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Abstract

A coconut is mainly rich in water, sugar, lipids, protein, amino acids, vitamins, minerals, and phytochemicals. These substances have shown positive benefits on both human and animal health, resulting in the reduction of the risk for several illnesses, including gastric ulcers. The main objective of this research is to determine how effectively the anti-ulcer medication omeprazole and the powdered coconut fruit and milk reduce stomach ulcers in rats. Out of the 65 rats utilized in this investigation, nine groups of six female albino rats, each weighing 150g±10g, were created. To create stomach ulcers, rats were given 300 mg/kg B.W. to induce ulcers. Gastritis, pH, acidity, length of stomach, ulcer score, ulcer index, percentage of ulceration, and preventive index were also measured in the study. Biochemical analyses included measurements of triglycerides (TG), high-density lipoprotein (HDL-c), low-density lipoprotein (LDL-c), very low-density lipoprotein (VLDL-c), liver enzymes (ALT, AST, & ALP), and oxidative enzymes (CAT, SOD, NAD, and GPx). Moreover, the HPLC method for phenolic compound identification. The results showed that the gastric ulcer group of rats exhibited considerably improved lipid fraction, liver activities, oxidative enzymes, and gastric ulcer when administered coconut fruit powder and its milk. Concluding the study, the gastric ulcer rats that were given 500 mg/kg of coconut milk showed better % of ulcer inhibition and improved biochemical analysis. This suggests that coconut fruit and milk can be used as natural dietary supplements to improve diets and reduce risk factors for chronic diseases like gastric ulcers.

Key words: Coconut, female albino rats, omeprazole, ulcer, oxidative enzymes , lipid fraction .

Introduction

Gastric ulcer is a common clinical manifestation worldwide with about 10 % prevalence in humans. It is characterized by benign lesions of gastric or duodenal mucosa, untreated lesions may cause gastric perforations, severe bleeding and even could cause death **Beiranvand and Bahramikia, (2020)**. It is a multifactorial disease with diverse etiological factors; it occurs when there is an imbalance between the aggressive endogenous factors (pepsin, HCl) and the defensive factors (prostaglandins, mucus and bicarbonate barrier and adequate blood flow). Various factors could promote this imbalance, including a stressful lifestyle, alcohol consumption, *Helicobacter pylori* infection, smoking, nutritional deficiencies, and using steroidal and non-steroidal anti-inflammatory drugs (NSAIDs) **Ahmed *et al.*, (2020) and Longo *et al.*, (2021)**. Drugs disrupt the gastric mucosal barrier without inhibiting acid secretion, cause reduction of prostaglandin synthesis, decrease gastric mucosal blood flow, increase inflammatory markers, and inactivate various growth factors that aid in the repair of gastric mucosa **Goswami *et al.*, (2017)**. Different pharmacological categories are present to treat gastric ulcer, whether by relieving pain, healing, and/or preventing the recurrence of ulcer **Beiranvand and Dezfoulian, (2021)**. Unfortunately, there is no complete recovery for peptic ulcer using these pharmacological approaches, besides arising of possible side effects **Meng *et al.*, (2019)**. The key defense factors of the gastricmucosa include the secretion of bicarbonate and prostaglandin, increased levels of antioxidants and maintaining adequate levels of Nitric Oxide (NO) dilates blood vessels, increases blood flow and stimulates gastric angiogenesis in the healing process of ulcers **Hoogerwerf and Pasricha, (2001)**. NO also stimulates cell proliferation of the gastric mucosa and granulation tissue formation at the base of an ulcer **Yang *et.al.*, (2000)**. **Rydning *et al.*, (1982)** who confirmed that alcohol, aspirin and NSAIDs can aggravate healing of peptic ulcer.

Coconut (*Cocos nucifera*) belonging to the Arecaceae family holds quite an importance in the Indian traditional medicinal system. *C. nucifera* inflorescence has been reported in literature to be useful in treatment of diarrhea, dysentery, diabetes, and dyspepsia **Rengjith *et al.*, (2013)**. Coconut meat is the white flesh found inside the hard, brown outer shell of the coconut. it is rich in several important minerals, especially manganese and copper. While manganese supports enzyme function and fat metabolism, copper assists bone formation and heart health. **Li and Yang, (2018)**. Coconut milk (CM) is the liquid that is extracted from the grated coconut white meat which is rich in saturated

fats and it is widely consumed in parts of Asia and South America. They are widely cultivated in the tropical climates as mentioned above and are exported as canned products to North America and Europe **Tinchan *et al.*, (2015)**. Coconut milk is made from coconut (*Cocos nucifera*) which belongs to the *Araceceae* family. Coconut milk is a liquid obtained through manual or mechanical force of coconut meat **Narataruksa *et al.*, (2010)**. Coconut milk contains essentially high amounts of protein, amino acids, water, sugar, fats, vitamins, and minerals **Effiong, (2003)**. The milk contains significant amounts of fat, but unlike other nuts, it provides fat that is mainly in the form of medium chain saturated fatty acids (MCFAs) that is abundant in mother's milk, lauric acid; this is converted in the body into a highly beneficial compound called monolaurin, an antiviral and antibacterial that destroys a wide variety of disease-causing organisms. According to the National Center for Biotechnology Information, lauric acid has many germs fighting, antifungal and antiviral properties that are very effective at ridding the body of viruses, bacteria, and countless illnesses **Baldioli *et al.*, (1996)**. It has been reported that CM has a greater protection in indomethacin induced ulceration when compared with coconut water **Nneli and Woyike, (2008)**. Decrease in ulcer index in the groups treated with coconut milk may be due to the presence of tannin, saponin and flavonoid present in the coconut milk as evident in the phytochemical screening of this study. Tannins, saponins and flavonoids have been reported to have anti-ulcer properties **Alkofahi and Atta, (1999)**. **Tolulope *et al.*, (2019)**, reported that coconut milk potentiates antiulcerogenic effects in Wistar rats with ethanol-induced gastric ulcer, with the highest dose (70 mg/kg) having the most therapeutic effect. Therefore, coconut milk represents a therapeutic alternative in the treatment or management of gastric ulcer.

Therefore, this work aimed to use different levels of coconut fruit powder and its milk for development of peptic ulcer formation in rats.

Material and Methods

Materials:

The fruits of the coconut were purchased at Cairo City's hypermarket in the Cairo Governorate, Egypt.

Experimental animals

A total of forty-eight mature male Sprague Dawley albino rats, weighing 140±10 g, were purchased from the Ministry of Health's Vaccine and Immunity Organization at Helwan Farm in Cairo, Egypt.

Chemicals and kits

Folin–Ciocalteu phenol reagent, and standard phenolic compounds were purchased from Sigma– Aldrich Inc. (St. Louis, MO). Chemical

kits from Technogene Chemical Co., Dokki, Cairo, Egypt were utilized to determine the levels of (TC, TG, HDL-c, ALT, AST, ALP, CAT, SOD, NAD, and GPx). A pharmaceutical company in Sheben El-Kom, Menoufia Governorate, Egypt, was the source of omeprazole, which is produced by Hikma Pharma.

Methods

Preparations of coconut fruits extract

The coconut fruit was finely crushed using an electric grinder (Toshiba El-Araby, Benha, Egypt) and stored in dark, stoppered glass bottles in a cool, dry place until needed. Grated coconut meat is pressed to produce a creamy white liquid that is then stored in dark, stoppered glass bottles in a cold, dry location until needed. This is known as coconut milk. To prevent their contents from oxidizing, **Elhassaneen *et al.*, (2020)** state that the coconut powder and coconut milk were stored in a cool, dry, and dark environment.

Identification and quantification of coconut husk and milk phenolic compounds by HPLC

With a few minor adjustments, the phenolic acid content of every extract was determined using an HPLC system in accordance with the technique outlined by **Kim *et al.*, (2016)**. An ODS column (5 μm, 4.6 mm × 250 mm, Agilent Technologies, Santa Clara, CA, USA) was used as the analytical column. Solvent A (water containing 0.1% (v/v) acetic acid) and solvent B (acetonitrile containing 0.1% (v/v) acetic acid) were used in a gradient elution. The following was the gradient program: The gradients are as follows: 0–2 min, 92–90% A in B; 2–27 min, 90–70% A in B; 27–50 min, 70–10% A in B; 50–51 min, 10–0% A in B; 51–60 min, 0% A in B (isocratic); and 60–70 min, 0 to 92% A in B. The injection volume was 20 μL, and the flow rate was kept constant at 1 mL per minute. At 280 nm, the UV detector was calibrated. Standard different phenolic compounds prepared in HPLC-grade methanol. By introducing various quantities of the phenolic acid standard into the HPLC apparatus, standard curves were created that allowed the amounts of phenolic acid to be calculated. Each peak area was computed in respect to a standard peak area, and the presence of the standard phenolic acids in the samples allowed for the verification of the peaks. The levels of each individual phenolic acid component were added up to determine the total phenolic acid content. produced in methanol of HPLC quality.

The induction of gastric ulcer

To produce gastric ulcers, rats were administered 300 mg/kg of aspirin diluted in 0.6 ml of 1% carboxymethylcellulose orally via gastric

tube once a day, as per the protocol outlined by **Clara *et al.*, 2012 and Abdelatif *et al.*, (2019)**.

Experimental design

The Animal House, Department of Nutrition and Food Science, Faculty of Home Economics, Menoufia University, Egypt, is where the study was conducted and approved. In the current study, 48 mature male Sprague Dawley Strain albino rats weighing 140±10g at 10 weeks of age were utilized. For a period of seven days, all rats received a basal diet that was prepared in accordance with **Reeves *et al.*, (1993)**. Rats are separated into nine groups following this adaptation period, with six rats in each group as follows: Group (I) rats fed on basal diet as negative control. Group (2) peptic ulcer rats and used as a positive control group. Group (3) A group peptic ulcer rats fed on coconut fruit powder by 2.5% of the weight of basal diet. Group (4) A group peptic ulcer rats fed on coconut fruit powder by 5% of the weight of basal diet. Group (5) A group peptic ulcer rats fed on coconut milk by 3.5ml of the weight of basal diet. Group (6) A group peptic ulcer rats fed on coconut milk by 7.0 ml of the weight of basal diet. Group (7) A group peptic ulcer rats fed on mixture coconut fruit and milk by 2.5% of the weight of basal diet. Group (8) A group peptic ulcer rats fed on mixture coconut fruit and milk by 5% of the weight of basal diet. Group (9) A group peptic ulcer rats administrated on Omeprazole drug by (10 mg/kg for 21 days) of the weight of basal diet. Rats were scarified after the animals had fasted for 12 hours at the finish of the 4-week experiment.

According to **Schermer (1967)**, blood samples were taken from the portal vein and placed into dry, clean centrifuge tubes for the purpose of separating the serum. The samples were centrifuged for ten minutes at 4000 rpm. Samples of serum were frozen at -18 °C for further chemical analysis.

Collecting gastric secretions and calculating the ulcer index

Under anesthesia by diethyl ether rats were sacrificed and their stomachs were ligated around both openings (cardiac and pyloric openings) and injected by 4 ml distilled water, the gastric juice was collected in sterilized tube, and centrifuged at 500rpm for 5 minutes to estimating gastric secretion parameters including volume in (ml), titratable acidity, MEq/l and total acid output MEq/h. Stomach examined for ulceration. Evaluation of degree of ulceration was expressed in terms of ulcer score which is calculated by dividing the total number of ulcers in each group by number of rats in that group according to **Robert *et al.*, (1968)**. The Ulcer index (U.I) was calculated by multiplying ulcer score x 100 according to **Khanavi *et al.*, (2012)**. According to **Bongu and Vijayakumar (2012),** the ulceration (%) is calculated by dividing the number of animals with ulcers by the total number of animals and

multiplied by 100. The preventative index was calculated using **Hano *et al.*, (1976)** approach.

Determination of the volume, pH, total acidity, and titratable acid output of gastric juice

Following the procedure outlined by **Anandan *et al.*, (2004)**, the volume of gastric juice was measured using a graduated cylinder. The pH value was measured using **Debnath *et al.*, (1974)** technique. Using **Anson (1938)** method, the total acidity of gastric juice was measured by titrating 1 ml of gastric juice in 10 ml of distilled water with 0.01 N NaOH and two drops of phenolphthalein as an indicator. The total amount of NaOH titratable acid production required to neutralize 100 milli grams of gastric juice according to **Deverport, (1972)**.

The estimation of the oxidative enzymes

Malondialdehyde (MDA), glutathione peroxidase (Gpx), catalase (CAT), and superoxide dismutase (SOD), was conducted in stomach tissue using the techniques outlined by **Misra and Fridovich (1972); Pagila and Valentaine, (1967); Takahara *et al.*, (1960), and Habig *et al.*, (1974)**, respectively.

Liver functions

The modified kinetic approach of **Hafkenscheid (1979)** was used to evaluate the activities of alanine amino transferase (ALT) in serum. The modified kinetic approach of **Henary, (1974) and Moss, (1982)** was used to estimate the activities of aspartate amino transferase (AST) in serum.

Lipid fraction

The following parameters were measured: total cholesterol (TC), triglycerides (TG), low density lipoprotein (LDL-c), very low lipoprotein (VLDL-c), and high-density lipoprotein (HDL-c) according to methods of **Allain (1974), Fossati and Principle, (1982), Lopez (1977) and Lee and Nieman (1996)**.

Statistical analysis:

SAS (1988) method was used to analyze the data using a completely randomized factorial design. When a significant main effect was found, the Student-Newman-Keuls Test was used to separate the means. Using the Costat Program, differences between treatments of ($P \leq 0.05$) were considered significant. One Way ANOVA was used to analyze the biological results.

RESULTS AND DISCUSSION

The phenolic compounds found in coconut fruit pulp and milk that were determined using an HPLC approach are shown in Table 1. The acquired results indicated that gallic acid, caffeic acid, and P-

hydroxybenzoic acid were the greatest phenolic components found in coconut fruit pulp. 3.85, 3.80, and 3.17 mg/100g were the corresponding readings. On the other hand, ellagic acid, synergic acid, and ferulic acid have the lowest levels of phenolic components in coconut fruit pulp. The amounts were, in order, 0.24, 1.18, and 1.45 mg/100g.

Conversely, the largest concentrations of phenolic compounds found in coconut milk powder were found in salicylic acid, chlorogenic acid, and catechin. 4.88, 1.14, and 1.10 mg/100 g were the corresponding values. In contrast, ellagic acid, synergic acid, and ferulic acid have the lowest levels of phenolic components in coconut milk. The amounts were, in order, 0.35, 0.30, and 0.14 mg/100g. Regarding vanillic acid and protocatechuic acid, they were not found under these conditions. These findings concur with those of **Hamrouni-Sellami *et al.*, (2013)**, who suggested that the increased concentration of phenolic compounds may be related to microwave-induced tissue disruption in plants, which leads to a greater release of phenolic compounds. Additionally, discovered that the dried husk had greater quantities of ferulic and 4-hydroxybenzoic acids than the fresh husk.

Furthermore, it was reported by **Valadez-Carmona *et al.*, (2016)** that eight phenols were found in both fresh and dehydrated coconut husks. When compared to the fresh coconut, the husk had higher levels of epicatechin, gallic, 4-hydroxybenzoic, ferulic, and syringic acids.

Table (1) Identified phenolics compounds in coconut fruit and its milk by HPLC technique

Phenolic compounds	Coconut fruit (mg/100g)	Coconut milk (mg/100g)
Gallic acid	3.80	ND
Caffeic acid	3.85	0.44
Ellagic acid	1.45	0.30
Vanillic acid	ND	ND
***P*-coumaric**	2.74	0.37
Chlorogenic acid	2.11	1.10
Synergic acid	1.18	0.35
Catechin	2.03	4.88
***P*-hydroxybenzoic acid**	3.17	0.75
Ferulic acid	0.24	0.14
Salicylic acid	2.69	1.14
Protocatechuic acid	ND	ND

ND= Not detectable

Influence of coconut fruit and its milk on gastric juice, pH, acidity, and stomach length of peptic ulcer rats

The stomach length of rats with peptic ulcers is displayed in Table (2). It is evident from the data that the positive control group's gastric juice was considerably higher than the negative control groups. The average values were 4.37 and1.52 ml, respectively. Data on the peptic ulcer treatment groups showed that the group fed 2.5% coconut fruit powder had the greatest values, whereas the group fed a basal diet with omeprazole medication had the lowest values, with a significant difference. The average readings were 2.11 and 1.59 ml, respectively.

In terms of pH, the results showed that the positive control group's pH was significantly lower than the control negative groups. The averages were 1.90 and 8.60, respectively. Data concerning the treated groups (those with peptic ulcers) showed that the group given an omeprazole-and basal diet had the highest pH values, whereas the group fed a basal diet containing 2.5% coconut fruit powder had the lowest pH values with a significant difference. The averages were 6.83 and 3.11, respectively.

The control positive group's acidity was found to be much higher than that of the negative control group, according to the data. The averages were 417.15 and 115.60 meq/L. Conversely, the data from the treated groups (those with peptic ulcers) showed that the group fed 2.5% coconut fruit powder had the greatest values, while the group fed a standard diet with omeprazole medication had the lowest values with significant variations. The averages were 295.40 and 193.60, respectively.

Data on stomach length revealed that the negative control group's value was significantly higher than the positive control group's. The average values were 89.10 cm and 96.40 cm. However, the results from the treated groups (those with peptic ulcers) showed that the group fed 5% coconut milk had the highest values, and the group fed a basal diet with 2.5% coconut fruit had the lowest values, with significant variations. The average readings were, respectively, 94.50 and 92.10 cm. These findings concur with those of Meng et al. (2019), who found that omeprazole and virgin coconut oil increased stomach mucus content and pH levels in a substantial ($P < 0.001$) way.

Furthermore, compared to the negative control group and the peptic ulcer groups that fed on varying amounts of powder, the positive control group's pH was 1.70, which was considerably lower ($P \leq 0.05$) **Khder *et al.*, (2018)**.

Table (2) Influence of coconut fruit and its milk on gastric juice, pH, acidity, and stomach length of peptic ulcer rats

Parameters / Groups	Gastric juice volume ml	pH	Acidity (Total acid) meq/L	Stomach length cm
G_1 C (-)	$1.52^{c}\pm1.71$	$8.60^{a}\pm0.23$	$115.30^{h}\pm0.22$	$96.40^{a}\pm2.15$
G_2 C (+)	$4.37^{a}\pm1.25$	$1.90^{g}\pm0.47$	$417.15^{a}\pm0.48$	$89.10^{c}\pm2.07$
G3 (2.5% coconut fruit)	$2.11^{b}\pm1.40$	$3.11^{f}\pm0.34$	$295.40^{b}\pm0.35$	$92.10^{b}\pm2.60$
G4 (5% coconut fruit)	$1.96^{b}\pm1.55$	$3.68^{e}\pm0.33$	$252.96^{d}\pm0.24$	$93.40^{b}\pm2.53$
G5 (250 ml coconut milk)	$1.85^{c}\pm1.42$	$4.00^{e}\pm0.24$	$283.93^{c}\pm0.40$	$93.00^{b}\pm2.35$
G6 (500 ml coconut milk)	$1.80^{c}\pm1.25$	$4.74^{d}\pm0.30$	$249.89^{d}\pm0.36$	$94.50^{a}\pm2.44$
G7 (Mixture coconut) 2.5%+3.5ml	$1.67^{c}\pm1.48$	$5.20^{d}\pm0.15$	$238.37^{e}\pm0.41$	$94.60^{a}\pm2.71$
G8 (Mixture coconut) 5%+7ml	$1.63^{b}\pm1.56$	$5.93^{c}\pm0.46$	$224.15^{f}\pm0.31$	$95.40^{a}\pm2.90$
G9 Omeprazole Drug)	$1.59^{c}\pm1.15$	$6.83^{b}\pm0.14$	$193.60^{g}\pm0.36$	$95.21^{a}\pm2.14$
LSD ($P \leq 0.05$)	**0.502**	**0.720**	**3.531**	**1.470**

Each value is represented as mean ± standard deviation (n = 3).
Mean under the same column bearing different superscript letters are different significantly ($P \leq 0.05$).

Influence of coconut fruit and its milk on ulcer score, ulcer index, % ulceration and preventive index of rats suffering from peptic ulcer

Table 3 shows the effects of varying coconut fruit and its milk levels on the ulcer score, ulcer index, percentage of ulceration, and preventative index of gastric ulcer rats. It was found that when compared to gastric ulcer rats provided with coconut fruit and its milk, the preventive index showed a reverse pattern. Positive control rats given aspirin exhibited an increase in ulcer score, ulcer index, and ulceration (%). The impact of aspirin on the stomach wall mucosa may be the cause of these effects.

In comparison to positive control rats, feeding gastric ulcer rats coconut fruit and milk resulted in decreased ulcer scores, ulcer indexes, and ulceration percentages and increased preventative indexes. Because coconut fruit and milk have a high level of flavonoids and phenolic compounds, which have good antiulcer effects, they possess potent antioxidant activity, which could be responsible for the decrease in ulcer score, ulcer index, ulceration percentage, and increase in preventative

index. These findings are consistent with those of **Beck *et al.*, (1990)**, who found that aspirin is much more gastrotoxic than other nonsteroidal anti-inflammatory drugs (NSAIDs) despite having a relatively low potency as a COX inhibitor. Aspirin is primarily responsible for its localized impacts throughout the drug's stomach absorption.

Additionally, **Tolulope *et al.*, (2019)** provide confirmation for these findings, stating that in Wistar rats with ethanol-induced stomach ulcers, coconut milk potentiates antiulcerogenic actions; the most beneficial dose (70 mg/kg) was observed. As a result, coconut milk provides a therapeutic substitute for the treatment or cure of stomach ulcers.

Table (3) Influence of coconut fruit and its milk on ulcer score, ulcer index, % ulceration and preventive index of rats suffering from peptic ulcer

Parameters / Groups	Ulcer score	Ulcer index	Ulceration %	Prevention index %
G_1 C (-)	------	------	------	------
G_2 C (+)	9.85^{a}±0.53	985.00^{a}±6.75	83.33^{a}±2.60	16.67^{e}±1.15
G3 (2.5% coconut fruit)	5.73^{d}±0.35	573.00^{b}±4.45	66.67^{b}±2.40	33.33^{d}±2.40
G4 (5% coconut fruit)	2.51^{e}±0.15	251.00^{d}±3.25	50.00^{c}±1.35	50.00^{c}±1.65
G5 (250 ml coconut milk)	5.30^{b}±0.50	530.00^{c}±4.15	83.33^{a}±2.30	16.67^{e}±1.22
G6 (500 ml coconut milk)	2.04^{c}±0.17	204.00^{e}±3.23	50.00^{c}±1.44	50.00^{c}±1.25
G7 (Mixture coconut) 2.5%+3.5ml	1.87^{d}±0.44	187.00^{f}±3.11	50.00^{c}±1.40	50.00^{c}±1.38
G8 (Mixture coconut) 5%+7ml	0.93^{e}±0.25	93.00^{g}±2.51	33.34^{d}±1.32	66.67^{b}± 1.43
G9 Omeprazole Drug)	0.85^{e}±0.65	85.00^{h}±2.65	16.67^{e}±1.20	83.33^{a}±2.27
LSD ($P \leq 0.05$)	**0.520**	**4.520**	**1.641**	**1.572**

Each value is represented as mean ± standard deviation (n = 3).

Mean under the same column bearing different superscript letters are different significantly ($P \leq 0.05$).

The influence of coconut fruit and its milk on the oxidative enzymes of rats suffering from peptic ulcer

The data in Table (4) illustrates the influence of coconut fruit and its milk on the concentrations of oxidative enzymes (CAT, GHS, SOD, and MDA) in rats with gastric ulcers. It is clear to see that there were significant variations between the CAT enzyme levels of the positive and negative control groups; the first group had the greatest value, while the other group had the lowest. The respective mean values were 1.23 u/g and 6.47 u/g. On the other hand, among the treated groups (peptic ulcer),

the 5% coconut combination of fruit and milk had the greatest CAT enzyme value, while the 2.5% coconut fruit had the lowest value, both of which were statistically significant. The average readings were 3.99 u/g and 5.36 u/g, respectively.

Conversely, there were significant variations between the GSH enzyme levels of the positive and negative control groups: the first group had higher values than the second group. The averages values were 338.50 and 153.00 u/g, respectively. In contrast, a 5% coconut fruit and milk combination had the highest GSH enzyme among the treated groups (peptic ulcer patients), while a 2.5% coconut fruit had the lowest value. These differences were statistically significant. In accordance, the mean values were 228.94 and 202.60 u/g.

Data on SOD enzyme levels showed that there were significant variations between the negative and positive control groups, with the negative group having the highest levels and the positive group having the lowest. The average values were 617.13 and 212.35 mmol/g, respectively. In terms of SOD enzyme, treatment groups (peptic ulcer) found that the 2.5% coconut fruit had the lowest values, and the 5% coconut mixture of fruit and milk had the greatest values, with a significant difference, 470.36 and 418.25 mmol/g on average, respectively.

Conversely, the MDA enzyme levels varied significantly between the positive and negative control groups, with the first group having the highest levels and the second group having the lowest. The corresponding relative mean values were 2.25 and 10.26 mmol/g. On the other hand, 2.5% coconut fruit had the highest MDA enzyme among the treated groups (peptic ulcer), but a 5% coconut fruit and milk combination had the lowest value. These differences were statistically significant. The relative mean values were 4.91 mmol/g and 6.28 mmol/g. Furthermore, a decline in nitrite MDA and a rise in GSH, CAT, SOD, and GPx levels indicated that coconut had a protective effect on rats when exposed to ethanol **Meng *et al.*, (2019)**.

In addition, the 14-day treatment of vitamin E and tender coconut water raised SOD and CAT levels while lowering MDA levels. We may therefore conclude that, in rats, tender coconut water can decrease MDA and improve antioxidant enzymes since SOD is an essential antioxidant metalloenzyme that effectively catalyzes the conversion of superoxide ions into oxygen and H2O2. By removing superoxide radicals, SOD protects cells against harm caused by free radicals. Following coconut treatments, there was a rise in catalase activity in the tissues, suggesting that the consumption of coconut lowers hydrogen peroxide content and its breakdown, which in turn reduces oxidative stress **Zulaikhah *et al.*, (2022)**.

Table (4) Influence of coconut fruit and its milk on oxidative enzymes of peptic ulcer rats

Each value is represented as mean ± standard deviation (n = 3).
Mean under the same column bearing different superscript letters are different significantly (P≤ 0.05).

Influence of coconut fruit and its milk on serum liver enzymes of peptic ulcer rats

The information in Table (5) illustrates how different amounts of coconut fruit and its milk affect the liver enzymes (ALP, AST, and ALP) in rats with peptic ulcers. According to the collected data, the negative control group had the lowest serum ALT level with statistically significant differences, whereas the positive control group had the highest values. The corresponding means were 60.68 U/L and 114.84 U/L. Conversely, the 2.5% coconut fruit group had the highest ALT

Parameters / Groups	CAT u/g	GSH u/g	SOD mmol/g	MDA mmol/g
G_1 C (-)	6.47^{a}±1.20	338.50^{a}±4.20	617.13^{a}±6.15	2.25^{d}±0.17
G_2 C (+)	1.23^{d}±0.75	153.00^{f}±2.52	212.53^{f}±3.21	10.26^{a}±1.02
G3 (2.5% Coconut fruit)	3.99^{c}±0.50	202.60^{e}±3.40	433.60^{d}±4.47	6.28^{b}±1.12
G4 (5% Coconut fruit)	4.32^{b}±0.17	215.42^{c}±3.65	451.35^{c}±4.65	5.62^{b}±0.72
G5 (250 ml Coconut milk)	4.41^{b}±0.83	203.75^{e}±3.42	418.25^{e}±4.11	5.72^{b}±0.94
G6 (500ml Coconut milk)	4.59^{b}±0.95	208.55^{d}±4.25	435.47^{d}±4.35	5.65^{b}±0.48
G7 (Mixture coconut)	5.10^{b}±1.02	213.70^{c}±3.38	455.15^{c}±4.50	5.28^{b}±0.36
G8 (Mixture coconut)	5.17^{b}±1.05	228.94^{b}±4.27	470.36^{b}±4.96	4.91^{c}±0.61
G9 (Omeprazole drug)	5.36^{a}±1.10	226.40^{b}±4.27	464.55^{b}±4.66	5.24^{b}±0.57
LSD (P ≤ 0.05)	**1.120**	**3.602**	**5.711**	**1.102**

levels of the treatment groups (peptic ulcer), and the 5% coconut fruit and milk combination showed the lowest values. There was a statistical significance in these differences. The corresponding means were 94.64 *8* and 63.26 U/L, respectively.

The obtained data showed that, with significant differences, the serum AST levels were highest in the positive control group and lowest in the negative control group. the average being 147.64 and 70.10 U/L, respectively. Conversely, the peptic ulcer treatment groups exhibited statistically significant differences in serum AST levels, with the greatest levels observed for 2.5% coconut fruit and the lowest for 5% mixture of coconut. The average values were 90.45 and 118.65 U/L, respectively.

The findings showed statistically significant differences between the two groups, with the positive control group having the highest serum ALP levels and the negative control group having the lowest. The relative mean values were 325.00 and 124.40 U/L, respectively. However, serum ALP levels varied among the treated groups (peptic ulcer), with statistically significant variations found between the highest observed serum ALP level for 2.5% coconut fruit and the lowest observed serum ALP level for 5% mixed coconut fruit and milk. The averages were 213.50 and 154.10 mg/dl, in that order. These results corroborate those of **Indra *et al.*, (2015)**, who suggested that coconut milk, which has high concentrations of potassium, magnesium, iron, and zinc along with a good supply of vitamins E and C, had a superior impact on improving the nutritional status of patients with liver cirrhosis. In relation to other acids and hyaluronic acids. Furthermore, **Outcheer *et al.*, (2013)** demonstrated that the oil found in coconut flesh shields albino rats exposed to the anti-folate mixture trimethoprim sulfamethoxazole against liver injury.

Additionally, dietary fibers can bind with bile acids and block their reabsorption in the liver, inhibiting the creation of cholesterol. This is why the diet supplemented with coconut powder enhanced the liver's enzymes and significantly lowered the activity of AST, ALT, and ALP enzymes in rats **Trinidad *et al.*, (2003)**.

Table (5) Effect of coconut fruit and its milk on liver enzymes of peptic ulcer rats

Parameters / Groups	ALT U/L	AST U/L	ALP U/L
G_1 C (-)	60.68^{f}±1.63	70.10^{g}±1.95	124.40^{g}±1.60
G_2 C (+)	114.84^{a}±1.75	147.64^{a}±2.45	325.00^{a}±4.50
G3 (2.5% coconut fruit)	94.64^{b}±3.45	118.65^{b}±3.46	213.50^{b}±1.61
G4 (5% coconut fruit)	86.74^{c}±3.24	113.55^{c}±1.48	193.00^{c}±3.20
G5 (250 ml coconut milk)	83.81^{c}±1.40	111.90^{c}±2.71	191.40^{c}±3.43
G6 (500 ml coconut milk)	64.32^{e}± 0.80	101.81^{e}±3.45	178.11^{d}±2.60
G7 (Mixture coconut) 2.5%+3.5ml	77.64^{d}±1.65	104.40^{d}±2.40	172.40^{e}±2.80
G8 (Mixture coconut) 5%+7ml	63.26^{e}±1.74	90.45^{f}±1.96	154.10^{f}±2.11
G9 (Omeprazole drug)	80.16^{d}±1.05	106.40^{d}±2.57	171.44^{e}±4.66
LSD (P ≤ 0.05)	**3.484**	**3.316**	**4.373**

Each value is represented as mean ± standard deviation (n = 3).

Mean under the same column bearing different superscript letters are different significantly (P≤ 0.05).

Influence of coconut fruit and its milk on serum lipid fraction of peptic ulcer rats

The information in Table (6) showed the average T.C., T.G., LDL-c, and VLDL-c (mg/dl) of rats with peptic ulcers that were fed different diets. Compared to the control (-ve) group, which recorded 75.50, 68.00, 15.42, and 13.60 mg/dl, respectively, the rates of control +ve had a significantly higher concentration of T.C., T.G., LDL-c, and VLDL-c, recording 163.76, 142.62, 114.97, and 28.52 mg/dl, respectively. Rats given a basal diet containing 2.5%–5% coconut fruit powder and 250–500 ml of coconut milk showed significant reductions in T.C., T.G., LDL–c, and VLDL–c concentrations. The average serum (HDL-c) level in rats with peptic ulcers fed various diets. It was evident that the control (+) group's mean value of (HDL-c) was lower compared to that of the control (-) group, which were 20.27 and 46.48 mg/dl, respectively, showing significant difference. Our findings are consistent with those of **Scheurig *et al.*, (2008)**, who found that Pantothense, an ingredient in coconut milk, helped improve HDL cholesterol good and decrease the harmful LDL cholesterol. Pantothense seems to decrease triglyceride and cholesterol concentrations in individuals with high cholesterol in several clinical trials. **Nevin and Rajamohan (2008)** found that virgin coconut oil raised high density lipoprotein cholesterol while lowering total cholesterol, triglycerides, and very low-density lipoprotein cholesterol and low-density lipoprotein cholesterol.

Furthermore, **Ekanayaka *et al.*, (2024)** showed that supplementing with coconut milk led to positive changes in the lipid profile, with an increase in HDL and a drop in LDL and non-HDL levels. Supplementing with coconut milk seemed to be more beneficial for the subgroup whose baseline LDL level was increased.

Table (6) Influence of coconut fruit and its milk on lipid fraction of peptic ulcer rats

Parameters / Groups	TC mg/dl	TG mg/dl	HDL-c mg/dl	LDL-c mg/dl	VLDL-c mg/dl
G_1 C (-)	75.50^{g}±2.42	68.00^{e}±0.70	46.48^{a}±2.31	15.42±1.13	13.60^{g}±1.60
G_2 C (+)	163.76^{b}±2.75	142.62^{a}±4.30	20.27±3.22	114.97±2.42	28.52^{a}±4.50
G3 (2.5% coconut fruit)	141.30^{b}±2.10	110.78^{b}±2.83	28.88^{e}±1.51	90.26±1.70	22.16^{b}±1.61
G4 (5% coconut fruit)	123.25^{c}±4.50	98.69^{c}±3.82	35.66^{d}±1.53	67.85±1.42	19.74^{c}±3.20
G5 (250 ml coconut milk)	120.50^{c}±1.90	70.45^{e}±4.76	40.08^{bc}±2.31	66.33±1.63	14.09^{c}±3.43
G6 (500 ml coconut milk)	112.95^{d}±2.74	87.05^{d}±1.55	43.10^{ab}±2.79	52.44±1.30	17.41^{d}±2.60
G7(Mixture coconut) 2.5%+3.5ml	102.13^{e} ±3.29	73.12^{e} ± 3.40	38.50^{cd}±2.89	49.31±1.24	14.32^{e}±2.80
G8 (Mixture coconut) 5%+7ml	82.85^{f}±3.70	68.28^{ef}± 3.03	44.90^{a}±2.46	24.29±1.45	13.66^{f}±2.11
G9(Omeprazole drug)	107.50^{c}±1.90	88.50^{c}±3.82	38.77^{d}±1.33	51.03±1.35	17.70^{e}±4.66
LSD (P≤ 0.05)	**2.891**	**2.610**	**1.208**	**1.9178**	**1.159**

Each value is represented as mean ± standard deviation (n = 3).

Mean under the same column bearing different superscript letters are different significantly (P≤ 0.05).

Conclusion

The high concentrations of antioxidant and active compounds in coconut fruit and milk enhanced the oxidative enzymes, liver enzymes, serum lipid profiles, and gastric ulcers in rats. Thus, this research highlights the potential of using coconut fruit and milk as natural dietary supplements to enhance diets and lower risk factors for long-term diseases, such as gastric ulcers.

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التأثيرات البيوكيميائية لفاكهة ولبن جوز الهند في الفئران المصابة بقرحة المعدة الناجمة عن الأسبرين

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الملخص العربى

جوز الهند غني بشكل أساسي بالماء والسكر والدهون والبروتين والأحماض الأمينية والفيتامينات والمعادن والمواد الكيميائية النباتية. وقد أظهرت هذه المواد فوائد إيجابية على صحة الإنسان والحيوان، مما أدى إلى تقليل خطر الإصابة بالعديد من الأمراض، بما في ذلك قرحة المعدة. الهدف الرئيسي من هذا البحث هو تقدير مدى فعالية مسحوق ثمار ولبن جوز الهند ودواء أوميبرازول المضاد للقرحة في تقليل قرحة المعدة لدى الفئران. من بين ٥٤ فأرًا تم استخدامها في هذا البحث، تم اعداد تسع مجموعات كل مجموعه تحتوي علي ستة فئران ألبينو اناث، وزن كل منها ١٥٠جم ± ١٠ جم. لاحداث قرحة في المعدة، تم إعطاء الفئران ٣٠٠ ملجم/كجم من وزن الجسم. للحث على القرحة. كما تم أيضا قياس التهاب المعدة، ودرجة الحموضة، والحموضة، وطول المعدة، ودرجة القرحة، ومؤشر القرحة، ونسبة التقرح، والمؤشر الوقائي. وشملت التحاليل البيوكيميائية قياسات الدهون الثلاثية (**TG**)، والبروتين الدهني عالي الكثافة (**HDL-c**)، والبروتين الدهني منخفض الكثافة (**LDL-c**)، والبروتين الدهني منخفض الكثافة جدًا (**VLDL-c**)، وإنزيمات الكبد (**ALT**، **AST**، و**ALP**) والإنزيمات المؤكسدة (**CAT**، **SOD**، **NAD**، **GPx**). وعلاوة على ذلك، استخدام جهاز **HPLC** للتعرف على المركبات الفينولية. أظهرت النتائج أن مجموعة الفئران المصابة بقرحة المعدة أظهرت تحسنًا كبيرًا في صورة دهون الدم وانزيمات الكبد والإنزيمات المؤكسدة وقرحة المعدة عند تناول مسحوق ثمار جوز الهند وحليبها. في نهاية التجربة، أظهرت الفئران المصابة بقرحة المعدة التي أعطيت ٥٠٠ ملجم / كجم من لبن جوز الهند نسبة أفضل من تثبيط القرحة وتحسين التحاليل الكيميائية الحيوية. يشير هذا إلى أنه يمكن استخدام فاكهة ولبن جوز الهند كمكملات غذائية طبيعية لتحسين النظام الغذائي وتقليل عوامل الخطر للأمراض المزمنة مثل قرحة المعدة.

الكلمات الافتتاحية: ثمار و لبن جوز الهند، اناث الفئران, أومبيرازول, قرحه المعده, الانزيمات المؤكسدة, انزيمات الكبد.