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Gastroprotective Studies of Raw and Processed African pear (*Dacryodes edulis* var. *edulis*) on Experimentally Induced Gastric Mucosa Ulcer in Wistar Rats

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Abstract

Gastric ulcer is acknowledged as one of the leading human gastroduodenal disorders in the globally; hence, a study on gastroprotective studies of raw and processed African pear (*dacryodes edulis* var. *edulis*) in Wistar rats was carried out. A total of 84 Wistar rats were used for the study. The study was conducted in two phases with 42 animals each and 6 rats randomly assigned into seven groups in each phase. The groups were the control and groups fed diets supplemented 15% raw sample, 30% raw sample, 15% roasted sample, 30% roasted sample, 15% boiled sample and 30% boiled sample with normal rats chow accordingly. Water and food were allowed *ad libitum* to all the groups. At the end of 28 days feeding period, Gastric acid and pH, mucus, pepsin secretion and ulcer studies were done using standard procedures. Results shows significantly ($p < 0.05$) high mean mucus output and gastric juice pH in group fed 30% raw compared to the control other experimental groups. The ulcer scores in the groups fed 15 % roasted was significantly ($p < 0.05$) higher compared to the control and other experimental groups. The pepsin secretion in the group fed 30 % boiled sample was significantly ($p < 0.05$) increased compared to the control group and other experimental groups. The basal gastric acid secretion was significantly ($p < 0.05$) higher in groups processed sample, compared to the groups fed raw sample and the control group. We therefore conclude that processed African pear contains phytochemical agents that increase gastric secretion probably via H_2-histaminergic receptors. Hence, consumption of processed of African pear pulp should be done with caution, while intake of raw pulp should be encouraged.

Keyword: African Pear; Raw and Processed; Pepsin; Mucus; Gastric Acid; Ulceration

1. Introduction

Gastric ulcer is recognized as one of the leading human gastroduodenal disorders in the globe. It is a condition described by a hurting lesional excoriation, lengthening up to the muscular mucosa of the gastric and/or duodenal wall, and not often to the lower region of the esophagus close to the *ostium cardiacum*. Gastric ulcer can be deactivating due to epigastralgias, even serious if the stomach is punctured, giving rise to internal bleeding and to some point death if not urgently treated.[1] The disease condition occurs following an imbalance between the aggressive factors of gastric acid and pepsin and/or the reduction of the cytoprotective capacities of the gastroduodenal mucosal barrier by the secretion of mucus, bicarbonate, prostaglandins, and cellular antioxidants.[2] The digestive tract is covered by a mucous membrane, a highly hydrated visco-elatsic gel containing bicarbonate which guards the gastric tissue from the mechanical pressure as well as the corrosive characteristics of gastric acid and pepsin.[3] In the pathogenesis of gastric ulcers, the main factors

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are infection with *Helicobacter pylori* bacteria, use of non-steroidal anti-inflammatory drugs (NSAIDs) such as acetylsalicylic acid (ASA), essentially chronic emotional stress, overconsumption of alcohol and related drinks, and tobacco smoking.[4] Worldwide, more than half of the globe's populace is pest-ridden by this bacteria and this rate reaches about 80 % in developing countries.[5] The long-term use of NSAIDs triggers gastric mucosal irritation and bleeding, and produce ulcers by inhibiting the cyclooxygenase enzyme in prostaglandin biosynthesis.[6]

A balanced therapy for peptic gastric lesion still remains subtle and search for safer potential drugs is pertinent. Although H_2 receptors (ranitidine, famotidine), proton-pump inhibitors (omeprazole, lansoprazole), antibiotics (metronidazole, amoxicillin, clarithromycin, tetracycline etc.) and other drugs are currently used for the proficient controlling of the peptic gastric lesion infections, they all have limitations, thereby compounding the disease condition.[3] Some of these side effects are abdominal pains, headache, vertigo, arrhythmias, metabolic disorders (hypomagnesemias, Vitamin B_{12} deficiencies, and hematopoietic changes), nephritis, bone damage, and some forms of cancer.[7] Hence, the need for alternative and more safer medicines or therapies, based on the much diversified natural potential of bioactive compounds in plants that would clearly be less aggressive than conventional drugs.[8] Studies have reported the gastroprotective and/or healing effects of many plant extracts on peptic ulcer induced in animal models.[8,9]

Dacryodes edulis is a versatile plant in African traditional medicine, as its various parts are employed for the treatment of several diseases. Its bark has long been used in Nigeria to cicatrize wound, and for the treatment of leprosy, dysentery, anaemia, spitting blood, debility, stiffness, tonsillitis and skin diseases.[10] It is commonly known as native (local) pear and is called "eben" by the Ibibios, "ube" by the Igbos, and "elemi" by the Yorubas of Southern, Eastern and Western Nigeria respectively.[11] The fruit is also known as *safou* in French and *orumu* in Benin.[12] On the basis of long-term and extensive field observations, two varieties of *D. edulis* were distinguished: *D. e.* var*. edulis* which is large, elongated, and cylindrical and the fruit pulp is thick and *D. e.* var*. parvicarpa* which is small, rounded or more or less conical, having a thin pulp.[13] Recently, Nwaogu and Oluwamukomi [14] and Agiang *et al.* [15] have reported that *D. edulis* var. is more nutritious and possesses antioxidant properties. Accordingly, Agiang *et al.* [15] revealed that the raw *D. e.* var*. edulis* contained more nutritional values than the processed *D. e.* var*. edulis.* The presences of these chemical compounds in foods have been reported to provide for the plant some healing abilities, capable of upholding the use of plants in ancient medicine. Studies have disclosed that processing affect the antioxidant properties and phytochemical profile of plant, including African pear pulp.[14,16] However, the reported significant influence of the various processing methods of *Dacryodes edulis* on the chemical compounds is worrisome, following their medicinal potentials. Hence, this study was aimed at investigating the effect of raw and processed African pear (*Dacryodes edulis* var. *edulis*) on gastric acid secretion and ulcerations in Wistar rats.

2. Materials and methods

2.1. Collection and preparation of samples

The mature fruits of *Dacryodes edulis* (D.e.var.*edulis*) were plucked directly from trees and were divided into three batches. Batch I was treated raw; the seeds were removed from the pulp, batch II was roasted over hot coals, with constant turning until soft, and batch III was put into boiled water poured into a bowl and left for five minutes to soften. In each case, the pulp was oven dried at 60^{o}C for 12 hours. All dried samples were then ground separately to fine powder and then filtered to pass through a 0.5 mm pore sieve and stored in different air tight containers in the refrigerator. Experimental design and animal grouping. A total of 84 Wistar rats with average weight of 110 ± 20 g were used for the study. The study was done in two phases with 42 animals each and 6 rats randomly assigned into seven groups in each phase; Group A (control group): Fed with normal rat chow and water *ad libitum*. Group B: Fed with 15% and 85% of raw African pear and rat chow respectively and water *ad libitum.* Group C: Fed with 30% and 70% of raw African pear and rat chow respectively and water *ad libitum.* Group D: Fed with 15% and 85% of roasted African pear and rat chow respectively and water *ad libitum.* Group E: Fed with 30% and 70% of roasted African pear and rat chow respectively and water *ad libitum.* Group F: Fed with 15% and 85% of boiled African pear and rat chow respectively and water *ad libitum.* Group G: Fed with 30% and 70% of boiled African pear and rat chow respectively and water *ad libitum.* Each phase of the experimental feeding lasted for 28 days with food and water given *ad libitum*.

Food intake and body weight changes were measured as follows:

- Daily Food intake (g/28days) = Food supplied to each rat – (leftover + spilled food)
- Body weight change (g) = Final weight – initial weight of each rat.

Phases of the study: Phase 1 used a total of 42 Wistar rats in 0.1N HCl was transferred to a tube and incubated for 30 minutes with 3.9ml of 25g/100ml 0.1N HCl of the substrate in a water bath at 370C. Ten mililitres of 10% trichloroacetic acid (TCA) was added and the tubes allowed tostands for 10 minutes before they were filtered using Whatman's filter paper No. 1. Blanks were made for each concentration by adding 10ml TCA before adding enzyme. Duplicate determinations were performed for each enzyme concentration. The optical density of the filtrate was measured at 280nm wavelength. For determination of the proteolytic activity of gastric secretion, the same procedure was followed at a concentration of 2% of 0.1N HCl (by adding 1ml of 2% of 0.1N HCl into each). A standard curve was constructed from which pepsin content of gastric secretion was determined.

Extraction of adherent mucus. Adherent mucus weight was determined according to the method described by Tan *et al.*[17]. The animals were starved for 24 hours. The stomach was removed and washed in normal saline and then opened along the greater curvature. It was again rinsed in saline and pinned to a cork board with dissecting pins. Mucus was extracted using a spatula from the spread stomach into a known weight of sample tube containing 2ml of water. The weight of mucus was derived from the difference in the initial and final weights of beaker + 2 ml of water.

2.2. Preparation of animals for collection of gastric acid

Prior to the collection of gastric acid, animals were fasted for 12 to 18 hours to get rid of any fecal matter in the stomach. This was followed by intraperitoneal injection of 25% ketamine at a dose of 6ml per Kg body weight of the animal. The animals were then laid on the dissecting board, an incision was made on the neck to expose the tracheal for trachea cannulation, and this was to allow for clear airways. Another incision was made on the abdomen about an inch along the linea alba. The stomach was exposed and the thread passed at the pyloric end under the pyloric sphincters. In this case, the small intestine and part of the liver were exposed. A transection of the duodenum was made near the pyloric sphincter and another cannula placed and firmly held with a thread, care was taken not to rupture blood vessels of the duodenum, and after this the stomach and other exposed structures were put back and covered with normal saline swab. An orogastric infusion tube was carefully passed from the mouth through the oesophagus to the stomach and ligated just behind the tracheal cannula to prevent reflux; care was taken not to damage the vagus nerve. The exposed end of the orogastric tube was passed through a water bath to maintain the perfusate at a physiological temperature (37^oC) and to pre-warm the experimental solution. The other end of the orogastric tube was attached to a 50ml syringe mounted on a perfusor pump. The animal was warmed with a table lamp to maintain the body temperature at 37^oC. The temperature was monitored with the help of a rectal thermometer.

2.3. Measurement of gastric acid output

The continuous perfusion method by Gosh and Schild,[18] modified by Amure[19] was used. The rats were first perfused with 0.9% normal saline at the rate of 1.0ml/min. The aliquots were collected after 10 minutes of infusion and were titrated with 0.01N of NaOH using phenolphthalein as an indicator. A pink colour discharge indicated the end point of the titration and the volume of the base used was read off and recorded. The first 10-20 minutes effluent collected was discarded to avoid erroneous acid secretion induced by trauma. When a stable acid secretion was obtained, histamine (a secretagogue) and cimetidine (histamine H_2-receptor antagonist) were administered and acid output determined every 10minutes using the method described above.

2.4. Ulcer study

Animals were starved for about 18hours prior to the start of the experiments. Then they were anesthetized with 25% urethane at a dose of 6ml/kg body weight i.p., the stomach was exposed via a midline incision along the linea alba. 2mls of equal volumes of acid (0.1N HCl) and alcohol (70% ethanol) was introduced into the stomach via a proximal end of the duodenum near the pyloric sphincter and kept in place with an eight shaped ligature a little above the incision. The incision was covered with a cotton wool dapped in normal saline. The animals were then kept for 1 hour. Thereafter, the stomach was isolated and excised through the greater curvature. It was then rinsed with normal saline. Pins were used to fasten the tissues in place for proper visualization. A magnification lens and a Vanier caliper were used to measure the ulcers. Ulcer scores were determined according to the method of Ohara *et al*.[20]

2.5. Statistical analysis

All data are presented as mean + SEM. The one way ANOVA was used to analyze the data, followed by a post-hoc test (LSD). The results were considered significant at $p < 0.05$.

3. Results

3.1. Effect of raw and processed African pear on mucus secretion in Wistar rats

The mean mucus output (g) in the control group and groups fed 15% raw, 30% raw, 15% roasted, 30% roasted, 15% boiled and 30% boiled samples were 0.02 ± 0.002, 0.02 ± 0.000, 0.06 ± 0.002, 0.04 ± 0.000, 0.03 ± 0.000, 0.05 ± 0.002 and 0.04 ± 0.000 respectively. The mean mucus output was significantly ($p < 0.05$) higher in groups fed 30% raw compared to the control other experimental groups (Table 1).

3.2. Effect of raw and processed African pear on ulcer score in Wistar rats

The mean ulcer scores were significantly higher ($p < 0.05$) in all the experimental groups compared to the control (Table 1). The ulcer scores in the groups fed 15% roasted was significantly ($p < 0.05$) higher compared to the control and other experimental groups.

3.3. Effect of raw and processed African pear on gastric juice pH in Wistar rats

The mean gastric juice pH in the control group and groups fed 15% raw, 30% raw, 15% roasted, 30% roasted, 15% boiled and 30% boiled samples were 3.65 ± 0.43, 2.42 ± 0.03, 5.30 ± 0.04, 2.58 ± 0.09, 5.10 ± 0.11, 5.00 ± 0.27 and 2.46 ± 0.62 respectively. The administration of the samples significantly ($p < 0.05$) increased the gastric juice pH compared to control group. The gastric juice pH was significantly ($p < 0.05$) higher in groups fed 30% raw sample compared to the control and other experimental groups (Table 1).

3.4. Effect of raw and processed African pear on pepsin secretion in rats

The mean pepsin secretion (mg/100 ml) in the control group and groups fed 15 % raw, 30 % raw, 15 % roasted, 30 % roasted, 15 % boiled and 30 % boiled samples were 0.45 ± 0.01, 0.46 ± 0.00, 0.48 ± 0.01, 0.50 ± 0.00, 0.49 ± 0.00, 0.53 ± 0.01 and 0.59 ± 0.01 respectively. The pepsin secretion in the group fed 30 % boiled sample was significantly ($p < 0.05$) increased compared to the control group and other experimental groups (Table 1).

Table 1 Effect of raw and processed African pear on mucus secretion, ulcer scores, gastric juice pH and pepsin secretion in Wistar rats

Groups	Mucus secretion (g)	Ulcer Scores	pH of gastric juice	Pepsin secretion (mg/100 ml)
Control	0.02 ± 0.002	3.40 ± 0.24	3.65 ± 0.43	0.45 ± 0.006
15% Raw Sample	0.02 ± 0.000	5.60 ± 0.93*	2.42 ± 0.03*	0.46 ± 0.004
30% Raw sample	0.06 ± 0.002*,a	7.00 ± 0.16*	5.30 ± 0.04*,a	0.48 ± 0.007*,a
15% Roasted Sample	0.04 ± 0.000*,a,b	17.80 ± 0.66*,a	2.58 ± 0.09*,b	0.50 ± 0.003*,a
30% Roasted sample	0.03 ± 0.000*,a,b,c	6.20 ± 0.85*,b,c	5.10 ± 0.11*,a,b,c	0.49 ± 0.004*,a
15% Boiled Sample	0.05 ± 0.002*,a,b,c,d	13.60 ± 0.51*,a,c,d	5.00 ± 0.27*,a,c	0.53 ± 0.007*,a,b,c,d
30% Boiled sample	0.04 ± 0.000*,a,b,d,e	8.10 ± 0.33*,a,b,c,d,e	2.46 ± 0.62*,b,d,e	0.59 ± 0.007*,a,b,c,d,e

Values are expressed as mean ± SEM, n = 6; *significantly different from Control at $p < 0.05$; a = significantly different from 15% Raw sample at $p < 0.05$; b=significantly different from 30% Raw sample at $p < 0.05$;c = significantly different from 15% Roasted sample at $p < 0.05$; d = significantly different from 30% Roasted sample at $p < 0.05$;e = significantly different from 15% Boiled sample at $p < 0.05$.

3.5. Effect of raw and processed African pear on Gastric acid secretion in Wistar rats

The mean basal gastric acid output (Aliquots/hr) in the control group and groups fed 15% raw, 30% raw, 15% roasted, 30% roasted, 15% boiled and 30% boiled samples were 4.05 ± 0.15, .28 ± 0.03, 6.73 ± 0.00, 8.10 ± 0.22, 6.8 ± 0.17, 11.30 ± 0.36 and 11.40 ± 0.046 respectively. The basal gastric acid output was significantly ($p < 0.05$)higher in all the experimental groups except in the case group fed 15% raw sample, which showed difference when compared to control group. Administration of histamine, an H_2 agonist, significantly ($p < 0.05$) raised gastric acid output in all the experimental groups compared to control. The increase was highest in the group fed 15 % boiled sample when compared to the control and other experimental groups. However, administration of cimetidine, H_2 blocker, significantly reduced the histamine induced acid secretion (Figures 1 and 2).

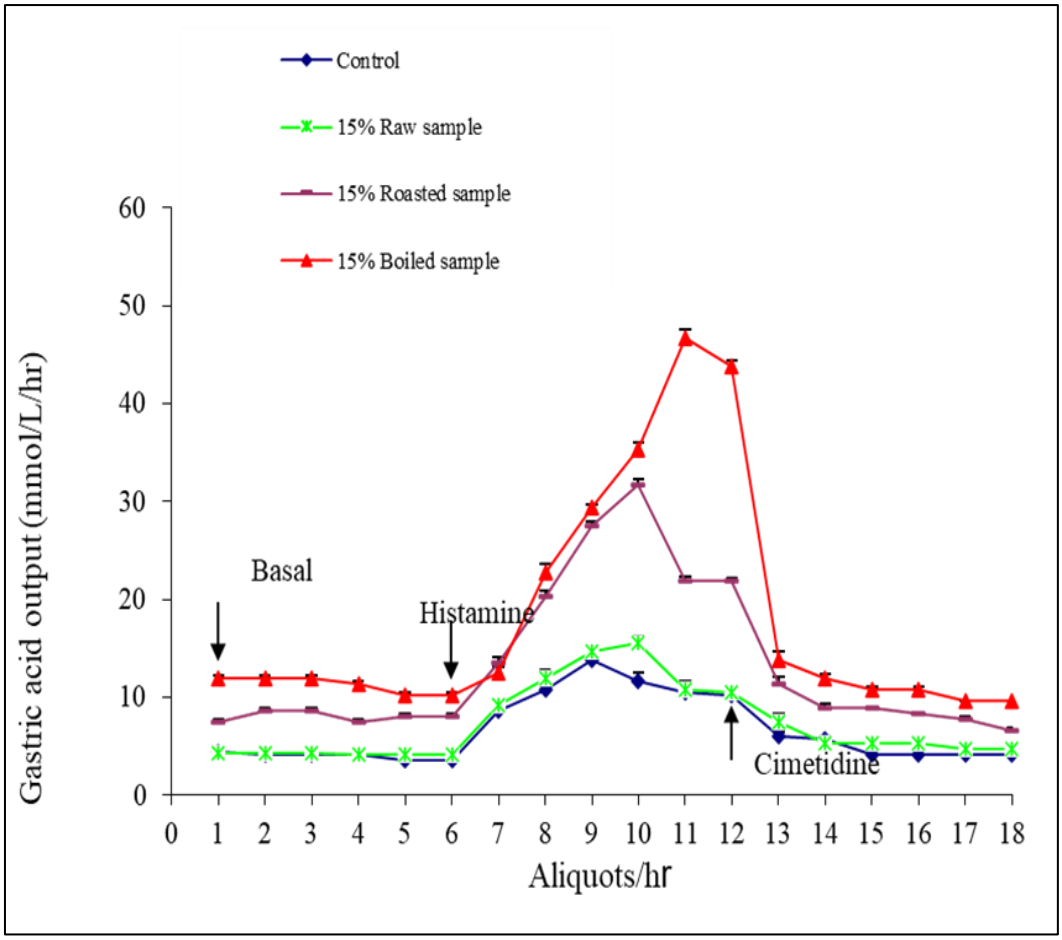

Figure 1 Gastric acid secretion in the control and groups fed 15 % raw, roasted and boiled samples; Values are expressed as mean ± SEM, n = 6

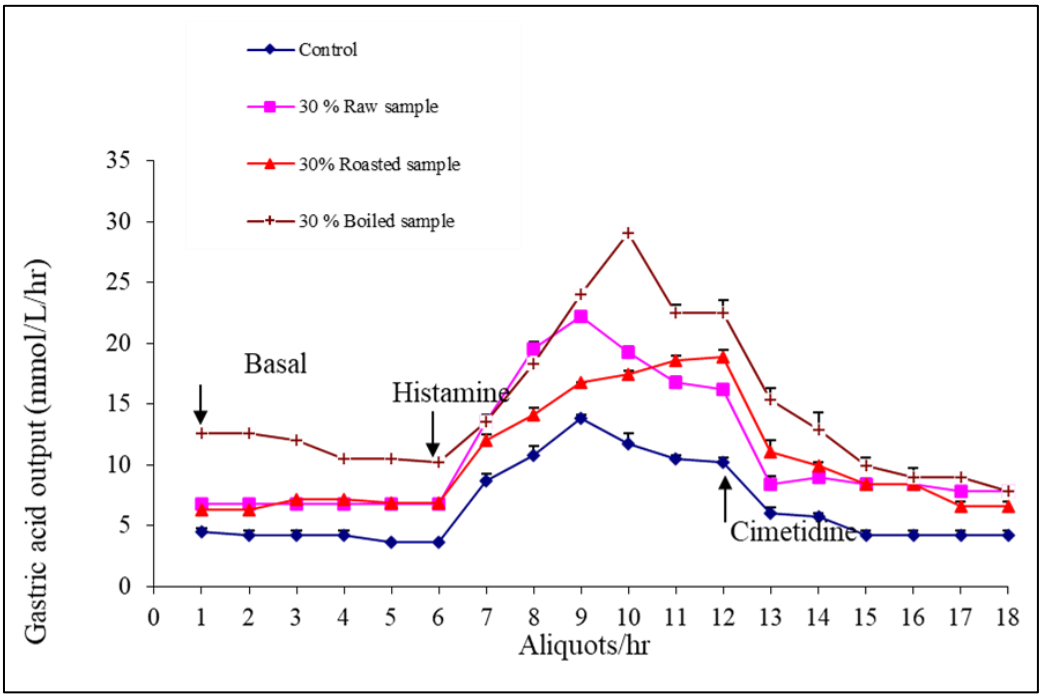

Figure 2 Gastric acid secretion in the control and groups fed 30% raw, roasted and boiled samples; Values are expressed as mean ± SEM, n = 6

3.6. Comparison of basal gastric acid secretion in control and experimental groups

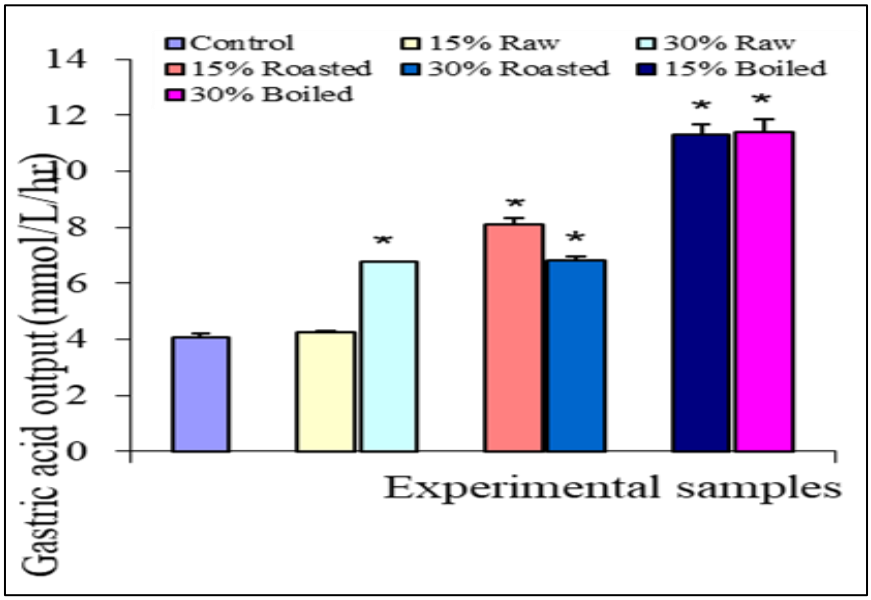

Figure 3 Basal gastric acid output between the control and groups fed 15% and 30% samples; Values are expressed as mean ± SEM, n = 6; * = significantly different from control at p < 0.05; a = significantly different from15% raw sample at p < 0. 05

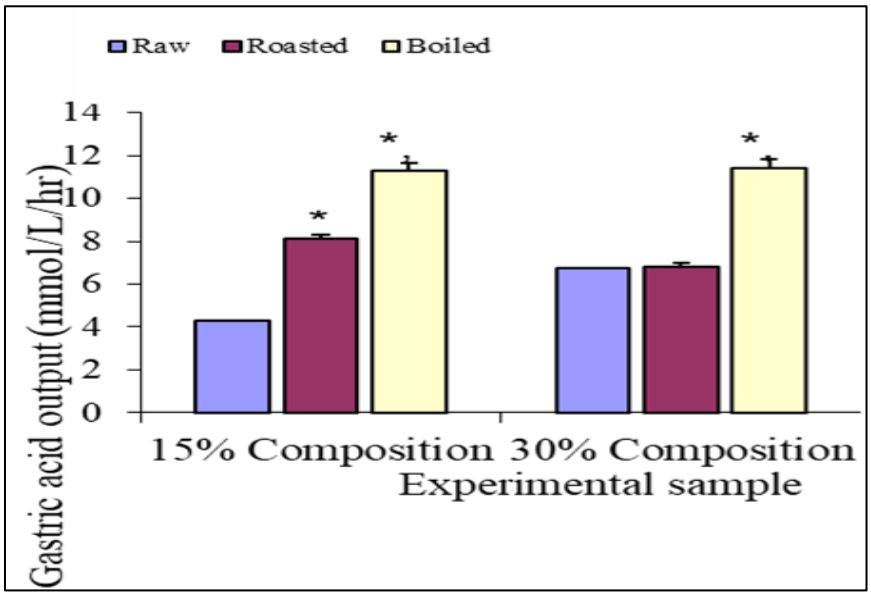

Figure 4 Comparison of basal gastric acid secretion between groups fed raw, roasted and boiled samples; Values are expressed as mean ± SEM, n = 6; * = significantly different from raw sample at p<0.05; a = significantly different from roasted sample at p < 0. 05

The basal gastric acid secretion was higher in all the experimental groups, compared to the control. Comparing the basal gastric acid secretion in groups fed 15% and 30% of the raw, roasted and boiled sample, there was no significant (p > 0.05) difference between groups fed 15% and 30% boiled samples, compared to those fed 15% and 30% raw and roasted samples (Figure 3). The mean gastric acid output (Aliquots/hr) in the groups fed 15% raw, 15% roasted and 15% boiled samples were 4.28 ± 0.03, 8.10 ± 0.22 and 11.30 ± 0.36 respectively. The basal gastric acid secretion was significantly (p < 0.05) higher in groups fed 15% roasted and 15% boiled sample, compared to the group fed 15% raw sample and the control group. Also, the basal gastric acid secretion was significantly (p < 0.05) higher in the group fed 30% boiled sample compared to groups fed 30% raw and 30% roasted samples. However, there was no significant (P > 0.05) difference in basal gastric acid outputs in group fed 30% roasted sample compared to group fed 30% raw sample (Figure 4).

3.7. Effect of raw and processed African pear on food intake and body weight changes

Results of the daily food intake and body weight changes of rats fed the experimental diets for 28 days and the control group is shown in Table 2. There was a gradual decrease ($p<0.05$) in body weight change with increase in the percentage of supplementation of the raw and processed African pear in the diet The feed intake was significantly ($p < 0.05$) higher (443.96 ± 4.84) in group fed 30% raw sample compared to the control (393.51 ± 8.08) and other experimental groups, though not different from group fed 15 % roasted sample (Table 2). The results show a significantly similar body weight changes in all the groups fed diets supplemented with raw and processed African pear compared with the control. The mean body weight changes were significantly ($p < 0.05$) higher (39.31 ± 1.13) in the group fed 30% raw sample compared to other experimental groups, though not different (38.49 ± 2.04) from the control group.

Table 2 Food intake and body weight changes of male Wistar rats fed diets supplemented with raw, roasted and boiled African pear

Groups	Initial weight (g)	Final weight (g)	Body weight change (g)	Food intake (g/28days)
Normal Control	73.14±0.94^{a}	111.63±2.85^{a}	38.49±2.04^{a}	393.51±8.08^{b}
15% Raw sample	82.30±0.49^{b}	111.64±2.85^{a}	29.34±2.71^{b}	414.39±6.20^{b}
30% Raw sample	94.14±0.30^{c}	133.45±1.24^{b}	39.31±1.13^{a}	443.96±4.84^{c}
15% Roasted sample	114.33±0.53^{d}	143.93±0.75^{c}	29.60±0.72^{b}	433.97±4.51^{c}
30% Roasted sample	103.53±0.84^{e}	128.43±0.92^{b}	24.91±1.01^{b}	405.75±9.64^{b}
15% Boiled sample	103.77±0.43^{f}	128.61±1.97^{b}	24.85±1.99^{b}	377.36±7.66^{a}
30% Boiled sample	126.48±1.21^{g}	145.04±2.42^{c}	18.56±1.91^{c}	363.91±6.99^{a}

Values are expressed as mean ± SEM, n = 6; Means with same superscript along each column were not significantly different ($P<0.05$)

4. Discussion

This study was designed to investigate the effect of raw and processed African pear on gastric acid secretion and ulceration. Results obtained from this study show that mean basal acid output in the boiled and roasted samples recipients was significantly higher compared with control and raw samples. The 30 % sample groups in turn were observed to have a significantly lower acid output compared to the 15 % sample groups. The samples also have percentage dependently increased gastric ulcerations compared with control. The result of this study is in line with the report of Nwaonukuru *et al.* [21] on the gastroprotective effect of *Dacryodes edulis*. The exact mechanism by which the processed samples increase basal and histamine induced gastric acid secretion is unknown. The high mucus secretion and high gastric pH juice obtained in the group fed 30 % raw samples might have been a contributor to the low gastric acid in this group of animals. Also, Nwaogu and Oluwamukomi[14] has reported that pulp of *D. edulis* var. possesses flavonoids, saponin, tannin, polyphenols which are capable of promoting gastric mucosal formation, reduce gastric acid secretion and inhibit pepsinogen production, thereby reducing gastric lesions and ulcers, and the concentration of these bioactive compounds were showed to be reduced by heat treatment (boiling and roasting) in the previous study. The results of this study contradict the recent report of Edem *et al.*[22], who stated that processed foods provides protection against a wide array of chronic metabolic diseases. The difference in the previous study may have been due to the processing method involved. However, it is obvious that the low levels of acid secretion and ulceration produced by groups fed 15% and 30% raw sample in this study could have also been due to the presence of these components in high quantities or some other mechanisms yet unidentified.

Histamine induced gastric acid output was blocked by administration of cimetidine. This is in line with the previous report by Ganong.[23] It has been suggested that subcutaneous histamine stimulates copious secretion of acid in rat's stomach through the H_2 receptor and the cellular mechanism involves the activation of cyclic adenosine monophosphate (cAMP) a process that is driven by H^+/K^+ ATPase.[24] Moreover, cimetidine a potent H_2 receptor blocker has been demonstrated in the present study to inhibit gastric ulceration in rats. Since H_2 receptors are located on the parietal cells, it is possible that the processed samples act on the parietal cell directly in order to stimulate gastric acid secretion and consequently induce gastric ulceration. It is also possible that the processed samples stimulates acid secretion via the H_2 histaminergic receptors, since subcutaneous administration of histamine was observed to produce a greater increase in gastric acid secretion compared with the control. Subcutaneous administration of cimetidine was observed to reduce gastric acid secretion in treated groups but to a level higher than that of control group.

Gastric ulceration arises when there is an imbalance between protective and aggressive factors. A previous report by Robert *et al.*[25] agrees with our study that an experimental substance can reduce gastric mucus and still offer cytoprotection. There are two layers of mucus, the loosely adherent and the firmly adherent mucus layers. [26] While the loosely adherent mucus layer can be easily excised, the firmly adherent layer is anchored firmly to the epithelium. Literature posits that the layer of mucus closest to the epithelium (firmly adherent mucus) is responsible for maintaining juxtamucosal pH through neutralization of inwardly permeating protons by secreted bicarbonate ions.[27] It is possible that though 15% raw and 30% roasted samples did not increase the mucus of the stomach that was actually excised for this study, it might have actually increased the layer of adherent mucus that has been implicated in cytoprotection, and hence, they were able to offer cytoprotection despite their low mucus secretion.

Results of this study showed that the mean food intake (g)/28 days of rats fed with 30% raw and 15% roasted samples of African pear were significantly ($p< 0.05$) higher compared to the control group and groups fed 15% and 30% boiled sample. The reason for the high food intake in the 30% raw sample group could be attributed to the high fat content in raw and roasted samples of African pear [15, 27]. According to Edem,[28] when moderate fats are added or present in diet, food consumption is more frequently increased than depressed and growth performance is improved. The decrease in food intake in the groups fed with 15% and 30% boiled samples when compared to both control could be due to the effect of boiling on some nutritional value of African pear, which could have reduced the level of consumption of the fruit. This observation is in line with investigations carried out by Omenna *et al.* [26], who postulated that processing of food results in loss of fats, which in turn reduces appetite. Palatability of food is an indirect measure of quality; it is the summation of the plant characteristics that determine the relish with which forage is consumed by an animal. Food processing has also been reported to affect the quality of food, especially nutrients that are heat sensitive, especially vitamins and other nutrients [27], which in turn may affect food palatability. Results of this investigation showed that all experimental animals and control animals recorded increases in body weight, which may be due to the fact that African pear is a nutritional fruit with a reasonable amount of both nutrients and phytochemicals, capable of building the body and providing energy needed by the body.[29] Though the reason behind the weight gain is not really known, however, Agiang *et al.* [15] reported that raw African pear contains adequate amount of nutrients, mostly the vitamins which are heat sensitive and needed for metabolism, thereby provided energy. This again, could have been the reason for the increase in weight of the rats fed with raw sample, as these nutrients, including carbohydrate provides energy.

5. Conclusion

Processed African pear samples stimulated acid secretion via the H_2 histaminergic receptors, since from this study, subcutaneous administration of histamine was observed to produce a greater increase in gastric acid secretion compared with control while subcutaneous administration of cimetidine was observed to reduce gastric acid secretion in treated sample groups but to a level higher than that of control group. Low consumption of boiled and roasted pulp of African pear should be avoided if possible or taken with caution by peptic ulcer diagnosed patients, while raw pulp African pear consumption should be encouraged.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

No conflict of interest.

Statement of ethical approval

The NIH guidelines for the care, handling, and use of experimental animals (NIH Publication, 1996) and compliance with arrive guidelines were judiciously followed. Moreover, the Animal Ethics Committee of the University of Calabar, Cross River State, Nigeria (approval number: 146/BCM/3019) approved the experimental protocol.

Availability of data

The datasets used/or analyzed during the current study are available from the corresponding author on reasonable request.

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Authors' Contributions

Conceptualization and design: ANI and MAA; Acquisition, analysis, and interpretation of data: ANI, JEM, EEA, BNT, UGB; Drafting, revision, and resources: ANI, MAA,EEA,BNT, EOO. All the authors read the manuscript and the authors' list and consented to the publication.

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