Effect of Coconut Oil Administration on Some Hemostatic Changes Associated with Obesity in Rats

Fatma Ahmed Mohamed, Nehal Mohammad Bahgat, Gehane M. Hamed, and Rania S.A. Eisa *
Physiology Department, Faculty of Medicine, Ain Shams University

Abstract
In the last few decades, coconut oil was claimed to have some beneficial health effects, attributed mainly to its medium chain triglycerides. It was, thus, intriguing to investigate the potential benefit of coconut oil in alleviating the prothrombotic tendency often encountered in obese individuals. The present study was carried out on 44 rats, of both sexes, aged 10 days at the start of the study. 31 out of 44 rats were offered high caloric diet (the cafeteria diet) for induction of obesity. Rats were allocated into the following groups: Group 1: Control rats (C) (n=13 rats), comprising rats fed on the standard chow diet all-over the study period (24 weeks). Group 2: Cafeteria diet-fed rats (Caf) (n=16 rats), comprising rats fed on cafeteria diet until the end of the study period and Group 3: Cafeteria diet/coconut oil-fed rats (Caf/Coco) (n=15 rats), comprising rats fed on cafeteria diet with coconut oil starting from the 16th week till the end of the study period. At the end of the study, the BMI was assessed in the 3 studied groups and blood samples were collected for determination of platelet count and aggregation, prothrombin time (PT), activated partial thromboplastin time (APTT), fibrin degradation products (FDPs), and plasma lipid profile. The encountered results revealed that the mean BMI of the cafeteria diet-fed rats was significantly higher than the BMI of control rats, and that the mean BMI of rats receiving cafeteria/coconut oil diet for 9 weeks was significantly decreased compared to their matched caf group. The PT, APTT and platelet count were all non significantly different in the three studied groups. Platelet aggregation, on the other hand, was significantly increased in the caf group compared to the control group, and significantly decreased in the caf/coco group compared to both the caf group and the control group. The plasma FDPs levels were not significantly different in the 3 studied groups. The lipid profile was insignificantly different in the 3 studied groups except in the caf/coco group which revealed a significant elevation of total cholesterol and HDL-c compared to caf group. The present findings, thus, point to the possible beneficial effect of coconut oil feeding on obesity - induced enhanced platelet aggregation.

{ * From M.Sc. Thesis, Physiology department }

Introduction
The prevalence of obesity is increasing worldwide, although the proportion varies from country to country and between geographical areas within a country (WHO, 1998). Obesity is known to predispose to a number of cardiovascular risk factors, including hypertension, elevated cholesterol and impaired glucose tolerance (WHO report, 2000). Also, Obese individuals are known to be susceptible to thrombotic diseases, though the underlying mechanism is still unknown (Yamamoto et al., 2005). In many areas of Sri Lanka, the coconut tree and its products have for centuries been an integral part of life, and it has come to be called the "Tree of life". However, in the last few decades, the relationship between coconut fats and health has been the subject of much debate. Around 92% of these fats are saturated fats, which led to the belief that coconut fats are 'bad for health', particularly in relation to ischemic heart disease. Yet, since most of the saturated fats in coconut oil are medium
chain fatty acids whose properties and metabolism are different from long chain triglycerides (LCT), they are now believed to be not as 'bad for health' as other saturated fats (Amarasiri and Dissanayake, 2006). In fact, the medium-chain fatty acids and monoglycerides found primarily in coconut oil were found to have miraculous healing power (Kabara, 2000). The author added that it is rare in the history of medicine to find substances that have such useful properties and still be without toxicity or even harmful side effects.

A link between coconut oil and obesity has been postulated. Thampan (1994) stated that any health condition is made worse if the metabolic rate is slower than normal because cells cannot heal and repair themselves as quickly. The author added that increasing metabolic rate, therefore, provides an increased degree of protection from both degenerative and infectious illnesses. Medium chain fats present in coconut oil were found to stimulate thermogenesis to a greater degree than does excess energy intake as LCT (Binnert et al., 1998). Further, excess energy derived from medium chain triglycerides (MCT) was reported to be stored with a lesser efficiency than is excess energy derived from dietary LCT (Hill et al., 1989 and Noguchi et al., 2002), resulting in greater loss of adipose tissue. Thus, MCT might be considered as agents that aid the prevention of obesity or potentially stimulating weight loss (St-Onge et al., 2003).

It was, thus, intriguing to investigate the effect of coconut oil feeding on the hemostatic changes associated with obesity, aiming at alleviating the prothrombotic tendency often encountered in obese individuals.

Materials and Methods:

Animals:

The present study was carried out on 44 rats, of both sexes, aged 10 days at the start of the study (Rodríguez et al., 2001). Rats were purchased from the Institute of Ophthalmology in Giza, and maintained in the Physiology Department animal house under standard conditions of boarding. NB: Rats were separated into male and female cages at the age of 40 days to prevent mating.

Experimental protocol:

Rats included in the present study were divided into 2 main categories: control rats and rats rendered obese by being fed high caloric diet (the cafeteria diet). All animals received standard rat chow in the first week for acclimatization to the new facilities, the cafeteria diet being started on the second week.

Rats were allocated into the following groups:

- **Group1:** Control rats (C) (n=13 rats), comprising rats fed on the standard chow diet all-over the study period (24 weeks).
- **Group2:** Cafeteria diet-fed rats (Caf) (n=16 rats), comprising rats fed on cafeteria diet, for induction of obesity till the end of the study period.
- **Group3:** Cafeteria diet/coconut oil-fed rats (Caf/Coco) (n=15 rats), comprising rats fed on cafeteria diet mixed with coconut oil (cafeteria/ coconut oil diet) till the end of the study period.

The diet formulae supplied to the three studied groups were as follows:

A) Standard chow diet

All rats in the period of acclimatization were fed on the formula AIN-93G (Reeves et al., 1993). Control rats were maintained on this formula till the age of 3 months. From the age of 3 months till the end of the study, control rats were fed on the formula AIN-93M (Reeves et al., 1993) which provided 18% of energy as protein, 76% of energy as carbohydrate, and 6% of energy as lipids, by dry weight.
B) The cafeteria diet

This diet was composed of the following items: patè, chips, chocolate, bacon, biscuits and standard chow diet, in a proportion of 2:1:1:1:1:1 (Berraondo et al., 2000). The 6 food items were crushed to a powder form and mixed thoroughly. The powder was then placed in a container and given to the animals.

C) The modified cafeteria diet with coconut oil

This diet consisted of a mixture of cafeteria diet and coconut oil (purchased from Morgan factory) in a ratio of 75 gm cafeteria diet and 25gm coconut oil (Zulet et al., 1999). This diet was supplied to rats in group 3 (Caf/Coco) for 9 weeks.

The three food formulae adopted in the present study were analysed in the National Nutrition Institute (NNI), according to AOAC 2003, to confirm their conformity to the cited diet characteristics. The energy provided by each diet formula, and by each of its different components, was found to be as follows:

<table>
<thead>
<tr>
<th>Diet</th>
<th>Energy / 100 gm diet</th>
<th>% of energy as CHO</th>
<th>% of energy as protein</th>
<th>% of energy as fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control diet</td>
<td>418.98</td>
<td>71.19</td>
<td>15.28</td>
<td>13.53</td>
</tr>
<tr>
<td>Cafeteria diet</td>
<td>571.54</td>
<td>32.76</td>
<td>8.19</td>
<td>59.05</td>
</tr>
<tr>
<td>Cafeteria/Coconut oil diet</td>
<td>642.1</td>
<td>24.44</td>
<td>5.34</td>
<td>70.22</td>
</tr>
</tbody>
</table>

The served amount of food was 10gm/rat at the start, and was increased gradually over the whole study period, reaching 25 gm/rat at the end of the study. Every day, the amount of food remaining from the previous day was weighed to monitor the daily food consumption.

The body weight of rats in the different groups was assessed weekly to monitor the progress in weight gain of each group.

Experimental Procedures:

On the day of sacrifice, overnight fasted rats were weighed and anesthetized by intraperitoneal injection of thiopental sodium (40 mg/Kg, b.w.), then the body height was measured. A midline abdominal incision was made. The abdominal aorta was exposed and cannulated with a catheter and three blood samples were collected into 3 plastic tubes, one of which contained 3.2% trisodium citrate (9 volumes of blood + 1 volume citrate), the 2nd tube contained a drop of heparin, and the 3rd tube contained EDTA.

The citrated blood sample was centrifuged at 1000 rpm for 5 min., platelet rich plasma (PRP) was pipetted off, and kept in a plastic tube for assessment of platelet aggregation. The rest of the sample was centrifuged at 3000 rpm for 15 min. for separation of platelet poor plasma (PPP). A part of the PPP was used in the assessment of platelet aggregation as described by Born (1962) and O’Brien (1963), and the rest kept in a plastic tube at room temperature for performance of the following coagulation tests (within 4-6 hours) :

1-Prothrombin time (PT), as described by Neofotistos et al. (1998), using kits (Neoplastine CI Plus) supplied by Diagnostica Stago.
2-Activated partial thromboplastin time (APTT), as described by Contant et al. (1983), using kits (C.K.Prest) supplied by Diagnostica Stago.
3-An aliquot of the PPP was stored frozen for determination of fibrin degradation products (FDPs), as described by Mirshahi et al. (1986), using kits supplied by Diagnostica Stago.
The heparinized blood sample was centrifuged at 3000 rpm for 15 min. and the separated plasma was used for determination of plasma levels of:
1-Triglycerides, as described by Fossati and Prencipe (1982), using kits supplied by Biolabo SA.
2-Total cholesterol, as described by Allian et al. (1974), using kits (CHOD-PAP) supplied by Greiner Diagnostic Gmbh.
3-HDL-C, as described by Lopes-Virella (1977), using kits supplied by Greiner Diagnostic Gmbh.
4-LDL-C, was calculated as follows: LDL-C = (TC) – (HDL-C + TG/5) (Friedewald et al., 1972).

The blood sample taken on EDTA was used for determination of platelet count, using coulter T- 660, depending upon electronic counting, according to the method described by Coulter (1956).

The body mass index (BMI) was calculated as follows: BMI = body weight in gm / square height in cm². The height of rats was measured as the distance (in cm) between the nose and the anus, using an ordinary ruler. The body weight (in gm) was determined by the use of the ordinary animal scale.

Statistical analysis
All statistical data and significance tests were performed by using SPSS (Statistical Program for Social Science) statistical package (SPSS Inc) version 10.0 (Armitage P and Berry G, 1987). Statistical significance was determined by one-way ANOVA (analysis of variance) for differences between means of different groups; further analysis was made by LSD (least significance difference) multiple-range test to find intergroupal differences; a probability of P< 0.05 was considered statistically significant. Chi-square test was used for comparison of qualitative variables.

RESULTS:
Changes in body weight and BMI in the different studied groups:

Figure (1) shows the progressive increase in body weight in the three studied groups, assessed weekly, throughout the study period up to the 8th week, the mean body weight of the cafeteria diet-fed rats was significantly lower than control rats. Starting from the 11th week to the 15th week, the mean body weight of cafeteria diet-fed rats was higher compared to the control rats.

After the 15th week, cafeteria diet-fed rats were subdivided into cafeteria diet fed group (caf) and cafeteria/coconut oil diet fed group (caf/coco). Mean body weights of the three studied groups were comparable from 16th to the 21st week. Then, mean body weight of the caf/coconut group was significantly lower than control and caf groups at the 23th week and from caf group at the 22th and 24th weeks.

As regards the BMI, which was calculated at the end of the study period, it was found that the mean BMI of the cafeteria diet-fed rats was significantly higher than the mean BMI of control rats (P<0.02). On the other hand, the mean BMI of caf/coconut oil fed rats was significantly decreased compared to their matched cafeteria diet-fed rats (P<0.01). However, it was insignificantly different from control group (table 1 and figure 2).
Fig. (1) The mean body weights (gm), assessed weekly, of control, cafeteria diet-fed and cafeteria/coconut oil diet-fed rats throughout the period of the study.

Fig. (2): Changes in BMI (gm/cm²) in Control (C), Cafeteria diet-fed (Caf) and Cafeteria/Coconut oil diet-fed (Caf/coco) groups.

Changes in the Hemostatic parameters:

Prothrombin time (PT), partial thromboplastin time (PTT) and platelet count were all non-significantly different in the three studied groups. Table (1); Figure (3).

Platelet aggregation, on the other hand, was significantly increased in the cafeteria diet-fed group compared to the control group (P<0.05), and significantly decreased in the cafeteria/coconut oil diet-fed group compared to the cafeteria diet-fed group (P<0.001) as well as the control group (P<0.01) (table 1 and figures 4 & 5).
As regard the plasma fibrin degradation products (FDPs), although the levels were not significantly different in the 3 studied groups, yet the number of observations of the higher levels (>5<20 and ≥20 µg/ml) was greater in the cafeteria diet-fed and cafeteria/coconut oil diet-fed groups compared to the control group (table 2).

**Changes in Plasma Lipid Profile**

Total cholesterol and HDL were significantly elevated in caf/coco oil fed rats compared to cafeteria diet fed rats (P < 0.01 & P<0.05 respectively). However, plasma TG and LDL-C levels were comparable in the three studied groups (table 1).

**Table 1: Body mass index (BMI, gm/cm²), Prothrombin time (PT, sec), Partial thromboplastin time (PTT, sec), Platelet number (Pl. no., x10⁹/mm³), Platelet aggregation (Pl. agg, %), Triglycerides (TG, mg/dl), Total Cholesterol (TC, mg/dl), High density lipoproteins cholesterol (HDL-C, mg/dl) and Low density lipoproteins cholesterol (LDL-C, mg/dl) in the 3 studied groups.**

<table>
<thead>
<tr>
<th>Group</th>
<th>BMI (gm/cm²)</th>
<th>PT (sec.)</th>
<th>PTT (sec.)</th>
<th>Pl. No. (x10⁹/Mm³)</th>
<th>Pl. Agg. (%)</th>
<th>TGs (mg/dl)</th>
<th>TC (mg/dl)</th>
<th>HDL-C (mg/dl)</th>
<th>LDL-C (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C (n=13)</td>
<td>0.54 ±0.02</td>
<td>17.85 ±1.68</td>
<td>19.62 ±1.43</td>
<td>740.45 ±37.64</td>
<td>64.89 ±2.03</td>
<td>57.16 ±4.51</td>
<td>75.7 ±3.5</td>
<td>40.55 ±3.63</td>
<td>23.61 ±2.28</td>
</tr>
<tr>
<td>Caf (n=16)</td>
<td>0.63 ±0.03</td>
<td>16.63 ±1.28</td>
<td>20.94 ±1.31</td>
<td>622.38 ±75.88</td>
<td>72.77 ±2.79</td>
<td>50.93 ±4.10</td>
<td>67.95 ±2.4</td>
<td>36.64 ±3.11</td>
<td>23.0 ±3.4</td>
</tr>
<tr>
<td>Caf/Coco (n=15)</td>
<td>0.52 ±0.02</td>
<td>15.93 ±0.72</td>
<td>18.07 ±1.42</td>
<td>711.93 ±68.19</td>
<td>53.57 ±2.47</td>
<td>54.80 ±6.33</td>
<td>83.3 ±2.7</td>
<td>46.54 ±2.83</td>
<td>24.1 ±3.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>C (n=13)</th>
<th>Caf (n=16)</th>
<th>Caf/Coco (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>P1</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>P2</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>P3</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

P1 : Significance of difference from control rats, calculated by LSD at P < 0.05 for unpaired data
P2 : Significance of difference from untreated cafeteria diet fed rats calculated by LSD at P < 0.05 for unpaired data
P3: Significance by 1-way ANOVA among the 3 studied groups
NS : No significant difference

**Table (2): Plasma FDPs in the 3 studied groups**

<table>
<thead>
<tr>
<th>Group</th>
<th>Numb. Of rats</th>
<th>&lt;5 µg/ml</th>
<th>5-20 µg/ml</th>
<th>&gt;20 µg/ml</th>
<th>P1</th>
<th>P2</th>
<th>P3</th>
</tr>
</thead>
<tbody>
<tr>
<td>C (n=13)</td>
<td>9</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caf (n=16)</td>
<td>7</td>
<td>5</td>
<td>4</td>
<td>NS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caf/Coco (n=15)</td>
<td>6</td>
<td>3</td>
<td>6</td>
<td>NS</td>
<td>NS</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

P1: Significance of difference compared to C group, calculated by chi square test.
P2: Significance of difference compared to Caf group, calculated by chi square test.
P3: Significance of difference between the 3 groups, calculated by chi square test.
NS : No significant difference

**Fig (3): Mean changes in Prothrombin time (PT, sec.) and Partial thromboplastin time (PTT, sec.), Platelet count (plat co, /1000) in Control (C), Cafeteria diet-fed (Caf) and Cafeteria/coconut oil diet-fed (Caf/Coco) rats.**

**Fig.(4): Mean changes in platelet aggregation (%) in Control (C), Cafeteria diet-fed (Caf) and Cafeteria/coconut oil diet-fed (Caf/Coco) rats.**

\[ a = \text{significant difference from the control group, calculated by LSD at } P < 0.05 \text{ for unpaired data.} \]
\[ b = \text{significant difference from the cafeteria/coconut oil diet-fed group, calculated by LSD at } P < 0.05 \text{ for unpaired data.} \]
Fig.(5): Platelet aggregation tracings of control (a), cafeteria diet-fed (b), and cafeteria/coconut oil diet-fed rats (c)
Discussion

The present study was carried out on a rat model of obesity, started in early life. Obesity was induced by feeding suckling rats a high caloric diet (the cafeteria diet). This diet was reported to be a palatable hypercaloric and hyperlipidic diet that can induce voluntary hyperphagia and fast body weight gain (Lowell et al., 2000 and Rodriguez et al., 2001b).

It is worth-mentioning that the increase in body weight of the cafeteria diet–fed rats (Caf) showed slowness at the beginning compared to the control group, the mean body weight being lower than that of the control rats. Thereafter, there was a catch up period from the ninth week on, where the weight gain of the cafeteria diet–fed rats became faster, and the mean body weight became greater than the mean body weight of the control group till the end of the experimental period. The initially observed slower weight gain in the cafeteria diet–fed rats may be explained by reduced food intake observed at the beginning of the period of the study. This initial decrease in food intake could have resulted from one or more of the many causes reported to contribute to decreased food intake, e.g., diet palatability, high energy content of the diet (Menaker and Navia, 1973), low protein content of the diet (Rothwell and Stock, 1988), and the high fat content of the diet, which has been reported to stimulate the release of cholecystokinin which decreases feeding mainly by activation of the melanocortin pathway of the hypothalamus (Guyton & Hall, 2006). The lack of marked weight gain in the cafeteria diet-fed rats is likely to be due to diet-induced thermogenesis (DIT), reported to be caused by increased fat content of the diet (Matsuda et al., 1997), or increased energy content of the diet (Rothwell and Stock, 1986), both of which are present in cafeteria diet. Rothwell and Stock (1982) explained cafeteria-diet induced thermogenesis by hypertrophy and hyperplasia of brown adipose tissue. Later on, Christoffolete and Moriscot (2004) explained DIT in rats fed cafeteria diet by increased total brown fat mitochondria, uncoupling protein percentage and total brown fat uncoupling protein.

Although weight gain of the cafeteria diet-fed rats was not marked, yet there was significant increase in BMI in this group, compared to the control group, indicating increased body adiposity (Guyton & Hall, 2006). The higher adiposity with cafeteria diet feeding was, also, reported by Rodrigue et al. (2004) and Matute et al. (2007). Increased adiposity in response to cafeteria diet feeding was reported to be related more to an increase in the amount of visceral fat rather than an increase in subcutaneous depot (Rodrique et al., 2004). The significant increase in BMI in the cafeteria diet-fed rats, without parallel marked overweight could be explained by reduced rate of increase in body length, observed in this group compared to the control group, resulting in a higher ratio between body weight and body length. The reduced rate of body length increase could be a manifestation of reduced growth hormone (GH) secretion in cafeteria-diet fed rats. Decreased GH secretion in cafeteria-diet fed rats was reported by DeSchepper et al. (1998) as well as by Zhou et al. (1998). The later demonstrated lower GH release from normal pituitary cells incubated in serum from overfed rats than after incubation with serum from non obese rats.

Addition of coconut oil to the cafeteria diet resulted in significant decrease in body weight of rats after 9 weeks, despite the high energy content of the diet. BMI was also significantly decreased, becoming even lower than the control value. Food intake of rats in this group was observed to be comparable to that of the cafeteria diet fed rats and control rats, which make increased energy expenditure induced by coconut oil feeding in these rats is the most likely explanation of the significant decrease in their body weights and BMI.

Weight lowering effect of coconut oil has been reported in previous literature
supply fat to fat cells or

to weight gain (Crozier et al.,

subsequently countered in the present study,

as fatty acid oxidation (St-Onge et al., 2003),

significantly enhanced in the caf group

compared to the control group, and was

significantly enhanced in the caf group

and BMI (Tsuji et al., 2001).

As regards the hemostatic mechanisms, the changes in both PT and

APTT in the caf and caf/coco groups were

statistically non-significant compared to the

control group. However, though platelet

count was not significantly different in the 3

studied groups, yet platelet aggregation was

significantly enhanced in the caf group

compared to the control group, and was

significantly decreased in the Caf/coco group

compared to both the control and the

caf groups. Plasma FDPs level was

comparable in the 3 studied groups.

The significant enhancement in

platelet aggregation observed in the

cafeteria diet-fed rats, despite insignificant change in platelet count point to altered

platelet function. The impact of obesity on

platelet aggregation has long been

recognized in both human and animal

studies. Sonhee et al. (2004) reported that

platelet aggregation was enhanced and

tended to have shorter lag time in obese

males compared to non obese males. Also,

Anfossi et al. (2004) found that central

obesity induced platelet resistance to the

antiaggregating effects of prostacyclin and

NO, due to impaired cyclic nucleotide

synthesis and action; the main effectors of

platelet antiaggregation. The authors added

that this accounts for platelet hyperactivity

in obesity. Also, platelets from obese

individuals were found to express leptin

receptors, which mediated enhanced

platelet aggregation to ADP after pretreatment with leptin (Corsonello et al., 2002).

Platelet aggregation was found to be

significantly decreased in coconut/cafeteria

diet -fed rats compared to cafeteria diet-fed

rats as well as control rats. This favorable

effect could be due to decreased body

adiposity and decreased leptin level which

was reported to be involved in increased

platelet activation in obese individuals. This

finding disagree with the findings of

Podbielski et al. (1989) and Pronczuk et al.

(1991) who reported increased platelet

factor 4 and platelet activation with coconut

oil feeding.

Concerning the plasma lipid profile, the

evacuated results disagree with of

earlier reports of Pagliassotti et al. (1996)

and Anurag and Anuradha (2002) that high

calorie diet- feeding and obesity result in
dyslipidemia,. Lack of dyslipidemia in the

present study may be explained by the early

start of the high calorie diet (3rd week of

rats’ lives). Serisier et al. (2008) reported

that younger animals were better able to

balance energy needs with energy

consumption and added that young animals

didn’t exhibit significant changes in

triglycerides and free fatty acid

concentrations compared to older animals.

When coconut oil was added to the

diet, a significant increase in total

cholesterol and HDL-cholesterol, compared

to the caf group, was observed. These

findings disagree with those of Kaunitz and

Dayrit (1992), and Kasai et al. (2003), who

reported hypocholesterolemic effect of

coconut oil. This difference could be due to

addition of coconut oil to cafeteria diet in

the present study, whereas in other studies,

coconut oil was administrated with standard

rat chow. The present findings, however,

agree with those of Pronczuk et al. (1991),

who reported increased total cholesterol

upon coconut oil feeding. Fortunately the

results encountered in the present study,
demonstrated that the increase in plasma cholesterol was mainly in the beneficial HDL-C fraction rather than the LDL-C fraction which agree with Nevin and Rajamohan (2004).

It could, thus, be concluded that administration of hypercaloric hypolipidemic diet early in life resulted in increased body adiposity with reduced rate of increase in body length, and altered platelet function leading to enhanced platelet aggregation. Coconut oil supplementation in diet induced significant decrease in body weight and adiposity, as well as platelet aggregation. These favorable effects were obtained after 9 weeks of starting coconut oil supplementation despite continued cafeteria diet feeding. These findings make coconut oil feeding a recommended tool of weight lowering to avoid the suffering of long term dietary restriction. It could also help in alleviating the platelet dysfunctions encountered in obese individuals.

Acknowledgment: The authors acknowledge the valuable advice and kind assistance of Dr. Ghada ZAS, National Nutrition Institute (NNI), in preparation of diet formulae used in the present study.

Abbreviations:

APTT (Activated partial thromboplastin time ), BMI (Body mass index), BW (Body weight), Caf (Cafeteria diet-fed), Cafe/coco (Cafeteria/coconut oil diet-fed), DIT (Diet induced thermogenesis), FDP (Fibrin degradation products), GH (Growth hormone), HDL (High density lipoproteins), LCT (Long chain triglycerides), LDL (Low density lipoproteins), MCT (Medium chain triglycerides), PT (Prothrombin time), TC (Total cholesterol), TG (Triglycerides)

References:

growth hormone secretion and normal plasma insulin like growth factor I concentrations. Growth Hormone & IGF Research, 8 (3): 397-401.


الملخص العربي
تأثر إعطاء زيت جوز الهند على بعض التغيرات في ميكانيكا وقف النزف المصاحية للسمنة في الفنران
فاطمة أحمد محمد - نهال محمد بهجت جميل - جيهان محمود حامد - رانيا صلاح السيد عيسى
قسم الفسيولوجى - كلية الطب - جامعة عين شمس

أجري هذا البحث لدراسة التغيرات التي تحدث في ميكانيكا وقف النزف المصاحية للسمنة. ومحاولة كشف إذا
كانت هناك استفادة من استخدام زيت جوز الهند في تقليل تحسن في هذه التغيرات.

وقد أجريت هذه الدراسة على 44 من فرنان التجارب البينية، الذين تم تقسيمهم إلى المجموعات التالية: مجموعة
الفنران الضابطة. وعدها 13 فنرًا، و تم تغذيتها بغذاء الفنران العادي طوال فترة البحث. المجموعة الثانية
مكونة من فرنان تم تغذيتها بغذاء عالى السعرات بداية من الأسبوع الثاني بعد الولادة ولمدة اربعة شهور لاحدة
سمنة بها. ثم تم تقسيمها إلى مجموعتين فرعيتين: مجموعة استمرت تغذيتها بغذاء عالى السعرات. وعدها 16 فنرًا
حتى نهاية فترة البحث. والمجموعة الأخرى. وعدها 15 فنرًا، اضيف زيت جوز الهند للغذاء عالى السعرات في
مختلفه.

وقد تم في جميع المجموعات تعبين مؤشر كتلة الجسم و أيضا زمن بروثورومين و زمن تروميبلاستين الجنسي
وجمع الصفائح الدموية ومستوى نواتج تكسير الفيبرين بالإضافة إلى تقدير مستوى دونه البلازما و عدد خلايا الدم
الخليخة.

وقد اظهرت النتائج أن الفنران التي تم تغذيتها بالغذاء عالى السعرات زاد موشرا كتلته الجسم فيها زيادة ذات دالالة
إحصائية مقارنة بالمجموعة الضابطة، بينما نقص موشرا كتلة الجسم بدرجة ذات دالالة إحصائية بعد اضافة زيت
جوز الهند إلى غذاء هذه الفنران لمدة 9 اسابيع و ذلك بالمقارنة بالمجموعة الغذائية بغذاء عالي السعرات.

وبالنسبة للتغيرات في ميكانيكا وقف النزف. لم تكن هناك تغيرات ذات دالالة إحصائية في قياسات تجلط الدم ( زمن
البرثورومين- زمن تروميبلاستين الجنسي- نواتج تكسير الفيبرين) في الفنران الغذائي عام السعرات
بضافة أو بدون اضافة زيت جوز الهند. مقارنة بالمجموعة الضابطة، بينما زاد تحميل الصفائح الدموية في الفنران
المغذي بالغذاء عالي السعرات زيادة ذات دالالة إحصائية مقارنة بالمجموعة الضابطة و قد ادى اضافة زيت جوز
الهند للغذاء الى نقص ذو دالالة إحصائية في تجمع الصفائح الدموية مقارنة بالمجموعتين الأخرىتين.

لم يحدث أي تغيير ذو دالالة إحصائية بين المجموعات الثلاثة في متوسط الدهون الثلاثية والدهون منخفضة الكثافة.
بينما زاد متوسط الكولسترول الكلي والدهون عالية الكثافة في المجموعة التي تغذى بغذاء عالي السعرات مضافا
اليها زيت جوز الهند بدرجة ذات دالالة إحصائية مقارنة بالمجموعة الغذائية بغذاء عالي السعرات.

و بهذا فإن النتائج هذه الدراسة تؤكد أن السمنة تؤثر سلبية على ميكانيكا وقف النزف بالتأثير على وظائف الصفائح
dمومية يزيدها. بينما اضافة زيت جوز الهند إلى الغذاء يقلل هذا التأثير. و بهذا قد يكون اضافة زيت جوز
الهند إلى الغذاء فائدة في تقليل الوزن الزائد و أيضا في تحسن وظائف الصفائح الدموية.