < 0.05$. These studies indicated immunomodulatory and hepatoprotective activity of basil extraction in "ethanol-induced liver damage" in rats [14].

RESULTS AND DISCUSSION

The serum TNF-α levels varied significantly among the experimental groups, as confirmed by one-way ANOVA analysis ($F = 8.053$, $P < 0.05$), indicating a significant effect of ethanol-induced liver damage and basil supplementation (Table 1). The inflammatory cytokines IL-6 and IL-1β followed a similar trend to TNF-α. One-way ANOVA analysis revealed a highly significant effect of ethanol and basil supplementation on IL-6 ($F = 55.82$, $P < 0.05$) and IL-1β ($F = 54.64$, $P < 0.05$), with both showing

Table 1. Statistical analysis of groups results according to ANOVA test

ANOVA summary	TNF-α	IL-6	IL-1β
F	8.053	55.82	54.64
P value	< 0.05	< 0.05	< 0.05
P value summary	***	***	***
Statistically significant? (P < 0.05)	Yes	Yes	Yes
R square	0.1724	0.5908	0.5856

Note: *Tumor necrosis factor-alpha (TNF-α), Interleukin-6 (IL-6) and Interleukin-1 beta (IL-1β), **P < 0.01, *P < 0.001, ***P < 0.0001
Source: compiled by the authors of this study

Table 2. Statistical analysis of groups, p value ≤ 0.05

Groups	N	Mean ± SD pg/mL	95% CI of diff.	Statistically significant? (P < 0.05)
Tumor necrosis factor-alpha (TNF-α)				
UC	30	288.4 ± 44.01		
EtOH	30	548.8 ± 83.78	-393.0 to -127.7	Yes (****)
LB+EtOH	30	449.3 ± 92.80	-293.5 to -28.23	Yes (*)
HB+EtOH	30	316.4 ± 57.37	-160.6 to 104.7	No
Interleukin-6 (IL-6)				
UC	30	70.53 ± 23.39		
EtOH	30	410.3 ± 167.7	-422.8 to -256.8	Yes (****)
LB+EtOH	30	366.8 ± 157.1	-379.3 to -213.2	Yes (****)
HB+EtOH	30	133.9 ± 86.76	-146.4 to 19.63	No
Interleukin-1 beta (IL-1β)				
UC	30	73.67 ± 26.21		
EtOH	30	373.1 ± 127.7	-364.1 to -234.8	Yes (****)
LB+EtOH	30	254.0 ± 116.7	-245.0 to -115.6	Yes (****)
HB+EtOH	30	152.3 ± 79.37	-143.3 to -13.95	Yes (*)

Note: US: Untreated group, EtOH: Ethanol treated group, LB+EtOH: Group treated with ethanol and low dose of extract, HB+EtOH: group treated with ethanol and high dose of extract, GraphPad prism results showed: P< 0.05, **P < 0.01, *P < 0.001, ****P < 0.0001.
Source: compiled by the authors of this study

strong statistical significance ($P < 0.05$, $R^2 = 0.5908$ and 0.5856, respectively) as showed in Table 1.

Tumor necrosis factor-alpha (TNF-α) is a key inflammatory cytokine involved in liver injury and immune response regulation [15]. In this study, ethanol exposure significantly elevated TNF-α levels, confirming its role in inducing hepatic inflammation. However, basil supplementation effectively mitigated this effect, with the high-dose basil (HB+EtOH) group showing TNF-α levels statistically similar to the control group, indicating strong protective activity.

The ethanol-only (EtOH) group exhibited a significant increase in TNF-α levels (548.8 ± 83.78 pg/mL, P < 0.0001) compared to the control (288.4 ± 44.01 pg/mL). This increase is well-documented in alcohol-induced liver damage, where ethanol metabolism generates oxidative stress, gut-derived endotoxins, and activation of Kupffer cells, all of which stimulate TNF-α production [17, 18]. Elevated TNF-α is a hallmark of alcohol-induced liver injury and a key driver of inflammation and hepatocyte apoptosis [19]. The low-dose basil (LB+EtOH, 50 mg/kg) group showed a significant reduction in TNF-α levels (449.3 ± 92.80 pg/mL, P < 0.05) compared to the ethanol-only group. This suggests that basil at this dose exerts anti-inflammatory effects, likely through its antioxidant and NF-κB inhibitory properties as illustrated in Table 2 and Fig. 1.

The high-dose basil (HB+EtOH, 100 mg/kg) group exhibited a greater reduction in TNF-α (316.4 ± 57.37 pg/mL). Importantly, this reduction was not statistically different from the control group (P > 0.05), indicating that basil at this dose was highly effective in counteracting ethanol-induced inflammation, restoring TNF-α levels to near-normal values. Unlike in the LB+EtOH group, where TNF-α levels were still significantly elevated compared to controls, the HB+EtOH group's TNF-α levels were statistically indistinguishable from the control group. This finding is highly favorable, as it suggests that high-dose basil extract was so effective in reducing inflammation that TNF-α levels remained

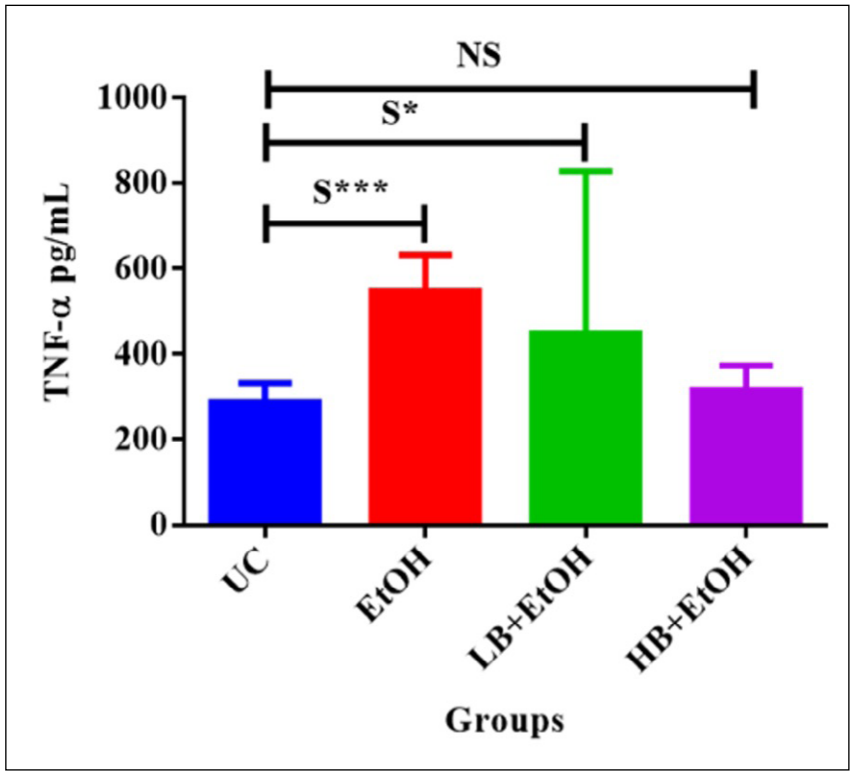

Fig. 1. Levels of TNF-**α** in the groups, P valie < 0.05; *P < 0.001; ***P < 0.0001
Picture taken by the authors

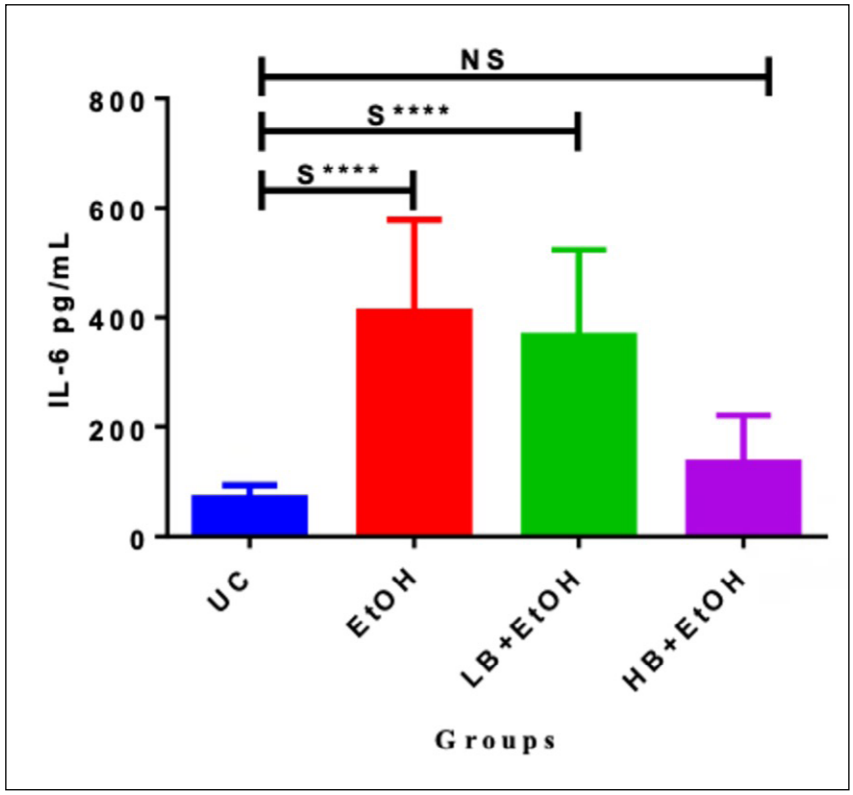

Fig. 2. Levels of IL-6 in the groups, P value < 0.05, ****P < 0.0001
Picture taken by the authors

stable, despite ethanol exposure. This indicates a strong protective effect, preventing TNF-α elevation and maintaining a normal inflammatory balance.

Hence, the absence of statistical significance between the HB+EtOH and control groups is not a defect but rather a confirmation of the high potency of basil against ethanol-induced inflammation. This supports the hypothesis that high-dose basil extract possesses strong hepatoprotective properties, with potential mechanisms involving antioxidant actions, suppression of pro-inflammatory signals, and regulation of immune activity [20].

Interleukin-6 (IL-6) is a pro-inflammatory cytokine responsible for immune modulation and liver inflammation

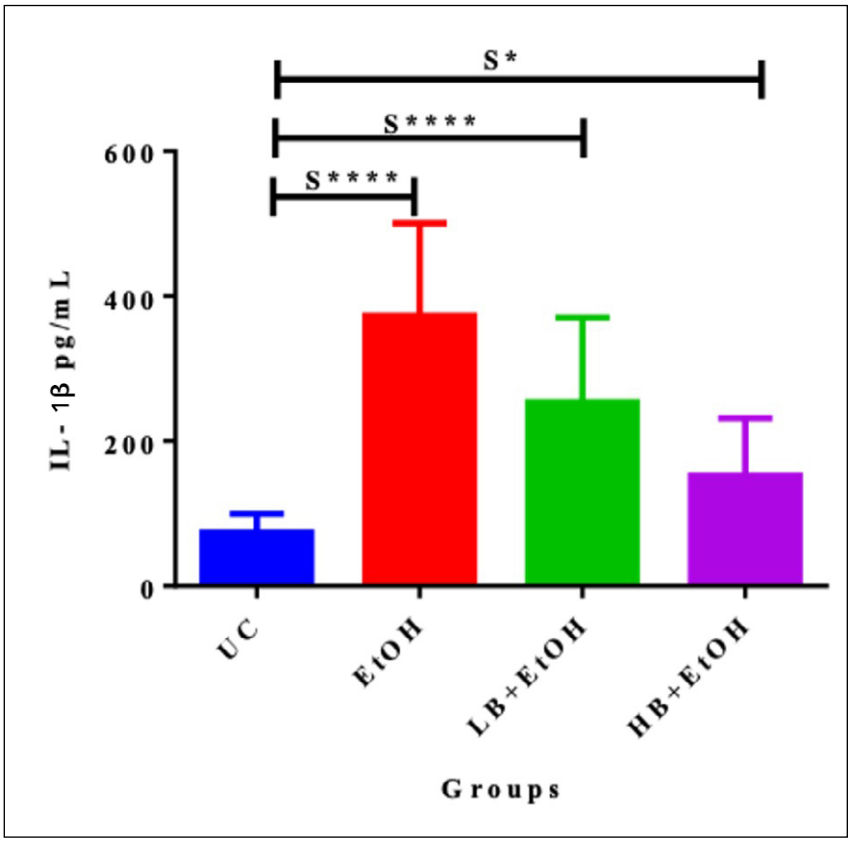

Fig. 3. Levels of IL-1**β** in the groups, *P < 0.001, ****P < 0.0001
Picture taken by the authors

[21]. The results of this current study show that ethanol exposure had a substantial elevation of IL-6 levels, while high-dose basil supplementation (HB+EtOH) returned the levels of IL-6 to nearly normal levels, as demonstrated by the lack of statistical difference between this group and the control. The ethanol-only (EtOH) group exhibited a dramatic increase in IL-6 levels (410.3 ± 167.7 pg/mL, P < 0.0001) compared to the control group (70.53 ± 23.39 pg/mL) as illustrated in Table 2 and Fig. 2.

This sharp elevated confirms the pro-inflammatory effect of chronic ethanol consumption, which stimulates hepatic immune cells (Kupffer cells) and causes oxidative stress, leading to IL-6 overexpression [22]. Elevated IL-6 has been correlated with hepatocellular damage, fibrosis, and the progression of alcohol-related liver disease. The low-dose basil (LB+EtOH) group showed a moderate decrease in IL-6 levels (366.8 ± 157.1 pg/mL, P < 0.0001) compared to the ethanol-only group. However, with this reduction, IL-6 remained significantly higher than that of the control group, showing that this dose provided only a partial protection against ethanol-induced inflammation. The HB+EtOH group exhibited a substantial reduction in IL-6 (133.9 ± 86.76 pg/mL), and most importantly, there was no statistical difference between this group and the control (P > 0.05).

This result suggests that high-dose basil supplementation fully counteracted the ethanol-induced IL-6 elevation, effectively restoring IL-6 levels to normal. The absence of a statistical difference between the HB+EtOH and control groups suggests that high-dose basil completely neutralized the IL-6 increase caused by ethanol. This is a highly favorable outcome, as it implies that basil at this dose was effective enough to prevent inflammatory changes rather than just reducing them [23].

Unlike the LB+EtOH group, where IL-6 remained significantly elevated, the HB+EtOH group's normalization of IL-6 suggests a strong anti-inflammatory response, possibly due to Inhibition of NF-κB activation, reducing IL-6 gene expression, antioxidant properties of basil polyphenols, scavenging reactive oxygen species (ROS) that drive inflammation, and Modulation of gut microbiota, reducing endotoxin-mediated cytokine release [24]. Since IL-6 is a key driver of liver inflammation and fibrosis, the ability of high-dose basil to completely prevent IL-6 elevation suggests a strong therapeutic potential for managing alcohol-induced liver injury [25]. These results indicate that basil supplementation at an optimized dose may help prevent progression to more severe liver conditions such as fibrosis or cirrhosis.

Interleukin-1 beta (IL-1β) is a key pro-inflammatory cytokine involved in liver inflammation and immune responses [26]. The results of this study confirm that ethanol exposure significantly elevated IL-1β levels, while basil supplementation, particularly at a high dose, effectively reduced IL-1β, though it remained

slightly elevated compared to the control group. The ethanol-only (EtOH) group exhibited a substantial increase in IL-1β levels (373.1 ± 127.7 pg/mL, $P < 0.0001$) compared to the control group (73.67 ± 26.21 pg/mL) as illustrated in Table 2 and Fig. 3.

This sharp rise highlights the role of ethanol in triggering liver inflammation through oxidative stress, gut-derived endotoxins, and activation of immune cells (Kupffer cells). IL-1β is a critical mediator in the pathogenesis of alcoholic liver disease (ALD), promoting hepatocyte apoptosis, fibrosis, and further inflammatory cytokine release. The low-dose basil (LB+EtOH, 50 mg/kg) group exhibited a marked reduction in IL-1β (254.0 ± 116.7 pg/mL, $P < 0.0001$) compared to the ethanol-only group. However, IL-1β levels remained significantly higher than the control group, indicating partial protection against ethanol-induced inflammation. he high-dose basil (HB+EtOH, 100 mg/kg) group further reduced IL-1β (152.3 ± 79.37 pg/mL, $P < 0.05$), bringing levels much closer to the control. While there was still a statistically significant difference from the control group ($P < 0.05$), the reduction was substantial, indicating strong hepatoprotective effects. Unlike TNF-α and IL-6, which were fully restored to normal with high-dose basil, IL-1β remained slightly elevated, though significantly reduced compared to ethanol exposure alone. This suggests that basil effectively mitigates ethanol-induced IL-1β elevation but does not completely normalize it. Possible reasons for this include: Different regulatory mechanisms: IL-1β production is tightly controlled by inflammasome activation, which may not be fully inhibited by basil at the given dose [27]. Residual oxidative stress and immune activation, while basil reduces inflammation, some residual ethanol-induced damage may still contribute to IL-1β production [28] [29]. Delayed response, IL-1β is one of the earliest cytokines activated in response to liver injury [30]. A longer duration of basil treatment may be needed for full normalization. Since IL-1β is a major contributor to liver fibrosis and hepatocyte apoptosis [31], the ability of high-dose basil to significantly lower IL-1β suggests potential therapeutic benefits in preventing the progression of alcohol-induced liver disease (ALD), steatohepatitis, and fibrosis. While IL-1β was not fully normalized, its substantial reduction implies that basil supplementation could still mitigate long-term liver damage and inflammatory progression.

CONCLUSIONS

The conclusion of this study demonstrate that *Ocimum basilicum* extract, particularly at high doses, effectively mitigates ethanol-induced liver inflammation by reducing pro-inflammatory cytokine levels. The high-dose basil group showed TNF-α and IL-6 levels statistically similar to the control group, suggesting a strong hepato-protective effect. Although IL-1β levels were not fully normalized, they were significantly reduced compared to the ethanol-only group. These findings highlight the potential of basil as a natural therapeutic agent for preventing liver damage and inflammatory diseases associated with chronic ethanol exposure.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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