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Original Research Article

Chronic Consumption of Open Market "Fresh" Palm Oil Alters Renal Handling of Na+, Cl, K+ and HCO3-

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Abstract

This study was carried out to find out the extent of oxidation and its effects of oxidation if any on the renal handling of Na^+, Cl^-, K^+ and HCO_3^- in rats fed open market purchased palm oil. Forty male wistar rats (weighing 140-160grams) were randomly distributed into three groups of ten rats each viz: control, fed normal rat chow, FPO_{mill} (fed 15% mill fresh palm oil diets), FPO_{market} (fed 15% w/w open market purhased fresh palm oil diets) and PPO (fed 15%w/w photoxidized palm oil diets) groups. Animals received water ad libitum. The experiment lasted for 12 weeks. Blood and Urine samples were collected at the end and the concentations of Na^+, Cl^-, K^+ and HCO_3^- were determined. Mean plasma concentrations of Na^+, Cl^+, and HCO_3^- of FPO_{market} and PPO groups were significantly ($P<0.001$, $P<0.001$ and $P<0.05$ respectively) lower than control and FPO_{mill} groups. Plasma potassium levels showed the reverse. The urine concentration of Na^+ and Cl^- of the FPO_{market} and PPO groups were significantly ($P<0.001$) higher than the control and FPO_{mill} groups; The urine concentrations of K^+ in the control and FPO_{mill} groups were significantly ($P<0.0$) higher than the FPOmarket and PPO groups. The urine bicarbonate levels of the FPO_{market} were significantly ($P<0.05$) lower than that of FPO_{mill}. The urine output of the FPO_{market} and PPO groups were significantly ($P<0.001$) lower than control and FPO_{mill} groups. In conclusion, open market "fresh" palm oil has undergone some oxidation and is detremental to health. Its consumption should therefore be discouraged.

Keywords: Fresh palm oil, photoxidation, hyponatremia, hyperkalemia, renal function parameters, urine output.

INTRODUCTION

Palm oil, extracted from the mesocarp of palm fruit tree *Elaesis guinneensis,* originates from Africa and has now spread throughout most of the world [1].

It is the most widely produced vegetable oil, and is consumed as cooking oil (fresh and thermally oxidized) in most parts of Africa, especially Nigeria [2-4]. Apart from being used as an ingredient in a variety of products, it is also used in the making of margarine and shortening [1]. Food producers choose palm oil because it has a distinct quality, requires little or no hydrogenation and prolongs the shelf-life of different products [1].

The nutritional and health attributes of palm oil have been well documented especially when eaten in its fresh form. Fresh palm oil is the largest natural source of tocotrienol which is the most potent form of vitamin E [1]. The combination of tocotrienols, carotenes and other antioxidants makes palm oil a super anti oxidant [5]. Apart from acting as an antioxidant, tocotrienol strengthens the immune system, protects the skin cells from toxins and ultra violet radiation [6]. In a study by [7], tocotrienols were shown to strengthen the heart so that it can better withstand stress. They showed that animals in which a heart attack had been induced had an increased survival rate, minimized injury generation rate as well as a potential to recover faster when administered fresh palm oil. Tocotrienols, being highly potent antioxidants have demonstrated remarkable anti cancer properties, far superior to most other antioxidants. They do not only prevent cancer from taking hold, but actively block its growth and initiate apoptosis, a process whereby diseased cells commit suicide or, simply put, programmed cell death [5]. Ordinarily vitamin E does not induce programmed cell death (apoptosis) in cancer cells; only tocotrienols have this effect. This research has therefore been so impressive that cancer researchers have called tocotrienols the most powerful natural anticancer agent known to science [5].

Fresh palm oil is also rich in carotenoids. In fact it is known as the richest natural source of carotenoids (about 15 times more than carrots and 300 times more than tomatoes) [1]. The human body uses carotenoids as vitamin A. They can also enhance the immune system function by a number of mechanisms, and can increase cardiovascular health. They also play an important potential role by acting as antioxidants, protecting cells and tissues from the damaging effects of free radicals [1].

When palm oil is being used in the oxidized state, it posses potential dangers to the physiological and biochemical functions of the body [4]. Oxidized palm oil produces an adverse effect on the plasma lipid profile, free fatty acids phospholipids and cerebrosides [4, 1]. Available evidence suggests that at least part of the oxidized palm oil impact on health is due to the generation of toxicants (free radicals) due to oxidation. Free radicals cause the generation of reactive oxygen species (ROS) in the system, which are very detrimental to health. Recent studies have also shown that consumption of oxidized unsaturated fatty acids may contribute to the pathogenesis of atherosclerosis by increasing lipid per oxidation and total cholesterol levels [8]. Additionally, oxidized palm oil induces reproductive toxicity and organ toxicity particularly of the kidneys [9, 4].

Studies have shown that exposure to sunlight leads to deterioration [10]. Therefore the palm oil we purchase from the market which is erroneously referred to as "fresh" palm oil may have suffered thesame fate. How far are we sure that the palm oil we purchase from the market is actually fresh? If it is not fresh, to what extent has the palm oil we purchase randomly from the open market been oxidized?

The kidneys are the major organs responsible for the filtration and excretion of waste products and toxins from the blood. Apart from this, they are also responsible for the balancing of electrolyte levels, pH regulation, releasing hormones, helping to control blood pressure, helping to produce red blood cells (erythropoiesis) and producing vitamin D which keeps the bones strong and healthy. However, this organ is very susceptible to oxidative stress which results from an imbalance of reactive oxygen species (ROS) and defense mechanisms, which result in cell damage [11]. In addition, the presence of inflammation is a well documented factor influencing the development of oxidative stress in dialysis patients [12]. ROS may affect cells of the host organism especially at sites of inflammation in addition to playing a role in the defense system against other agents. This effect plays a role in a variety of renal diseases such as glomerulonephritis and tubulointerstitial nephritis which can contribute to proteinuria and other conditions [13, 14]. ROS are also known to contribute to the pathogenesis of ischemic reperfusion injury in the kidney [15].

Our study therefore aims at determining the extent of oxidation of market palm oil by comparing its effects on renal handling of some electrolytes (Na^+, Cl^-, K^+, and HCO_3^-) in rats fed with three diets viz: mill purchased palm oil (FPO_{mill}) market purchased palm oil (FPO_{market}), and photoxidized palm oil (PPO).

MATERIALS AND METHODS

Experimental Animals

Forty Wistar albino rats each weighing between 140–160 g at the beginning of the experiment were randomly assigned to four groups of ten rats each, respectively: Control (fed normal rat chow), FPO_{mill}, fed 15%w/w mill purchased fresh palm oil diets), FPO_{market} (fed 15% w/w market purchased fresh palm oil diet) and photoxidized palm oil group (PPO; fed 15% w/w photoxidized palm oil diet).

The palm oil diets were prepared according to Osim *et al*., [3] and Owu *et al*., [4]. Briefly, 85 grams of rat chow was mixed with fifteen grams of fresh or photoxidized palm oil, which is the usual composition of a typical Black African diet. The oxidized forms of palm oil (PPO) were prepared using the methods of Raza *et al*., [30]. The peroxidation number of PPO was 5.16. The peroxide value was determined using the standard AOCS method [31].

All animals were fed their separate diets for 12 weeks. Free access to water was allowed for each animal. They were each kept in separate metabolic cages throughout the duration of the experiment.

Note that the fresh palm oil purchased from the mill was stored in a dark container throughout the duration of the experiment and the diets were freshly prepared daily. The research was conducted in accordance with internationally accepted principles for care and use of animals as recommended in the declaration of Helsinki [32].

Experimental Procedure

After anaesthesizing the animal intraperitoneally with a mixture of 1% (w/v) alpha chloralose and 25% (w/v) urethane in normal saline at a dose of 5ml/kg body weight, the right femoral vein was cannulated for the infusion of inulin and para-amino hippuric acid. A priming dose of inulin (16mg/kg body weight) and para-amino hippuric acid (PAH) (8mg/kg body weight) were given to the animals with a syringe, after which the cannula was connected to the infusion pump (11plus, Havard apparatus, Holliston MA, USA) for the infusion of sterile normal saline solution containing inulin (36mg/ml) and para-amino hippuric acid (PAH) (5.8mg/ml) [16, 17] at a rate of 0.06ml/min.

Through a small lower abdominal incision, the urinary bladder was cannulated with a short retaining catheter (pp100, polythene tubing). The urethra was ligated to prevent voiding of urine. After the equilibration period (a period within which three consecutive 20min urine collections yielded constant or the same volume) of 60mins, urine samples were collected in preweighed vials for another 60min period. The urine samples were thereafter stored in a freezer until when required. Terminal blood samples were also collected from the left femoral vein into heparinized tubes and blood plasma was immediately separated by centrifugation (3000 x g for 10 mins). Separated plasma was put in Eppendorf tubes and stored in the freezer until when needed.

Statistical Analysis

The results were expressed as mean ± standard errorof mean (SEM). The results were analysed using graphPad prism software version 5(GraphPad Software, SanDiego, CA). One-way analysis of variance (ANOVA) was used to compare means followed by a post hoc Bonferroni test where *p* values were significant. $p = 0.05$ was considered significant.

RESULTS AND DISCUSSION

RESULTS

Table-1: Comparison of The Mean Concentation of Electrolytes: Na+, K+, Cl- and HCO3 - In the Plasma of Control, Rats and Rats Fed FPOmill, FPOmarket and PPO Diets

Parameter	Control	FPO_{mill}	FPO_{market}	PPO
Na^+ **(Meq/L)**	142.36±1.42	141.26± 1.30^{NS}	$136.71±1.25^{***,r}$	$135.60±0.32^{***,r,s}$
Cl^- **(Meq/L)**	109.63±1.31	$103.16±1.23^{NS}$	$93.15±1.36^{***,C}$	$90.15±3.72^{***,C,S}$
K^+ **(Meq/L)**	3.50±1.23	$3.43.02±0.58^{NS}$	$5.81± 0.60^{*,d}$	$5.41±0.31^{*,d,s}$
HCO^-_3 **(Meq/L)**	24.36± 0.42	$24.24±0.63^{NS}$	$23.33±.41^{NS,d}$	$23.83±0.51^{NS,d,s}$

KEY:

NS; *; *** = not significant; P<0.05; P<0.001 vs control
d; c; r = P<0.05; P<0.01; P<0.001 vs FPO_{mill}
S = not significant vs FPO_{market}

Table-1 shows the comparison of the mean concentrations of Na^+, Cl^-, K^+ and HCO_3^- in the plasma of control, FPO_{mill}-; FPO_{market}- and PPO- diet fed rats.

The sodium and chloride levels in the plasma of FPO_{market} and PPO- diet fed animals were significantly (P<0.001) lower than those of the control and FPO_{mill}. When the sodium and chloride concentrations of the FPO_{market} and PPO groups were compared with each other, there was however no significant difference.

The potassium concentrations in the plasma of FPO_{market} and PPO groups were significantly (P<0.01) higher than that of the control and FPO_{mill} groups. There was also no significant difference when the mean concentrations of FPO_{market} and PPO were compared with each other.

When the mean bicarbonate ion concentrations in the plasma of FPO_{market} and PPO were compared with those of the control and FPO_{mill}, there was a significant (P<0.05) decrease. There was no significant difference when the mean plasma potassium concentrations of FPO_{market} and PPO were compared with each other.

Table-2: Comparison of The Concentation of Electrolytes Na^+, K^+, Cl^- and HCO_3^- In The Urine of Control, And Rats Fed FPO_{mill}, FPO_{market}, PPO and TPO Diets

Parameter	Control	FPO_{mill}	FPO_{market}	PPO
Na^+ **(Meq/L)**	27.23±0.15	$27.17±0.67^{NS}$	$33.32±0.13^{***,r}$	$32.15±0.66^{***,r,s}$
Cl^- **(Meq/L)**	20.21±0.26	$20.61±0.32^{NS}$	$32.11±1.12^{***,r}$	$34.25±0.13^{***,r,s}$
K+ **(Meq/L)**	35.00±1.15	$34.28±0.79^{NS}$	$27.13±1.25^{**,c}$	$26.17±1.31^{**,r,s}$
HCO^-_3 **(Meq/L)**	13.12±0.51	$14.23±0.21^{NS}$	$10.13±0.31^{NS,d}$	$10.34±0.85^{NS,s}$

KEY

NS; **; *** = not significant, p<0.01; p<0.001 vs control.
c; r; d = p<0.01 and p<0.001 vs FPO_{mill}
S = not significant vs FPO_{market}

Table-2 shows the comparison of the mean urine concentrations of Na^+, Cl^-, K^+ and HCO_3^- in the control, FPO_{mill}, FPO_{market} and PPO groups.

Mean concentrations of Na^+ and Cl^- in the urine of FPO_{market} and PPO groups were significantly

(P<0.001) higher than those of the control and FPO_{mill} groups. There was no significant difference in the mean concentration of these electrolytes when the FPO_{market} and PPO groups were compared with each other.

K^+ levels in the FPOmarket and PPO groups were significantly (P<0.01; 0.001 respectively) lower than the control and FPO_{mill} groups. No significant difference was found when the mean urine concentration of K^+ in the FPOmarket and PPO groups were compared with each other.

The mean urine bicarbonate concentration of FPO_{market} animals was significantly (P<0.05) lower than the FPO_{mill} diet fed group. However, there was no significant difference in the mean urine concentration of bicarbonate when the control, FPO_{mill} and PPO groups were compared with each other.

Table-3: The Comparison Of The Urine Output Between The Control, FPO_{mill}, FPO_{market} And PPO Groups

Parameter	Control	FPO_{mill}	FPO_{market}	PPO
H_2O output (µl/min)	4.43±0.02	4.40 ± 0.15^{NS}	$2.25\pm0.01^{***,r}$	$2.31\pm0.03^{***,r,s}$

KEY:

NS, *** = not significant and P<0.001 vs control
r = P<0.001 vs FPO_{mill}
S = not significant vs FPO_{market}

Table-3 shows the comparison of the urine output betwen the control, FPO_{mill}, FPO_{market} and PPO groups.

The mean urine output of the FPO_{market} and PPO groups were significantly (P<0.001) different from the control and FPO_{mill} groups. However, when the mean urine output of the FPOmarket and PPO were compared, there was no significant difference.

DISCUSSION

This study was done to compare the renal handling of some electrolytes (Na^+, Cl^-, K^+ and HCO_3^- by rats fed with "fresh" palm oil bought from the open market and those fed with Fresh palm oil purchased directly from the mill (protected from sun light or light from any source) with that of rats fed with photoxidized palm oil.

The results showed that mean concentrations of Na^+ and Cl^- of the control and FPO_{mill} were significanlty higher than those of the FPO_{market} and PPO groups. However, these values (mean concentration of Na^+ and Cl-) were not significantly different from the each other when the control and FPO_{mill} groups were compared. These results are very similar to the results we got in a previous study [18], where the concentration of Na^+ and Cl^- in the plasma of animals fed with oxidised palm oil were significantly lower than those of fresh palm oil fed animals and control animals. The animals in the FPO_{market} and PPO groups are hyponatemic in this study just like the animals fed oxidesed palm oil in our previous study [18]. The concentration of Na^+ and Cl^- in the urine of FPO_{market} and PPO were also high compared with the control and FPO_{mill} groups. Previous studies have shown that chronic consumption of oxidized palm oil results in the destruction of the hemopoeitic system [19], liver, and kidneys [3] and reduce glomerular filtation as well as renal blood flow [20]. Also [21] stated that acute kidney injury may also lead to a reduction of urine Na^+ concentration. In this study therefore, the reduction in urine concentration of Na^+ and Cl^- may have been as a result of an acute kidney injury.

Potassium was significantly (P<0.01) higher in the plasma of the FPO_{market} and PPO- diet fed animal groups when compared with the control and FPO_{mill} groups. However, the levels of potassium in the urine of the FPOmarket and PPO groups was lower than those of the control and FPO_{mill} groups. Under normal circumstances, potassium levels in plasma are lower than the urine levels [22]. This is as a result of the fact that it is secreted at the distal convoluted tubule of the kidneys [22]. In this study, however, the urine concentration is lower in the FPO_{market} and PPO groups compared with the control and FPO_{mill} groups. Chronic consumtion of oxidized oils may lead to an increase in the concentration of reactive oxygen species [10] which may also lead to the destruction of tissues [23]. The disruption in the K^+ balance seen in this study may be either as a result of an acute kidney injury leading to destruction of the aldosterone receptors on the distal convoluted tubule [24] or an injury of the adrenal glands, leading to a lower concenration of aldosterone. All of the above could lead to impaired renal excretion of potassium [25].

The bicarbonate levels in the plasma of FPO_{market} and PPO were significantly lower than that of FPO_{mill} and control groups. This picture is in line with our previous study [18]. Therefore, this reduction in bicarbonate ion concentration in the plasma of the above mentioned groups may be as a result of the hyponatremia explained above [26]. Also, adrenocortical insufficiency may lead to an accumulation of hydrogen ions in circulation [27]. Bicarbonate ions are usually responsible for the buffering of this acidity. The cosumption of bicarbonate ions as a result of th above mentioned buffering, may

account for the reduction of bicarbonate ion concentration in the FPO_{market} and PPO groups in this study.

The urine out put by FPO_{market}- and PPO- diet fed animals was reduced compared with that of control and FPO_{mill} –diet fed animals. Normally, an increase in the excretion of Na^+ should be accompanied with increased water excretion [28]. However, the reverse is the case in this study. This is indicative of a kidney disease and is in line with what other studies have shown that low urine flow rate is indicative of a kidney disease [29]. Also, Adam *et al.*, [8] reported that chronic consumption of thermally oxidized palm oil could lead to the generation of ROS. These species are known to cause oxidative stress in high concentrations to which the kidneys are highly susceptible [3, 11]. As a result of oxidative stress a chronic kidney disease may develop, which could be responsible for the results we have in this study.

The renal handling of electrolytes (Na^+, Cl^-, K^+ and HCO_3^-) by the control and FPO_{mill}-diet fed animals was the same.

The renal handling of electrolytes (Na^+, Cl^-, K^+ and HCO_3^-) by the FPO_{market}- and PPO-diet fed animals was also similar.

The renal handling of electrolytes by FPO_{market}- and PPO-diet fed animals was significantly different from that of control and FPO_{mill}-diet fed groups.

CONCLUSION

Consumption of "fresh" palm oil purchased from the open market adversely affects the ability of the kidneys to regulate electrolytes in the same way photoxidized palm oil does. Therefore, open market palmoil has undergone photoxidation and its consumption should be discouraged.

If fresh palm oil must be sold in the open market, it must be sold in completely dark containers to prevent photoxidation.

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