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Research article

Comparative Effects of *Cocos Nucifera* Oil and Ketogenic Diet on Obese Sprague Dawley Rats

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ABSTRACT

This study evaluated the effects of formulated ketogenic diet (KG) and coconut (hot and cold pressed) oils on obese Sprague Dawley rats. Thirty rats weighing between 180-200g were divided into six groups of five rats each. Group 1 was fed with the standard rat chow (normal control) while groups 2 to 6 were fed with high fat diet (HFD) for 4 weeks to induce obesity. Thereafter, the obese animals were treated for another 2 weeks on the following formulated diets: group 2 was maintained on HFD only, groups 3 to 6 were placed on cold-pressed coconut (CPC) oil diet (HFD+CPC), hot-pressed coconut (HPC) oil diet (HFD+HPC), KG diet (HFD+KG) and orlistat (FATNIL®) (20mg/kg), an established anti-obesity agent respectively. After the expiration of 4 weeks, there were significant ($p < 0.05$) increase in body weight, body mass index (BMI) and adiposity index (AI). Also, there was increase in low density lipoprotein (LDL), total cholesterol (TC) and triacylglycerol (TAG) levels in the HFD fed groups. However, after 2 weeks of intervention treatment, there was significant ($p < 0.05$) reduction in the body weights, BMI, AI, HDL cholesterol and TC levels of the rats in the HFD+CPC, HFD+KG and orlistat treated groups. The CPC oil, KG and orlistat diet treated groups showed marked decrease in serum transaminases activities, catalase activity, superoxide dismutase (SOD) activity and hepatic malondialdehyde (MDA) level. This study revealed that CPC oil and short term consumption of KG diet demonstrated a lipid-lowering effect and reduction in indicators of obesity.

Keywords: *ketogenic diet, obesity, malondialdehyde, orlastst, cholesterol.*

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INTRODUCTION

Obesity and overweight are characterized by excessive fat accumulation usually caused by disproportionate caloric intake and calories spent (Zhao et al., 2005). Both obesity and overweight are no longer non-communicable diseases (NCDs) of just the developed world but as a new trend that can be observed where low- and middle-income populations are facing high double-burden of infectious and chronic diseases (WHO, 2021). The risk of dying of NCDs is greatest when in combination with infectious diseases that weaken the immune system such as human immunodeficiency virus (HIV), tuberculosis, hepatitis or parasitic infections such as malaria (WHO, 2021).

An increase in consumption of diets rich in fats, carbohydrate, processed foods, increased sedentary lifestyles as well as decreased consumption of fruits and vegetables haves resulted in high incidence of obesity. Additionally, genetics, cultural beliefs and urbanization also play major role (WHO, 2017). Obesity increases the risks of type 2 diabetes, high blood pressure, cardiovascular diseases (Gerbaix et al., 2012). The double burden of disease that is now being experienced in most low- and middle-income countries may

be associated with inadequate prenatal and young children nutrition while being exposed to high-fatty, sugary, salty and energy-dense diets lacking in micronutrient which are usually lower in cost (WHO, 2017). These dietary habits in combination with lower physical activity may explain the increase in childhood obesity while under nutrition remains present in the population.

To highlight the magnitude of this situation, according to WHO report in 2016, 650 million adults are obese where obesity and overweight are linked to more deaths globally than underweight (WHO, 2017). In Nigeria, the prevalence of overweight and obesity are tending towards epidemic proportions with prevalence of overweight being up to 35% in 2013 (Chukwuonye et al., 2013). To curtail this trend, numerous diets and super foods are being introduced with claims of health benefits and weight loss.

A ketogenic diet is one rich in fat with adequate amount of protein and low in carbohydrates. Studies have shown that this diet is beneficial against diabetes, epilepsy, cancer and even Alzheimer's disease (Zhou et al., 2001; Hemingway et al., 2007; Gasior and Hartman, 2007; Westman et al., 2008). Other studies have shown that a ketogenic diet helps in weight loss (Foster and Hill, 2003; Yancy et al., 2004). Coconut oil has been shown to have the potential to protect against not only heart disease but a wide variety of chronic health problems including diabetes and cancer as well as a means to prevent and even treat infectious diseases (Fife, 2006). Coconut is widely grown and consumed in Nigeria. In this study a ketogenic diet and coconut oils are compared to a reference weight loss drug, Orlistat (FATNIL®).

MATERIALS AND METHODS

Reagents and Chemicals: All the reagents used for this study were of analytical grade and were obtained from Sigma Chemicals Co, London and Aldrich Chemical Company, USA. The commercial kits used were HDL cholesterol, total cholesterol, alanine amino transferase (ALT), aspartate amino transferase (AST), urea, creatinine, and triacylglycerol (Randox Laboratories Limited, UK). The reference anti-obesity drug Orlistat (FATNIL®) was obtained from Micro Laboratory Limited, India

Collection and Identification of Coconut (*Cocos nucifera*): Matured coconuts (*Cocos nucifera*) were obtained from New Benin market in Edo State, Nigeria. Samples of the coconut were thereafter identified in the Department of Plant Biology and Biotechnology, University of Benin, Nigeria.

Animal Model and Handling: Thirty male rats of the Sprague-Dawley strain were obtained from the animal house, Biochemistry Department, Faculty of Life Science, University of Lagos, Nigeria. The rats were allowed to acclimatize to their new environment for two weeks with free access to standard rat chow and water before the experiment commenced.

Ethical Approval: Ethical approval was received from University of Benin Research Ethics Committee for the use of experimental rats for this study. The experimental animals were handled in accordance with international standard guidelines of the Canadian Council on Animal Care Guidelines and Protocol Review.

Preparation of Coconut Oil Sample: The coconut meat (white part of the coconut) was blended with an industrial blender (3000W by S&L) in 200cm3 of distilled water to obtain a mixture of coconut chaff and milk. To prepare the cold pressed coconut oil, the mixture was first separated using a muslin cloth and thereafter, the oil was extracted from the coconut milk by cold extraction method. The coconut milk was kept in the refrigerator (4ºC) and left for 24 hours. The resulting mixture separates into two phases; a top layer of thick emulsion and an aqueous layer underneath. Thereafter, the top thick layer was carefully removed and placed in a heating pan and allowed to heat at low temperature 60ºC. The resulting oil was allowed to cool and filtered again to remove any debris. The oil was then used as source of fat to formulate diet-rich coconut feed. The hot pressed oil was also prepared after separating the mixture as previously described, the oil was extracted from the coconut milk (supernatant obtained) by heating at 100oC for 60 min until the water completely evaporated. The oil was then used as source of fat to formulate diet-rich coconut feed (Agarwal *et al*. 2013).

Preparation of Experimental Diets: Three different diets were administered to the experimental rats in the respective groups consisting of high fat (HF), ketogenic (KG) and normal diets. The macronutrient composition of the diets is presented in Table 1. The HF (high fat) and ketogenic diets were formulated and compounded based on a combination of described by Murphy *et al*. (2005) and Gerbaix *et al*. (2012). The components of the diets are shown in Table 2. Diets were prepared weekly to guarantee palatability and extend shelf life.

Table 1:
Macronutrient Composition of Diets

Macronutrients	High fat diet	ketogenic diet	Normal diet
Carbohydrates	31.4%	11.4%	59.0%
Proteins	18.6%	18.6%	18.6%
Fats	50.0%	70.0%	22.4%

(Murphy et al., 2005; Gerbaix et al., 2012)

Experimental Design and Feeding: The experimental animals were divided into six (6) groups of five (5) rats each. Diets were formulated for the different groups of rats; group 1 was fed standard diet while Groups 2-6 were fed high fat diet (HFD; Tables 1 and 2). The rats were fed once daily on these diets for a period of four (4) weeks. After 4 weeks, cold pressed coconut oil (CPC = HFD+CPC), hot pressed coconut (HPC = HFD+HPC) oil, and ketogenic diets (KG = HFD+KG) were added to the diets in Groups 3-5 the animals in Group 6 were maintained on a HFD and were administered the anti-obesity drug, orlistat (20mg/kg; p.o.) orally for 2 weeks. Food and water spillage were minimized to the barest minimum. Their cages were cleaned daily throughout the duration of the experiment.

Table 2:
Composition of the Experimental Feed

	Macronuttrients	High fat diet	Ketogenic diet	Coconut oil Diet	Normal diet
Carbohydrates (g)	Corn flour	377	161	200	585
	Coconut flour	-	-	177	-
Proteins (g)	fish meal	223	263	223	181.4
Fats (g)	Mangarine	200	139	80	40
	Soya oil	67	300	-	53g
	Coconut oil	-	-	187	-
Others	Vitamin	12	12	12	15
	Mineral	58	63	58	64
	Fiber	62	62	62	62
Total (g)		1000	1000	1000	1000
Total (kcal)		4800	5650	4800	3900

Biochemical Assays: Anthropometric parameters (body weights, body mass index, and adiposity index) were evaluated as described by Lategan *et al*. (2014); David *et al*. (2020). Alanine amino transferase (ALT) and aspartate amino transferase (AST) enzymes activities were determined using commercially available kits. The method described by Reitman and Frankel (1957) was employed. Bilirubin was determined using Radox kit and described by Jendrassik and Grof (1938). Oxidative stress indices of the liver and kidneys were measured using marker of lipid peroxidation (malondialdehyde), superoxide dismutase (SOD) and catalase activities. Malondialdehyde (MDA), indices of lipid peroxidation was assayed for by the method described by Varshney and Kale (1990). SOD activity was determined by the method of Misra and Fridovich (1972) and catalase activity by the method of Cohen *et al* (1970). Total cholesterol concentration was determined using the method described by Allain *et al* (1974), high density lipoprotein cholesterol (HDL-Chol) and triacylglycerol concentration by method described by Tiez (1990; 1999). The low density lipoprotein cholesterol (LDL-Chol) and very low density lipoprotein cholesterol (VLDL-C) concentrations were estimated by calculation using the formulae by Friedewald *et al*. (1972) as shown below:

LDL-C = Total Cholesterol - (HDL-C +Triglycerides/5)
VLDL-C = Triglycerides/5

Statistical Analysis: Statistical analysis of the data obtained was done using SPSS (Statistical package for social sciences) version 15.0. Data were expressed as mean ± SEM (standard error of mean). One way analysis of variance (ANOVA) was used to determine the existence of statistical significance between variables. Turkey HSD post-hoc test was employed to check the level of significance at $p < 0.05$.

RESULTS

Effect of Coconut Oils and Ketogenic Diet on Some Anthropometric Parameters in Sprague Dawley Rats fed High Fat Diet: The effects of diets formulated with coconut oil [cold (CPC) and hot pressed (HPC)] and ketogenic (KG) diets were evaluated on some anthropometric parameters (body weights, body mass index, and adiposity index) in Sprague Dawley rats fed with high fat diet (Figures 1 and 2). Figure 1 shows the changes in the body weights of rats fed on the high fat diet (HFD) for 4 weeks and thereafter supplemented with coconut oils (cold or hot pressed), ketogenic diets and the anti-obesity drug, orlistat (20mg/kg) for another 2 weeks. At week four (4), there was significant ($p < 0.05$) gain in body weights of all the groups of rats fed on the HFD in comparison with the normal diet fed rats (normal control) who were fed on the standard diet only.

At week 6, rats on the HFD (Group 2; negative control) without treatment or diet supplementation continued to gain body weights which was evident in their percentage weight gain, adiposity index and body mass index (BMI) when compared with the normal control (Figure 2). But supplementation of the HFD with the ketogenic, coconut oil (cold or hot pressed) diets or administration of orlistat resulted in significant ($p < 0.05$) reduction in body weight in comparison to the negative control. Animals in group 3 (HFD + CPC oil) had the highest percentage weight loss compared to the other groups placed on the new diet regimen as against the HFD group which maintained a constant body weight gain throughout the course of the study.

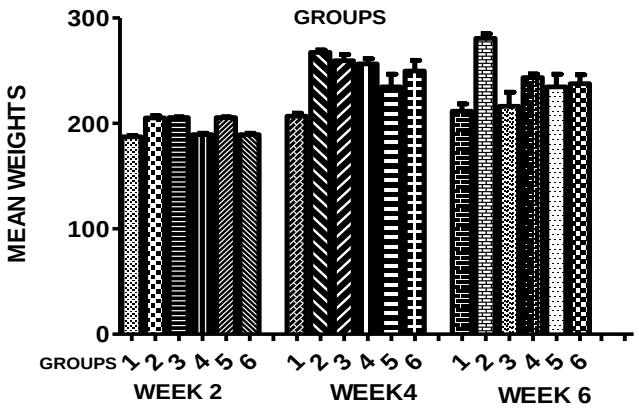

Figure 1:
Effects of KG Diet, CPC and HPC Oil Diets on Mean Body Weights of HFD fed Sprague Dawley Rats
Values are expressed as mean± SEM, *n* = 5 / group. 1 = Standard diet only; 2 = High fat diet (HFD) only; 3 = HFD + cold pressed coconut oil; 4 = HFD + hot pressed coconut oil; 5 = HFD + ketogenic diet; 6 = HFD + anti-obesity drug, orlistat®

Effects of CPC, HPC Oils and KG Diets on Lipid Profile of Sprague Dawley Rats fed on HFD: Tables 3 and 4 represent the lipid profile of HFD fed Sprague Dawley rats treated with CPC, HPC, KG diets and the anti-obesity drug, orlistat at 4 and 6 weeks, respectively. At week 4, there was

significant ($p < 0.05$) elevation of LDL cholesterol, triacylglycerols and total cholesterol levels in the HFD fed groups of rats compared to the normal control (Table 3). At week 6, total cholesterol, triacylglycerols and HDL cholesterol levels were reduced (Table 4) in all the HFD rats supplemented with the other diets (CPC, HPC and KG) or reference drug (orlistat) in the following order: Group 6 (HFD + orlistat) > Group 5 (HFD + Keto diet) > Group 3 (HFD + CPC) > Group 4 (HFD + HPC) as against the negative control.

Effect of Coconut oils and Ketogenic Diet on Liver Function indices of HFD fed Sprague Dawley Rats: The effects of the different treatments and diets on liver function enzymes of Sprague Dawley rats fed on a HFD are presented in Table 5. There were significant ($p < 0.05$) reductions in the activities of serum ALT and AST in the HFD groups that were fed on the KG, coconut oil (hot and cold pressed) diets and the anti-obesity drug, orlistat compared with the negative control. Although this decrease was not significantly ($p > 0.05$) different from each other, the values increased towards the normal control. Total bilirubin concentration increased in a non-significant ($p > 0.05$) manner in all the groups studied when compared to the normal control.

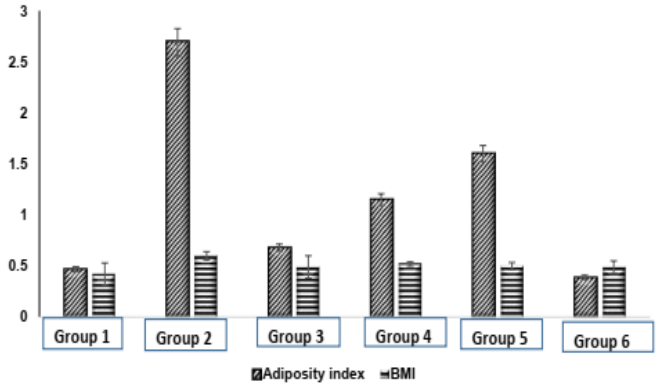

Figure 2:
Effects of KG diet, CPC oil and HPC oil diet on Body Mass Index and Adiposity Index in Sprague Dawley Rats fed on HFD
Values are expressed as mean ± SEM, *n* = 3 / group. Different lowercase letters represent significant difference between means at $p < 0.05$. 1 = Standard diet only; 2 = High fat diet (HFD) only; 3 = HFD + cold pressed coconut oil; 4 = HFD + hot pressed coconut oil; 5 = HFD + ketogenic diet; 6 = HFD + anti-obesity drug, orlistat®

Table 3:
Effects of KG Diet, CPC and HPC Oil Diets on Lipid Profile of Sprague Dawley Rats Fed on HFD at week 4

Groups	Total Cholesterol (mg/dL)	Triacylglycerols (mg/dL)	HDL (mg/dL)	LDL (mg/dL)
Normal Control	116.67±9.24 [a]	105.91±6.52[a]	53.33 ±5 .45[a]	42.15±6.99[a]
HFD	174.24±6.41[b]	146.6±2.27[b]	71.15± 5.23[b]	73.77±3.14[b]
HFD+ CPC	193.59 ± 10.00[c]	149.48 ± 5.74[c]	58.04 ± .91[a]	105.65 ± 11.48[c]
1HFD +HPC	186.25± 7.83[b]	183.35± 6.22[d]	54.60± 4.66[a]	94.97±3.61[d]
HFD + KG	202.21 ± 4.85[c]	155.94 ± 10.26[c]	78.91 ± 2.2c	92.13 ± 3.54[e]
HFD + orlistat	167.07 ±4.73[b]	139.4± 9.70[b]	73.81±6.04[b]	65.38±6.79[b]

Values are expressed as mean ± SEM (n = 5). Means with different superscripts differ significantly at $p < 0.05$. Normal control = Standard diet only, HFD = High fat diet only, HFD + CPC = High fat diet + cold pressed coconut oil, HFD + HPC = High fat diet + hot pressed coconut oil, HFD + KG = High fat diet + ketogenic diet, HFD + orlistat = High fat diet + anti-obesity drug, orlistat®

Table 4:
Effects of KG Diet, CPC and HPC Oil Diets on Lipid Profile of Sprague Dawley Rats Fed on HFD at Week 6

Groups	Total Cholesterol (mg/dL)	Triacylglycerol (mg/dL)	HDL (mg/dL)	LDL(mg/dL)
Normal control	117.03 ± 4.14[a]	100.57 ± 5.08[a]	63.4 ± 2.90[a]	33.49 ± 5.94[a]
HFD	245.15 ± 10.8[b]	201.15 ± 13.01[b]	46.22 ± 2.8[b]	158.5 ± 11.45[b]
HFD+ CPC	169.67 ± 4.86[c]	110.58 ± 1.69[c]	63.29 ± 3.74[a]	84.26 ± 6.99[c]
HFD +HPC	178.78 ± 5.78[d]	147.21 ± 4.23[d]	68.97 ± 10.5[a]	63.63 ± 1.48[c]
HFD + KG	161.20 ± 5.61[c]	142.98 ± 1.69[a]	63.29 ± 3.74[a]	84.26 ± 6.99[c]
HFD+ orlistat	136.71 ± 6.02[f]	106.11 ± 6.09f	59.55 ± 6.25[b]	55.94 ± 2.16[f]

Values are expressed as mean ± SEM (n = 5). Means with different superscripts differ significantly at $p < 0.05$. Normal control = Standard diet only, HFD = High fat diet only, HFD + CPC = High fat diet + cold pressed coconut oil, HFD + HPC = High fat diet + hot pressed coconut oil, HFD + KG = High fat diet + ketogenic diet, HFD + orlistat = High fat diet + anti-obesity drug, orlistat®.

Table 5:
Effects of KG Diet, CPC and HPC Oil Diets on some Liver Function indices of Sprague Dawley Rats fed on HFD

Groups	AST (U/L)	ALT (U/L)	Total Bilirubin (mg/dL)
Normal control	25.37 ± 4.25[a]	15.13 ± 3.07 [a]	2.85 ± 0.42[a]
HFD	46.08 ± 8.31[b]	21.70 ± 2.70[b]	3.67 ± 0.47[b]
HFD + CPC	33.19 ± 1.19[c]	17.51 ± 0.88 [a]	3.01 ± 0.12[b]
HFD + HPC	34.01 ± 1.39[c]	18.95 ± 0.57 [a]	3.44 ± 0.27[b]
HFD + KG	35.91 ± 3.02[c]	19.82 ± 1.42 [a]	3.32 ± 0.05[b]
HFD + orlistat	36.56 ± 2.21[c]	10.43 ± 0.24[a]	3.03 ± 0.03[b]

Values are expressed as mean ± SEM (n = 5). Means with different superscripts differ significantly at $p < 0.05$. Normal control = Standard diet only, HFD = High fat diet only, HFD + CPC = High fat diet + cold pressed coconut oil, HFD + HPC = High fat diet + hot pressed coconut oil, HFD + KG = High fat diet + ketogenic diet, HFD + orlistat = High fat diet + anti-obesity drug, orlistat®

Table 6:
Effects of KGD, CPC and HPC Oil Diets on Liver Malondialdehyde, Superoxide Dismutase and Catalase activities of Sprague Dawley Rats fed on HFD

Groups	MDA (U/mg protein x 10^{-2})	CAT (U/mg protein)	SOD (U/mgprotein)
Normal control	4.25 ± 0.56^{a}	0.39 ± 0.131^{a}	0.13 ± 0.013^{a}
HFD	6.25 ± 0.09^{b}	0.15 ± 0.050^{b}	0.10 ± 0.012^{b}
HFD + CPC	3.50 ± 0.13^{c}	0.049 ± 0.025^{c}	0.18 ± 0.012^{c}
HFD + HPC	5.23 ± 1.41^{c}	0.046 ± 0.016^{c}	0.19 ± 0.036^{c}
HFD + KGD	4.94 ± 0.15a	0.046 ± 0.003^{c}	0.19 ± 0.034^{c}
HFD + orlistat	3.05 ± 0.19^{c}	0.29 ± 0.004^{a}	0.12 ± 0.022^{a}

Values are expressed as mean ± SEM (n = 5). Means with different superscripts differ significantly at p < 0.05. Normal control = Standard diet only, HFD = High fat diet only, HFD + CPC = High fat diet + cold pressed coconut oil, HFD + HPC = High fat diet + hot pressed coconut oil, HFD + KGD = High fat diet + ketogenic diet, HFD + orlistat = High fat diet + anti-obesity drug, orlistat®

Effect of CPC oil, HPC oil and KG Diets on some Oxidative Stress indices of HFD fed Sprague Dawley Rats: Liver Catalase (CAT) and superoxide dismutase (SOD) activities were significantly ($p < 0.05$) lower in the rats that were fed on HFD only in contrast to the rats in the normal control and other treated groups (Table 6). Whereas, the malondialdehyde (MDA) level in the liver was significantly increased in the HFD fed rats when compared to the groups treated with ketogenic diet, coconut oil and orlistat. However, HPC oil rich diet fed rats and the KG diet gave a higher MDA levels when compared to the CPC oil rich diet fed rats (group 3). The group that received Orlistat and those fed on standard diet showed lowest activities of CAT and SOD.

DISCUSSION

Obesity is a non-communicable disease that is a risk factor to numerous comorbidities and deleterious health disorders. Although, obesity is influenced by genetics the current epidemic of this disorder appears to be driven principally by sedentary lifestyle factors which include high-energy diets with little or no physical activity (Blakemore and Froguel, 2008; Sharon, 2017). These immensely contribute to the energy imbalance that leads to overweight and obesity.
A high fat diet combined with a moderately high carbohydrate diet can be used to cause obesity and other metabolic disorders including diabetes and cardiovascular disease in animals (Crescenzo *et al*., 2015).

It is documented that weight gain and increase in anthropometric parameters are markers of obesity (Angeloco, 2012). This was affirmed in this study with the demonstration of increase in body mass index (BMI) and adiposity index (AI) as indicators of obesity. A short term dietary induction of obesity using high fat diet was also reported by Crescenzo *et al* (2014). They also reported an observed decrease in oxidative capacity of obese rats' liver resulting in enhanced oxidative stress.

Dietary interventions are one of the lifestyle methods that have been used to effectively control obesity. Appropriately tailored diet regimens for weight reduction have been shown to be effective for the management of obesity. One diet regimen that could prove to be very effective for rapid weight loss is a combination of very-low-carbohydrate and high-fat diet often referred to as ketogenic diet (Wheless, 2006). This agrees favourably with the observed reduction in body weight and other anthropometric parameters such as the AI and BMI of rats given the ketogenic diet in this study. In recent years, coconut oil (Coccus nuccifera) has been documented as a potential source of saturated fats in foods. It is highly publicized by health specialists that the fat contained in coconut oil is capable of promoting good health which includes weight loss, lower cholesterol, prevent the risk of cardiovascular diseases and also prevent bone loss, dental caries, dermatitis and inflammation etc (Wallac, 2019; Siong *et al*., 2020).

In this study, cold pressed coconut (CPC) oil supplemented diet was able to reverse weight gained by rats fed on the high fat diet. CPC has been reported to possess a lowering effect on triglycerides, cholesterol and LDL when compared to hot pressed coconut oil rich diet (HPC) (Asia Pacific Coconut Community, 2015; Siong *et al*., 2020 Guturu and Duchini, 2012). Studies have reported that coconut oil has a lipid lowering effect, oils rich in polyunsaturated fatty acids, present in coconut oil promote the reduce blood cholesterol in patients with atherosclerosis (Teitelbaum & Walker, 2001). The levels of low density lipoprotein (LDL) appeared to be relatively higher in individuals consuming safflower oil or butter as compared to those consuming coconut oil (Feranil *et al*. 2011). Nevertheless, orlistat and ketogenic diet showed the highest potentials of lowering cholesterol and HDL-cholesterol levels. This may be possible especially with the fact that orlistat is a lipid lowering drug, which acts by inhibiting cholesterol synthesis through inhibition of HMG-CoA reductase enzyme, a rate limiting enzyme for cholesterol synthesis; hence there is no effect on triglyceride concentration.

Ketogenic diets are known to induce satiety in animals due to the high fat content. It was first reported by Robert Atkins that ketogenic diet was essential to weight loss (Katz, 2003). Ketogenic diet inhibits lipogenesis (lipid synthesis) and induces lipolysis. In a nutshell, low-carbohydrate diets (ketogenic diet) work by producing ketosis. Ketosis is a metabolic condition in which the rate of ketone bodies synthesis is higher than the rate of their metabolism (Voet *et al*., 2008). With minimal carbohydrate intake, there is little glucose to convert to glycogen. Without glycogen stores, body metabolism adjusts predominantly to lipolysis and excretion of lipids, leaving ketones behind in the blood. Ketosis has a significant influence on suppressing hunger. Thus, ketogenic diet may be a good regulator of the body's calorie intake and mimics the effect of starvation in the body. In other words, it's believed that ketogenic diet can place the body in a state of

ketosis by elevating the production of D-β-hydroxybutyrate and acetoacetate and acetone which is usually expelled through the lungs (Dashti *et al*., 2004). Another mechanism may also be through the improvement in the activities of the hunger hormones leptin and ghrelin (Mawer, 2017).

Coconut oil have been reported to temporarily increase metabolic rate and just like ketogenic diet, it may speed up the rate of lipolysis, that is the rate by which fatty acids are been released from the breakdown of fats (Zimmermann *et al*., 2004). When coconut oil is broken down, it produces a higher amount of ketone bodies compared to longer chain fatty acids. Research has shown evidence of this mechanism as the reason why coconut oil causes muscle preserving effect during caloric restriction in obese people (Zimmermann *et al*., 2004). lipid-lowering effect, weight loss and reduction in other obesogenic indices observed in the HFD fed rats that was later treated with coconut oil diets was evident in this study. Humans burn more calories when the fats they consume are replaced with medium chain fats. Moreover, medium chain triglycerides have been reported to boost metabolism by increasing energy expenditure by 120 calories per day (Zimmermann *et al*., 2004). The varied effects of CPC and HPC coconut oils in the HFD rats as noticed in this study could be ascribed to the mode of preparation of the two types of coconut oil; which makes one (CPC) retain more chemical components and the other (HPC) lost most of the nutritional components through heating.

The liver is an essential metabolic organ of the body that stores and allows the proper distribution of fuel and maintains the body's homeostasis. An obesogenic diet like that described in this study can compromise the liver's functionality due to its association with hyperlipidaemia and obesity (MacManaman *et al*., 2013). This was indicated in this study by the marked increase in the liver function enzymes (AST and ALT) in the HFD fed rats as against the rats exposed to the diets/drugs which showed close to normal activities of these enzymes. Evidences of oxidative stress were noticed in the rats fed on HFD as opposed to the CPC, HPC, KG and orlistat treated rats. Hepatic MDA levels, reduced SOD and CAT activities are indicators of a compromised hepatic oxidative status which also points to suspected abnormal functioning liver.

High fat diet can induce oxidative stress by significantly increasing lipid peroxidation that enhances the activities of reactive oxygen species and free radicals. Once the antioxidant status of the body is compromised, it either fails to recruit antioxidant enzymes such as superoxide dismutase, catalase etc, or produce lesser amount to mop up the system (MacManaman *et al*., 2013). As a result of these, the oxidative species overwhelms the body's antioxidant capacity and oxidative stress ensues. However, supplementation of the HFD with the different diet regimen ameliorated the damaging effect of HFD.

CPC oil and short term consumption of KG diet demonstrated a lipid-lowering effect, reduction in indicators of obesity, ameliorative effect on hepatic functions and oxidative stress status in the experimental animals. In this study, cold-press coconut oil diet merits further consideration as an edible oil diet with potential therapeutic application in obesity compared to other ketogenic diets. Therefore, these diet regimens could be employed in effective management of weight gain and obesity.

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