

Antioxidant and Hepatoprotective Potentials of Lemon Juice and Sorghum *Ogi* (*lemon-ogi*) Mixture against Paracetamol-Induced Liver Damage in Rats

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Abstract

This study is aimed to determine the hepatoprotective activity and antioxidant properties of aqueous extract of Sorghum porridge (*Ogi*), a Sorghum fermentation product, mixed with lemon to form *Lemon-Ogi* mixture at various concentrations after inducing liver injury in rats. Rats were divided into 9 groups (n=4) the positive control group, negative control group, normal control and the treated groups (which include extracts at varying concentrations between 0-50% v/v). Hepatotoxicity was induced in rats by oral administration of paracetamol (1 g/kg body weight) suspended in normal saline on the first day of the experiment. After 7 days of post-treatment with the test mixture (*Lemon-Ogi*), biochemical markers such as L-aspartate aminotransferase (AST), L-alanine aminotransferase (ALT), Gamma Glutamyl transferase (GGT) were estimated. This was followed by the measurement of liver cytosolic antioxidant enzymes such as superoxide dismutase, catalase, and peroxidase. The data were analyzed using one-way analysis of variance (ANOVA) followed by Duncan *post hoc* test. The test mixture at concentrations of 30-50%v/v significantly ($P \leq 0.05$) reduced the elevated levels of AST, ALT, GGT when compared with the negative control animals. The *Lemon-Ogi* mixture also showed a significant ($P \leq 0.05$) increase in the reduced levels of superoxide dismutase (SOD), catalase, and peroxidase when compared with the negative control. The effects of the *Lemon-Ogi* mixture on these parameters were comparable with those of the standard drug, Vitamin E at 50% (v/v) concentration. The findings of this study indicate that *Lemon-Ogi* mixture showed a potential hepatoprotective activity and the protective action might have manifested by restoring the hepatic SOD, catalase, and peroxidase levels. The results of this study validate the traditional use of Sorghum porridge steep water as a strong antioxidant.

Keywords: Paracetamol-Hepatotoxicity, Biochemical Marker Enzymes, Antioxidant Enzymes, Lemon, *Ogi*

1. Introduction

A number of cereals and legumes that are readily available in Nigeria have been found to have nutrient potentials that could complement one another if properly processed and blended (Aremu *et al.*, 2011). It is therefore imperative that efforts are put in place to formulate composite blends and scientific studies are carried out to ascertain the nutritive adequacy of these locally available blends (cereal and legumes) for possible use as complementary foods, especially by the rural and poor urban mothers during weaning period.

Fermentation is an old method whereby foods are processed and also food spoilage is prolonged. Fermentation is a low-cost and the most economical technique of production and preservation of foods (Egounlety, 2002). Fermented foods and beverages are defined as those products that have been subjected to the effect of microorganisms or enzymes to cause desirable biochemical changes (Adebayo, 2007).

Fermented cereal porridge called '*Ogi*' is a popular fermented product in Western part of Africa. Apart from being a staple food it is used for weaning toddlers between the ages of 1-2 yrs (Aderiye & David, 2013; Milete *et al.*, 2008; Ogunbanwo *et al.*, 2004). Fermentation of *ogi* is usually done by lactic acid bacteria most of which have been reported to possess probiotic properties which when administered in adequate amounts confer a health benefit on the host (Ogunbanwo *et al.*, 2004; Kaur *et al.*, 2002; Mensah *et al.*, 1990; Adebayo & Aderiye, 2008). Pathogenic bacteria have been reported to be inhibited by probiotic organisms. The ability of *ogi* to inhibit the growth of bacterial etiological agent of diarrhea *in vitro* has also been reported (Adebayo & Aderiye, 2008).

Lemon (*C. limon*) is well known for its medicinal power and is used in many different ways (Abeyasinghe *et al.*,

2007). The health benefits of lemon are due to its many nourishing elements like vitamin C, vitamin B, phosphorous, proteins and carbohydrates. Lemon is a fruit that contains flavonoids, which are composites that contain antioxidant and cancer fighting properties (Abeyasinghe *et al.*, 2007). Lemon has a strong, purifying, citrus scent that is revitalizing and uplifting. Lemon consists of 68 percent dilemonene, a powerful antioxidant (Polydera & Taoukis, 2005). It is delightfully refreshing in water and may be beneficial for the skin. Lemon may be used to enhance the flavour of foods.

Lemon juice is characterized by the presence of significant amounts of the flavanones, hesperidin 20.5 mg/100 ml (Polydera & Taoukis, 2005) and 16.7 mg/100 ml of eriocitrin (Penniston *et al.*, 2008). Lemon juice is also quite rich in flavones: diosmin has been recognized as one of the main flavonoid components of this juice. Recently, however, more data published on di-C-glucosyl flavones showed them to be present in significant amounts. *C. limon* juice has been reported to be rich in diosmetin 6, 8-di-C-glucoside 4.95 mg/100 mL (Bonaccorsi *et al.*, 2005). and also contains apigenin di-C-glucoside 1.17 mg/100 mL (Leuzzi *et al.*, 2000; Rauf *et al.*, 2014).

Studies have not been carried out to check whether a mixture of *Lemon juice and Ogi* steep water (*lemon-ogi* mixture) can have hepatoprotective effect against paracetamol-induced liver injury in rats. The purpose of this study therefore is to evaluate the hepatoprotective potentials and antioxidant activities of aqueous extract of Sorghum porridge (*Ogi*) fortified with lemon juice.

2. Materials and Methods

2.1 Materials

2.1.1 Collection of Plant Materials

Dirt-free sorted white sorghum grains were bought from a local market in Osogbo, Osun State, Nigeria. Lemon fruits were plucked from a lemon tree in Ede, Osun State, Nigeria.

2.1.2 Preparation of Sorghum Steep

The White sorghum grains were sorted to remove grit, dirt and decomposing ones and later washed. Two hundred gram of the grains was weighed and steeped in plastic container containing 300ml clean water. The container was covered and the grains fermented for 72h. After steeping, the water was decanted and the grains were wet-milled. The resulting pastes were sieved using sterile muslin cloth (Adebayo *et al.*, 2007). The filtrates were collected into different sterile containers and allowed to settle for 3 days during which fermentation took place by the natural flora of the grains. Varying concentrations of the lemon juice with *ogi* steep water were prepared to give *Lemon-Ogi* mixture of between 0% to 50% (v/v) concentrations.

2.1.3 Experimental Animals

Healthy wistar albino rat of either sex weighing between 70 – 110 g were housed under uniform husbandry conditions and given pelleted diet (Ace Feed Ltd) and water *ad libitum*. Animals were maintained under standard environmental condition (28-30°C, 60-70 % relative humidity, 12-h dark / light cycle) in plastic cages with free access to standard laboratory animal diet (Vital finisher) and drinking water. They were left to acclimatize to laboratory conditions for 14 days prior to commencement of the experiment.

2.1.4 Reagents and Chemicals

Kits for ALT, AST and GGT were procured from Linear Chemicals S.L. (Barcelona, Spain).

All other reagents of BDH and E. Merck analytical grade were obtained from Sigma Chemicals Company (USA) and Merck (Germany).

2.2 Methods

2.2.1 Paracetamol Dose Regimen

Paracetamol tablets were obtained from Evans Medical PLC., Nigeria. Each tablet contains 500 mg of paracetamol. The dose administered to the mice to induce hepatotoxicity was set at 1g/Kg body weight (Oyedepo, 2014). The paracetamol was turned into a fine powder using a mortar and pestle to increase the dissolution. The powdered paracetamol was suspended in normal saline and was administered orally according to the body weight of the rats.

2.2.2 Hepatoprotective Activity

The hepatoprotective activity was evaluated according to the method described by Ahmed *et al.*, (2001). Thirty-six rats were randomly selected and divided into nine groups of four animals each. They were grouped as follows:

- **Group I:** Group I served as normal controls and received only the vehicle (5 ml/kg body weight of normal saline) daily.
- **Group II:** served as the paracetamol-treated /negative controls and received the vehicle (5 ml/kg/ day) after an initial single dose of paracetamol (1.0 g/kg) on the first day of the experiment.
- **Group III:** served as the Positive control group and were treated for 7 days with the standard drug (Vitamin E) at a dose of 1g/ kg/ day following the initial administration of 1.0 g/kg paracetamol.
- **Group IV:** received a daily treatment of 2ml (0%v/v) lemon-ogi mixture for 7days. The treatment started three hours after the initial administration of a single dose of 1.0g/kg paracetamol.
- **Group V:** received a daily treatment of 2ml (10%v/v) lemon-ogi mixture for 7days. The treatment started three hours after the initial administration of a single dose of 1.0g/kg paracetamol.
- **Group VI:** received a daily treatment of 2ml (20%v/v) lemon-ogi mixture for 7days. The treatment started three hours after the initial administration of a single dose of 1.0g/kg paracetamol.
- **Group VII:** received a daily treatment of 2ml (30%v/v) lemon-ogi mixture for 7days. The treatment started three hours after the initial administration of a single dose of 1.0g/kg paracetamol.
- **Group VIII:** received a daily treatment of 2ml (20%v/v) lemon-ogi mixture for 7days. The treatment started three hours after the initial administration of a single dose of 1.0g/kg paracetamol.
- **Group IX:** received a daily treatment of 2ml (20%v/v) lemon-ogi mixture for 7days. The treatment started three hours after the initial administration of a single dose of 1.0g/kg paracetamol.

After the treatments were done for seven days all animals were anesthetized and sacrificed for biochemical studies on the eighth day of the experiment (Oyedepo and Odoje, 2014).

All the rats were sacrificed by cervical decapitation; blood samples were collected through retro-orbital plexus and allowed to clot for 30 min at room temperature. The clear serum was separated by centrifugation at 4000 rpm for 10 min and serum samples were stored at -40°C until use for the determination of biochemical parameters. Hepatic tissues were carefully excised, cleaned, and homogenized in cold 1.15% KCl and 10 mM phosphate buffer with EDTA (pH 7.4) and centrifuged at 10,000 rpm for 10 min. The supernatant was further centrifuged at 13,000 rpm for 60 min to obtain a cytosolic extract for the estimation of liver cytosolic superoxide dismutase (SOD), catalase, and peroxidase (Oyedepo and Odoje, 2014).

2.2.3 Biochemical Parameters

The functional state of the liver was determined by estimating the biochemical parameters such as Serum Aspartate aminotransferase (AST), alanine aminotransferase (ALT) and Gamma Glutamyl aminotransferase (GGT). AST and ALT were determined by the method described by Reitman and Frankel (1957) using Randox diagnostic kits. GGT activity was determined following the principle described by Szasz (1969).

2.2.4 Antioxidant Enzymes Assay in Liver

2.2.4.1 Superoxide Dismutase Activity

Superoxide dismutase activity SOD was assayed as described by Beauchamp and Fridovich (1971) and Chidambara *et al* (2002) based on the reduction of nitroblue tetrazolium (NBT) to water insoluble blue formazan. The assay mixture contained 0.5 mL of liver homogenate, 1 mL of 50 mM sodium carbonate, 0.4 mL of 24 μ m

NBT, and 0.2 mL of 0.1 mM EDTA. The reaction was initiated by adding 0.4 mL of 1 mM hydroxylamine hydrochloride. The developed blue color in the reaction was measured at 560 nm. Zero time absorbance was taken at 560 nm followed by recording the absorbance reading every 30 s for a period of 5 min at 25°C. The above-mentioned reaction mixtures without the liver homogenate served as control. The rate of increase in absorbance units (A) per minute for the control and for the test sample(s) was determined and the percentage inhibition for the test sample(s) was calculated by the following formula:

$$\% \text{ Inhibition} = \frac{[(\Delta A_{560\text{nm}/\text{min}})_{\text{control}} - (\Delta A_{560\text{nm}/\text{min}})_{\text{test}}]}{(\Delta A_{560\text{nm}/\text{min}})_{\text{control}}} \times 100$$

where $(A_{560 \text{ nm at } 5 \text{ min and } 30\text{s}} - A_{560 \text{ nm at } 30\text{s}}) / 5 \text{ min} = \Delta A_{560 \text{ nm/minute}}$.

Units of the SOD activity were expressed as the amount of enzyme required to inhibit the reduction of NBT by 50% and the activity was expressed as units per mg of protein (Habbu *et al.*, 2008).

2.2.4.2 Catalase Activity

The activity of catalase was determined by an adaptation of the method of Aebi (1984). 1 mL of the liver homogenate was taken in a test tube and 1.9 mL of phosphate buffer (50mM, pH 7.4) was added to it. The reaction was initiated by the addition of 1 mL of 30 mM H₂O₂. A mixture of 2.9 mL of phosphate buffer and 1 mL of H₂O₂ without the liver homogenate served as the blank. The decrease in absorbance due to the decomposition of H₂O₂ was recorded at 240 nm against the blank. Units of catalase were expressed as the amount of enzyme that decomposes 1 μM of H₂O₂ per min at 25°C and the activity was expressed in terms of units per milligram of proteins (Habbu *et al.*, 2008).

2.2.4.3 Peroxidase Activity

Peroxidase was assayed by the method of Nicholas (Nicholas, 1962). One milliliter of the 10 mM KI solution and 1 mL of 40 mM sodium acetate were added to 0.5 mL of the liver homogenate. Then the absorbance of potassium per iodide was read at 353 nm, which indicated the amount of peroxidase. Then 20 μL of 15 mM H₂O₂ was added to the reaction mixture followed by recording the change in the absorbance for a period of 5 min. Units of the peroxidase activity were expressed as the amount of enzyme required to change the optical density by 1 unit per min. The specific activity was expressed in terms of units per milligram of Proteins (Habbu *et al.*, 2008).

2.3 Statistical Analysis

The values were expressed as mean ± SEM. Statistical analysis was performed by one way analysis of variance (ANOVA) followed by Duncan post hoc test. Values were considered statistically significant at P < 0.05.

3. Results

3.1 Weight Changes in the Experimental Animals

The effects of 7 days post-treatment with *Lemon-Ogi* mixture and vitamin E on body weight of rats is presented in table 1. Rats in eight of the groups gained weight while the rats in group 2 (negative control) lost weight. This could be as a result of loss of appetite exhibited by the rats in the negative control group about 24 hours after the paracetamol treatment and throughout the duration of the experiment.

Table 1: Weight Changes in Rats Treated with Lemon/Ogi Steep Mixture after 21 Days of Treatment with Lemon-Ogi Mixture and Vitamin E

Group	Treatment	Weight (g)	
		Initial Weight	Final Weight
Group I <i>Normal Control</i>	Normal saline	106.0 ± 1.25	123.0 ± 1.25
Group II <i>Negative Control</i>	PCM + 1ml/kg b.w. normal saline	105.7 ± 1.19	85.0 ± 0.94
Group III <i>Positive Control</i>	PCM + 1.0g/kg b.w. vitamin E	105.0 ± 0.47	117.3 ± 1.19
Group IV	PCM + 0% (v/v) <i>lemon-ogi</i> mixture	85.0 ± 0.92	118.3 ± 1.96
Group V	PCM + 10% (v/v) <i>lemon-ogi</i> mixture	65.0 ± 0.94	94.3 ± 2.42
Group VI	PCM + 20% (v/v) <i>lemon-ogi</i> mixture	64.7 ± 1.44	98.0 ± 1.70
Group VII	PCM + 30% (v/v) <i>lemon-ogi</i> mixture	68.7 ± 0.72	97.3 ± 0.54
Group VIII	PCM + 40% (v/v) <i>lemon-ogi</i> mixture	66.0 ± 0.47	106.3 ± 0.72
Group IX	PCM + 50% (v/v) <i>lemon-ogi</i> mixture	86.7 ± 0.72	146.7 ± 0.72

Values are expressed as mean ± SEM; n=4

Table 2: Effect of Varied Levels of Lemon - Ogi Mixture and Vitamin E on Serum Enzymes in Paracetamol-Induced Hepatotoxicity in Rats

Group	Treatment	Level of Liver Marker Enzymes (IU/L)		
		AST	ALT	GGT
Group I Normal control	Normal saline	12.28 ± 0.16	12.70 ± 0.057	10.67 ± 0.14
Group II Negative Control	PCM + 1ml/kg b.w. normal saline	36.10 ± 0.06*	51.30 ± 0.46*	48.70 ± 0.50*
Group III Positive Control	PCM + 1.0g/kg b.w. vitamin E	16.60 ± 0.23** (83.96)	18.2 ± 0.69** (85.75)	14.64 ± 0.26** (89.56)
Group IV	PCM + 0% (v/v) lemon-ogi mixture	32.05 ± 0.32 (17.00)	41.50 ± 0.24 (25.39)	42.82 ± 0.37 (15.46)
Group V	PCM + 10% (v/v) lemon-ogi mixture	19.15 ± 0.53** (71.16)	29.18 ± 0.01** (57.31)	30.43 ± 0.45 (48.04)
Group VI	PCM + 20% (v/v) lemon-ogi mixture	16.89 ± 0.31** (80.61)	29.06 ± 0.37** (57.62)	27.11 ± 0.01** (56.77)
Group VII	PCM + 30% (v/v) lemon-ogi mixture	14.82 ± 0.08** (89.34)	24.85 ± 0.23** (68.52)	24.25 ± 0.34** (64.29)
Group VIII	PCM + 40% (v/v) lemon-ogi mixture	14.43 ± 0.12** (90.97)	22.82 ± 0.14** (73.78)	21.13 ± 0.17** (72.50)
Group IX	PCM + 50% (v/v) lemon-ogi mixture	14.20 ± 0.16** (91.94)	20.58 ± 0.19** (79.59)	19.45 ± 0.26** (76.91)

All values are expressed as means ± SEM (n = 4). Figures in parentheses are the percentage protections in individual biochemical parameters from their elevated values caused by paracetamol toxicity. The percent protection was calculated as follows:

$$100 \times \frac{\text{Value of negative control} - \text{value of normal sample}}{\text{Value of negative control} - \text{Value of normal control}}$$

*Significant increase compared to the normal control group ($P \leq 0.05$). **Significant reduction compared to the negative controls ($P \leq 0.05$).

3.2 Serum Enzymes Activities in Experimental Animals

The results of the investigation of the hepatoprotective activity of lemon-ogi mixture are as shown in Table 2. The table shows that in the negative control group, significant acute hepatocellular damage was manifested by

the elevated levels ALT, AST, and GGT when compared with those of the normal control rats. But the oral administration of *lemon-ogi* mixture at the concentrations of 10-50% (v/v) for 7 days significantly ($P \leq 0.05$) reduced the elevated levels of AST, ALT and GGT when compared with negative control rats, and these biochemical parameters were comparable with Vitamin E (Fig. 1, Table 2). The lemon-ogi mixture at 30 -50% (v/v) have a higher percentage protection in AST activity than the standard drug (Vitamin E). However, Vitamin E has higher percentage protection in ALT and GGT activities than the mixture at every concentration.

3.3 Activities of the Antioxidant Enzymes in the Liver of Experimental Animals

Table 3 shows that, as compared to the control, the negative control animals exhibited significantly ($P \leq 0.05$) lower levels of SOD, catalase, and peroxidase activity in liver cytosol.

On the contrary, the groups which received the *lemon-ogi* mixture both at the concentrations of 10 - 50% v/v for 7 days showed a significant ($P \leq 0.05$) dose-dependent increase in the reduced levels of SOD, catalase, and peroxidase. The test mixture (with the exception of the 0% concentration) has a moderate activity in terms of the restoration of reduced enzyme levels as compared to the standard drug, Vitamin E, which also significantly ($P < 0.01$) restored the altered levels of SOD, catalase, and peroxidase.

4. Discussions

Paracetamol (Acetaminophen) is a widely used analgesic and antipyretic agent. Hepatotoxic doses of acetaminophen deplete the normal levels of hepatic glutathione, when NAPQI covalently binds to cysteine groups on proteins to form 3-(cysteine-S-yl) acetaminophen adducts ((Pablo *et al.*, 1992; Timenstein & Nelson, 1989). The glutathione protects hepatocytes by combining with the reactive metabolites of paracetamol thus preventing their covalent binding to liver proteins (Vermeuler *et al.*, 1992).

In the present investigation, it was observed that the animals treated with acetaminophen presented significant liver damage (Fig.1, Table 2). The post-treatment with varying concentrations of *Lemon-Ogi* mixture significantly attenuated the elevated levels of the serum marker enzymes. However, it was observed that as the lemon concentration of the formulation increases, there is a gradual and significant decrease in the activities of serum marker enzymes. The normalization of the serum marker enzymes by varying the concentrations of lemon juice with *Ogi* steep water suggests that they are able to condition the hepatocytes so as to protect the membrane integrity against acetaminophen induced leakage of marker enzymes into the circulation. The above changes can be considered as an expression of the functional improvement of hepatocytes, which may be caused by an accelerated regeneration of parenchyma cells (Oyedepo, 2011). Lipid peroxidation has been postulated to be responsible for the destructive process of liver injury due to acetaminophen administration (Pablo *et al.*, 1992).

Furthermore, the *lemon-ogi* mixture showed a potential *in vivo* antioxidant activity as it (with increasing lemon concentration in the mixture) elevated the reduced levels of liver cytosolic SOD, catalase, and peroxidase activity. These antioxidant enzymes are involved in the reduction of reactive oxygen species (ROS) and peroxides produced in the living organism thus play a vital role in the maintenance of a balanced redox status. The restoration of the SOD activity toward a normal value indicates that the *Lemon-Ogi* mixture can help in cellular defense mechanisms by preventing cell membrane oxidation (Lee *et al.*, 2003).

Table 3: Effect of *Lemon-Ogi* Mixture on the Levels of Antioxidant Enzymes in Paracetamol-Induced Hepatotoxicity in Rats

Group	Treatment	SOD (units/mg of protein)	Catalase (units/ mg of protein)	Peroxidase (units/ mg of protein)
Group I <i>Normal Control</i>	Normal saline	18.70 ± 0.563	120.48 ± 1.76	0.78 ± 0.08
Group II <i>Negative Control</i>	PCM + 1ml/kg b.w. normal saline	4.76 ± 0.876**	35.84 ± 3.58**	0.21 ± 0.07**
Group III <i>Positive Control</i>	PCM + 1.0g/kg b.w. vitamin E	16.89 ± 0.543* (87.02)	144.25 ± 5.60* (128.08)	0.80 ± 0.05* (103.51)
Group IV	PCM + 0% (v/v) <i>lemon-ogi</i> mixture	7.69 ± 0.538 (21.02)	56.70 ± 8.79 (24.65)	0.30 ± 0.081 (15.79)
Group V	PCM + 10% (v/v) <i>lemon-ogi</i> mixture	9.97 ± 0.348 (37.37)	90.77 ± 8.90* (64.90)	0.35 ± 0.034 (24.56)
Group VI	PCM + 20% (v/v) <i>lemon-ogi</i> mixture	10.67 ± 4.58* (42.40)	109.60 ± 5.77* (87.15)	0.41 ± 0.017* (35.09)
Group VII	PCM + 30% (v/v) <i>lemon-ogi</i> mixture	12.05 ± 0.530* (52.30)	118.75 ± 6.67* (97.96)	0.49 ± 0.012* (49.12)
Group VIII	PCM + 40% (v/v) <i>lemon-ogi</i> mixture	13.85 ± 0.522* (65.21)	120.78 ± 8.90* (100.35)	0.58 ± 0.04* (0.65)
Group IX	PCM + 50% (v/v) <i>lemon-ogi</i> mixture	14.49 ± 0.271* (69.80)	134.57 ± 9.10* (116.65)	0.69 ± 0.011* (84.21)

Values are mean ± SEM (n=4), using one way ANOVA followed by Duncan post-hoc test. Figures in parentheses are the percentage restorations of antioxidant activity from their reduced value caused by paracetamol toxicity. The percent restoration was calculated as follows:

$$100 \times \frac{\text{Value of sample} - \text{Value of negative control}}{\text{Value of normal control} - \text{Value of negative control}}$$

. *P ≤ 0.05 when compared with the negative control group. ** P ≤ 0.05 when compared with the normal control group.

An increase in the catalase activity with respect to paracetamol treatment indicates that the mixture can play an important role in scavenging hydrogen peroxide. Similarly, an increase in the peroxidase activity indicates that the extract also helps in the restoration of vital molecules such as NAD, cytochrome, and glutathione (Lee *et al.*, 2003). This study corroborates the recent findings of Awika and Rooney (2004), who reported that various sorghum products possess high antioxidant activities.

However, it was observed that the group treated with the highest concentration of lemon juice (i.e. 50%) has the most significant in effect on the liver enzymes (AST, ALT, GGT) as well as on the antioxidant enzymes when compared to the negative control group (Table 2). This must be as a result of the increased concentration of lemon juice in the formulation. According to Polydera and Taoukis (2005), Lemon juice is good source of Vitamin C and Vitamin E, which are attributed for the endogenous antioxidant protection system of the body.

5. Conclusion

The results of the present study indicate that the lemon-ogi mixture exhibited a potential hepatoprotective activity against paracetamol induced hepatotoxicity. The hepatoprotection of the *lemon-ogi* mixture against paracetamol-induced hepatotoxicity in rats could be attributed to its ability to restore the antioxidant enzymes SOD, catalase, and peroxidases of liver cytosol or to the free radical scavenging activity of the mixture

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