Gastro-protective effect of palm kernel (*Elaeis guineensis*) oil enriched diet on indomethacin induced gastric ulcer in rats

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Palm kernel oil (PKO) resembles coconut oil in preparations and both are the only sources of lauric oil available on the world market. It has been shown to be used to control diseases such as cancer, obesity, and immune disorders, as an anti-aging and antioxidant agent. The present study investigated the gastric protective property of PKO against indomethacin-induced gastric ulceration in rats. 20 male Wistar rats were divided into 4 groups of 5 each. All mice in groups 2, 3 and 4, except group 1, served as controls, were fed a daily diet of 5, 10 and 25% PKO, respectively. Rats were fed a PKO-supplemented diet for 21 days prior to ulceration with a single oral dose of indomethacin (40 mg/kg body weight) on day 22. Gastric volume, gastric acid concentration, gastric pH, bicarbonate, mean ulcer index and percentage ulcer inhibition were determined. The results showed a significant decrease (P < 0.05) in gastric juice volume, gastric acid concentration and mean ulcer index compared with mice of group 1 (control group), ants. However, a significant increase (p<0.05) was observed in gastric pH, bicarbonate, and percentage ulcer index in groups 3 and 4 rats compared to group 1 rats. These findings are indicative of the gastro-protective potentials of PKO to be capable of ameliorating indomethacin-induced gastric ulceration in rats. The probable mechanisms of action of PKO need further investigation.

Key words: Gastro-protective, NSAIDS, palm kernel oil, rats.

INTRODUCTION

Oil palm (*Elaeis guineensis*) is mainly grown in tropical regions such as Africa, Latin America, South Pacific and Southeast Asia (Atasie and Akinhanmi, 2009). Oil palm produces two different oils namely palm oil produced...
from the fibrous middle coat of the fruit and palm kernel oil extracted from the white flesh of the seeds (Norizzah et al., 2014).

Palm kernel oil is referred to as lauric oil because of its high lauric acid composition (C12, 48%), a medium-chain fat comparable to that in breast milk and with similar nutritional effects (Kabara, 1990; Pantazaris and Ahmad, 2004). It has been used to control diseases such as immune disorders, cancer, obesity, and also as an anti-aging agent, antioxidant, and in mainstream medical practice as an antidote for toxicity and wound healing (Asagba et al., 2008; Ekpa, 1995).

A gastric ulcer is a benign lesion of the mucosal epithelium during gastric exposure to excess acid and active pepsin. Positive peptic ulcers, in which the gastric mucosa is damaged and perforated, leading to bleeding, are common worldwide and affect up to 10% of the world's population (Zapata-Colindres et al., 2006).

The pathophysiology of this disease is a multifactorial process caused by an imbalance of gastric mucosa (pepsin) and gastric mucosal destructive factors (acidity) and by endogenous and exogenous causes include infection, Helicobacter pylori, acids, smoking, stress, pepsin, stress, and harmful agents such as excessive alcohol intake and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) (Syam et al., 2009).

NSAIDs such as indomethacin are an example of the major known causative factors responsible for the development of peptic ulcers. These drugs are widely prescribed mainly for pain and inflammation (Brooks et al., 1999). Long-term NSAID use has been shown to cause gastric mucosal injury mainly by decreasing prostaglandin (PG) synthesis through inhibition of the enzyme cyclooxygenase (COX) and partly through a COX-independent mechanism (Wallace, 2009).

The scientific community is increasingly interested in the production of anti-cancer drugs of plant origin that are effective, non-toxic, and inexpensive, have no side effects and are easily accessible to the public, especially in developing countries. Therefore, the present study investigated the possible gastroprotective effect of a diet rich in palm kernel oil (without guinensis) on indomethacin-induced gastric ulceration in rats.

**EXPERIMENTAL DESIGN**

Twenty (20) male Wistar rats were used for this study. The mice were 8 to 10 weeks old and weighed between 170 and 200 g. They were randomly divided into four groups of 5 mice each. Rats from each group were housed in separate cages at the Animal House, Madonna University, Nigeria, following a natural day and night cycle. Rats were fed a standard diet and given water liberally. They were given two weeks of acclimatization and then treated as follows: Group 1: control group; the rats in this group received only mouse chow. Group 2: Rats in this group were fed a 5% PKO-enriched diet. Group 3: Rats in this group received a 10% PKO-rich diet. Group 4: Rats in this group received a diet rich in 25% PKO. All animals were housed and handled according to research protocols established by the National Research Center Animal Care Committee, following the recommendations of the National Institutes of Health Guidelines on the Care of Animals and the Use of Laboratory Animals (Publication No. 85-23, revised 1985).

**Chemicals and drugs**

Indomethacin was purchased from Kapit Pharmaceutical Limited, Nigeria Diet formulation.

**Formulation of diet**

The test diet was formulated by mixing 5, 10 and 25 ml of palm kernel oil with 100 g of rat food.

**Induction of gastric ulceration**

Causing gastric ulcers After 21 days of treatment, rats were fasted for 24 h before inducing gastric ulcers by taking a single dose of indomethacin 40 mg/kg body weight.

**General determination of stomach injury**

Rats' stomachs were opened along the greater curve, rinsed with saline and stapled for gross examination. After immobilizing the tissues by immersing them in 10% formalin for 24 h, it was rinsed under running water and examined for ulcers. Ulcers were counted with a magnifying glass (X magnification). The gastric mucosa was carefully examined for the presence of ulcers and their number was counted using an illuminated magnifying glass (10x) (Zagholool et al., 2015), ulcer index (UI) scores were calculated based on an arbitrary scale according to Zhou et al. (2020).

**Determination of pH of gastric juice**

The pH of gastric juice is determined by a pH meter. The acidity of the gastric juices of each rat was assessed individually by immersing the pH indicator strip (PHS-25 pH meter; Microfield, UK) into the gastric juice immediately after gastric bypass. The results are expressed as the pH value.

**Determination of gastric juice volume**

This was performed as previously described by Heeba et al. (2009). 4 h after gastric ulcer induction, rats were anesthetized, laparotomy for gastrectomy and gastric juice extraction to determine gastric juice volume. 5 ml of distilled water was added to the gastric juice and the resulting solution was centrifuged at 3000 rpm for 10 min.

**Determination of bicarbonate in gastric juice**

HCO3− secretion was measured in the gastric cavity as previously described by Takeuchi et al. (1992).

**Determination of gastric acid secretion**

Gastric acid in mEq/L was determined in the supernatant (2 ml) by titration with 0.0025 N NaOH using Topfer reagent and phenolphthalein as indicators (Adefisayo et al., 2018). A burette was installed in the laboratory and 0.01 N NaOH was prepared and...
Table 1. Effect of palm kernel oil enriched diet on gastric juice volume, gastric acid concentration, gastric pH, bicarbonate and mean ulcer score.

<table>
<thead>
<tr>
<th>Group</th>
<th>Gastric juice volume (ml/4 h)</th>
<th>Gastric acid concentration (mEq/L)</th>
<th>Gastric pH</th>
<th>Bicarbonate (mmol/L)</th>
<th>Mean ulcer score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1: Control</td>
<td>3.80 ± 0.09</td>
<td>0.93±0.11</td>
<td>2.0 ± 0.02</td>
<td>14.94 ± 0.94</td>
<td>2.75 ± 0.25</td>
</tr>
<tr>
<td>Group 2: 5% PKO diet</td>
<td>2.50 ± 0.02</td>
<td>0.73±0.31</td>
<td>4.5 ± 0.04*</td>
<td>21.34 ± 1.34</td>
<td>2.13 ± 0.32</td>
</tr>
<tr>
<td>Group 3: 10% PKO diet</td>
<td>2.10 ±0.02*</td>
<td>0.70±0.08*</td>
<td>5.5 ± 0.03*</td>
<td>25.24 ± 1.05*</td>
<td>1.63 ± 0.24*</td>
</tr>
<tr>
<td>Group 4: 25% PKO diet</td>
<td>1.40 ±0.03*</td>
<td>0.5±0.02*</td>
<td>6.13±0.01*</td>
<td>38.19±2.45</td>
<td>0.25 ± 0.14*</td>
</tr>
</tbody>
</table>

Values expressed as Mean ± SEM; of 5 rats per group. One-way ANOVA and LSD revealed significant differences between the control and the treated groups with PKO. *P<0.05.

Source: Author

poured into the burette. 50 ml of distilled water was added to the gastric juice (liquid fraction) inside the single bottles. 25 ml of gastric juice was pipetted into a beaker and 3 drops of Tوضيحة تمت إزالة الصورة ولا يمكنني قراءة النص. 

gastric acid concentration (mEq/L) in groups 3 and 4 rats (0.70 ± 0.08) and (0.5±0.02), respectively when compared with the control group (0.93 ± 0.11).

Gastric pH showed a significant increase (p<0.05) in groups 2, 3 and 4 rats (4.5±0.04), (5.5±0.03) and (6.13±0.01), respectively when compared with the control group (2.0±0.02).

Furthermore, bicarbonate concentration (mmol/L) increased significantly (p<0.05) in groups 2, 3 and 4 rats (21.34±1.34), (25.24±1.05), and (38.19±2.45), respectively when compared with the control group (14.94±0.94).

The mean ulcer score index decreased significantly (p<0.05) in groups 3 and 4 rats (1.63±0.24) b and (0.25±0.14), respectively when compared with the control group (2.75±0.25).

As shown in Figure 1, the percentage ulcer inhibition increased significantly (p<0.05) in a dose-dependent manner in groups 2, 3, and 4 rats, respectively.

DISCUSSION

The present study determined the gastroprotective effect of a diet rich in palm kernel oil on indomethacin-induced gastric ulceration in rats. The significant increase in ulcer index, as well as gastric acid volume, following oral administration of indomethacin in untreated, ulcerated rats, can be attributed to the generation of free radicals or inhibition of synthesis prostaglandins, as reported in the work of Ajani et al. (2014), Hong et al. (2014), Inas et al. (2011), and Lichtenberger (2005). Decreased prostaglandin levels are thought to be due to decreased gastroprotection as well as increased gastric secretion, which are important events in the etiology of mucosal ulcers (Bech et al., 2000; Biplab et al., 2011). Untreated ulceration in this study is consistent with an earlier study by Vijender et al. (2012). Stomach acid is an important factor in ulcer formation in rats. However, gastric acid secretion is increased and the acid can further digest the gastric wall, leading to the formation of gastric ulcers (Goel and Bhattacharya, 1991).

The significant reduction observed in PKO-treated rats may be due to the presence of flavonoids (such as
The authors have not declared any conflict of interests.

Figure 1. Effect of PKO on % Ulcer Inhibition. Each bar is expressed as mean SEM (n=5). One way ANOVA and LSD revealed significant differences between the control and treated groups. *p<0.05.

Source: Author
REFERENCES


