Health Impact of Traditional Egyptian Ghee"*Samna baladi*" Comparing to Plant Ghee in Experimental Rats

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ABSTRACT

The present work aimed to compare the health impact of traditional Egyptian ghee (samna baladi) EG and plant ghee (palm oil) PG from through indication fatty acid composition and biochemical effects. The studied parameters included the body weight gain of rats, blood lipid profile, kidney and liver functions and antioxidant enzymes. Twenty one Albino rats were randomly divided into three groups of 7 rats each. Control group was fed the basal diet which containing 10% corn oil, and replaced corn oil with 10% ghee(EG) and 10% plant ghee(PG) for the two experimental groups. The results indicated that when EG administered to rats along the feed experiment (6 weeks) insignificantly reduced the body weight gain, and oxidative stress and had protective effects on the liver comparing with the control group and experimental (PG) group. There was a significant increase in the level of TBARS in PG group. The results on blood lipid profiles showed marked and significant elevation in the total lipid, triglycerides, total cholesterol and low density lipoprotein (LDL) in PG group and marked increase in the level of HDL in EG group. Also antioxtidant enzymes activity in liver increased in EG group, while reduced in PG group. The study concluded that the health impact of traditional Egyptian ghee is better than plant ghee, however further research on the health effect of using Egyptian ghee in cooking are needed, to identify the effect of heat on oxidation of the fatty acids.

***Key words*: traditional ghee- plant ghee- rats- lipid profile.**

INTRODUCTION

Ghee in Egypt is called "samna baladi".It is commonly made from buffalo milk and sometimes from cow milk. The texture, color and taste of the traditional Egyptian ghee depends on the quality of milk or butter source used in the process and the duration of boiling. Ghee made from buffalo milk are white in color, while those made from cow milk is often yellowish (Aboudonia and AIagamy, 1993).

Ghee is a product that is made by indigenous methods in many countries around the world, largely in Asia (India and Pakistan), the Middle East and Africa. A recent definition of ghee is stated as a product exclusively obtained from milk, cream or butter by processes which result in almost total removal of water and non-fat solids, with an especially developed flavor and physical structure (Afsaneh *et al.*, 2016).

According to FAOSTAT (2014) the Egypt production of total butter and ghee 2012 were (124,550) Tons .Most of the Egyptians prefer to consume the "samna baladi" because of its desirable taste and distinctive smell, therefore its use is common in many Egyptian households, and it is usually used in preparation of several dishes and sweets. Since the fifties of the last century, the organizations responsible for food and nutrition have recommended the replacement of ghee by hydrogenated vegetable oils because of the seriousness of animal ghee on health of heart and arteries.

Hydrogenation improves the texture and increases oxidative stability of the products. Unfortunately, during hydrogenation process some double bonds can be isomerized and converted from *cis* state to *trans* state. *Trans* fatty acids are known to have negative effects on health and can cause different diseases (Iqbal, 2014). Palm oil is now used widely as an alternative to hydrogenated oils because of its semisolid texture at room temperature and its less content of trans fatty acids.

Ghee in spite of being a rich source of cholesterol and saturated fatty acids, the research results on the relationship between consuming ghee and human health are conflicting. For examples, Ahmadias *et al*. (2008) mentioned that the consumers should not forgot that ghee, which is traditionally made from milk fat has high amounts of saturated fatty acids and its production method should be carefully supervised. On the other hand, Rani *et al*, (2011) mentioned that because of the high conjugated linoleic acid content of milk fat (ghee), it increases anti-oxidant status and anti-atherogenic potency in experimental Wistar rats. Also, according to Chinnadurai *et al* (2013), ghee improves blood lipid profiles by increasing HDL content and reduces lipid peroxidation. Therefore, this study aims to identify the effect of traditional Egyptian ghee and plant ghee (palm oil) on some biochemical parameters of male Albino rats.

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MATERIALS AND METHODS

Materials

-Buffalo butter was prepared by housewives in the villages from Behaira Governorate.

-Corn oil was obtained from a local market, Alexandria, Egypt.

- Plant ghee (palm oil): the most famous consumed vegetable ghee was obtained from the local market, Alexandria, Egypt.

- Chemicals used for the study were purchased from El Gomhorya Company in Alexandria.

- Male Albino rats weighing 130-140 g were obtained from the Animal House of the Institute of Graduate Studies and Research, Alexandria University, Egypt.

Methods

Preparation of traditional ghee

Traditional ghee (buffalo milk fat) prepared at home according to the Egyptian traditional method.

- Four kilograms of buffalo traditional butter were heated at 120^0C until release the melted fat and drive off moisture. The product was strained to remove residues (morta).Clear liquid was then decanted in a glass jars. It contains not less than 99.5% fat and not more than 0.1% moisture (Aboudonia and AIagamy,1993).

Fatty Acid Composition

Fatty acid composition analysis of the traditional ghee and plant ghee were done at the Agriculture Research Center, Doky, Giza.

Preparation of fatty acid methyl esters: The fatty acid methyl esterswere prepared using transesterification with cold methanolic solution containing potassium hydroxide as catalyst. The fatty acid methyl esters were identified by G.C capillary column according to the methods of IOOC (2001).

Identification of fatty acid methyl ester by GLC: Agilent 6890 series GC apparatus provided with a DB-23 column (60mx 0.32 x 0.25µm). Fatty acid results after the previous procedure steps were transformed into methyl esters and directly injected into the GC.Carrier gas was $N_2$with flow rate of 2.2 ml/min, splitting ratio of 1:80.The injector temperature was 270°C. The temperature setting were as follow :130° to 210°C at 3min, and held time 10 min. at 210°C.

Animals experiment design

Twenty one Albino rats, ten weeks old and weighing 120-140g were used. The animals were housed 7 per cage (plastic cages) with sawdust bedding and maintained in an air-conditioned animal house at a controlled temperature (22 ± 2°C) and relative humidity (60 ± 10%) with a photoperiod of 12h light/12h dark (Childs *et al.,* 2002).

The animals were kept on a basal diet and tap water provided *ad libitum* for two weeks for an adaptation period before the start of the experiment. Then the rats were given the basal diet (Table1).

Table1. Basal diet composition(1)

Diet ingredient	%
Skim milk powder	37.5
Corn starch	29
Sucrose	13.5
Fat source	10
Sawdust powder	5
*Mineral mixture	4
**Vitamin mixture	1

(1)Source: Walter, (1981)

*Salt mixture from: Assiutcunnilingus molds for the production of salt and mineral feed additives.

**Vitamin mixture :from MuvcoMedical Professions for Veterinary Products& Fodders Additions Co.

Each group of rats was fed the basal diet containing one of the research fats, as follows:

control: the rats were fed the basal diet containing corn oil..

Group1: the rats were fed the basal diet containing traditional ghee (samna).

Group2: the rats were fed the basal diet containing plant ghee.

The experimental duration was 6 weeks.

Hematological Parameters

The blood samples were collected in two tubes: one containing EDTA (anti-coagulant) and the other containing Heparin (anti-coagulant). Non-coagulated blood by EDTA was tested shortly after collection by particle counter (from ERMA INC.-Tokyo. Model PCE-210) for measuring total red blood cells (RBC), white blood cell count (WBC), platelet count (PLT), hemoglobin (Hb), hematocrit (Ht).

Blood biochemical parameters and enzyme activities

The other part of heparinized blood samples were placed immediately on ice. Plasma was obtained by centrifugation of samples at 860 gx for 20 min, and was stored at -80°C until used for analyses. Stored plasma samples were analyzed for total, urea, creatinine (Walters and Gerade, 1970).

Plasma samples were analyzed for total lipids, cholesterol and triglycerides (TG) according to the methods of (Zollner and Kirsch, 1962; Bucolo and

David, 1973; Fossati and Prencipe,1982), respectively. High-density lipoprotein-cholesterol (HDL-c) was determined according to the methods of (Grove, 1979, and Burstein *et al*, 1980). Low-density lipoprotein-cholesterol (LDL-c) was determined by calculation (cholesterol-(TG/5+HDL). All previous tested parameters were determined using commercial kits from Bio-systems S.A. (Spain), Diamond (Germany) and Randox (United Kingdom).

The method of Reitman and Frankel, (1957) was used to assay the activities of plasma and liver ALT and AST, whereas the ALP activities were determined according to the methods of Kind and King, (1954). Glutathione reduced (GSH) was determined according to the method of Jollow *et al.* (1974).Superoxide dismutase (SOD) activity was measured according to Mishra and Fridovich (1972). Catalase (CAT) activity was determined using the Luck method involving the decomposition of hydrogen peroxide (Luck, 1974). Glutathione S-transferase (GST) activity was determined according to Habig *et al.* (1974). Thiobarbituric acid reactive substances (TBARS) were measured by the method of Tappel and Zalkin (1959). The assay was done strictly according to the procedure given along with the kits.

Statistical analysis

Results were expressed as the mean ± standard deviation from at least three independent tests. Data for multiple variable comparisons were analyzed by one-way analysis of variance (ANOVA). For the comparison of significance between groups, Duncan's test was used a post hoc test according to the statistical package program (SPSS version 16.0). The significance level was set at $P < 0.05$.

RESULTS AND DISCUSSION

Fatty Acid Composition

Fatty acid contents of traditional ghee and plant ghee has been summarized in Table (2). Fatty acids composition of EG and PG were relatively high in saturation (71.57% and 51.74%), respectively. The result show that short chain fatty acids (C4:0 to C10:0) and medium chain fatty acids (C12:0 to C14:0) contents of ghee (EG) were higher than those of plant ghee (PG), (22.28%) and (1.35%), respectively. While palmitic (C16:0) and oleic(C18:1) were the main fatty acids in PG (45.28%, and 37.11 %), compared to (34.96%, and 21.19%) in EG. This results are in agreement with the findings of Dorni *et al* (2018) who found that milk ghee contained wide range of fatty acids, and palmitic acid was noticed to be the highest and ranged from (37.14 to 41.58%) followed by oleic acid which ranged from (19.49 to 25.12%).

Data in Table (2) show that the total trans fatty acids were (0.69%) and (0.15%) for EG and PG, respectively. Atta *et al.* (2013) found that total trans fatty acids content of vegetable ghee 1 was (0.21%),while the percentage of total trans fatty acid were more in vegetable ghee 2 was (10.26%),this is may be due to the vegetable ghee 2 including hydrogenated oils which their is role considered as a source of trans fatty acid.This is indicated that PG in the present study not content of hydrogenated oil, because its content a small amount of trans fatty acids (commercial palm oil).Also, the results in agreement with Deosarkar and Khedkar (2015) reported that milk ghee contained trace amount of trans fatty acid which ranged from (0.32% to 0.73%). May and Nesaretnam, (2014) and Morsi *et al.* (2014) found that Palmitic acid is the major SFA in palm oil (44%0, counter- balanced by almost 39% mono unsaturated oleic acid, and 11% polyunsaturated linoleic acid.

Table 2. Fatty acid percentage contents of Egyptian ghee and plant ghee

Fatty acid%	EG	PG
C4:0(butyric)	0.85	0
C6:0 caproic acid	0.67	0
$C_{8:0}$(Caprylic)	1.17	0.03
$C_{10.0}$(Capric)	2.51	0.03
$C_{12:0}$(Lauric)	3.2	0.26
$C_{14:0}$(Myristic)	13.88	1.03
$C_{17:0}$ (Heptadecanoic)	1.71	0.1
$C_{16:0}$(Palmitic)	34.96	45.28
$C_{18:0}$(Stearic)	12.34	4.6
$C_{20:0}$(Arachidic)	0.28	0.41
Σ SFA	71.57	51.74
C14:1(Caproic)	1.56	0
$C_{16:1}$(Palmitoleic)	2.31	0.21
$C_{18:1}$(Oleic)	21.19	37.11
$C_{20:1}$(Eicosenoic)	0.03	0.17
Σ MUSA	25.09	37.49
$C_{18:2}$(Linoleic)	1.4	10.41
Σ $C_{18:2}$ Trans	0.69	0.15
$C_{18:3}$(Linolenic)	0.48	0.21
CLA(Conjugated linoleic)	0.77	0
Σ PUSFA	2.57	10.77
n6:n3	2.92	49.57

EG: Egyptian ghee (samna) PG: plant ghee
Σ SFA:Saturated fatty acids,
Σ MUSFA: Monounsaturated fatty acids,
Σ PUSFA:Poly Unsaturated fatty acids,
Σ Trans:Trans fatty acids

As shown from (Table2) the highest contents of stearic acid (C18:0) and linoleinc (C18:3) were (12.34% and 0.48%) in EG, respectively compared to those of palm oil, (4.6% and 0.21%), respectively. These

findings are in agreement with the results reported by Atta *et al.* (2013) who found that the stearic acid C18:0 in vegetable ghee was ranged btween (4.79 - 4.38%).

Conjugated linoleic acids CLA and butyric acid, both of which have positive healthy effects in the body. In the present study Egyptian ghee(EG) contains (0.77%) conjugated linoleic acid CLA and butyric (0.85%), while Pegolo *et al* (2017) found that the main conjugated linoleic acid, rumenic acid, represented 0.45% of total milk fatty acids.

Body weight and Body weight gain of rats

The data in Table (3) show the effect of feeding Egyptian ghee and plant ghee on body weight and percentage body weight gain of rats. There were no significant variances in initial body weight, final body weight among the studied groups.

Initial body weights of the three groups were almost similar, the body weights ranged from (134 to 138g). The data show that the highest percentage gain in body weight was (45.52%) for PG group, the lowest weight gain was for EG group (39.86%) compared to control group (41.91%). The difference between EG group and PG group was significant (Table 3). Reduction in the weight gain in EG group might be due to the high content of short and medium chain fatty acids in ghee (22.28%) compared to (1.35%) in plant ghee. St-Onge and Jones (2000) found that when medium chain fatty acids replace long chain triglycerides in the diet, these different metabolic routes appear to promote satiety faster and increased energy expenditure, possibly leading to weight control. Besten *et al*, (2015) mentioned that dietary short chain fatty acids SCFAs protect against obesity. Malinska *et al*, 2015 found a reduction in visceral and ectopic lipid accumulation in rats fed with milk ghee. Lie *et al* (2016) reported that short and medium chain fatty acids, are very useful for the body weight management and can be processed by the liver and burnt as energy, not passing into the adipose tissue or contributes to weight gain.

In addition, as shown from Table (2) conjugated linoleic acid of Egyptian ghee was (0.77%) could also be one of the reasons for weight gain reduction. Also, Blankson *et al* ,(2000) they suggested that conjugated linoleic acid may reduce body fat mass in humans.

Hematological parameters

The hematological results are shown in Table (4). There were non-significant differences in WBC, Hb, and HT values among the control and experimental groups. On the other hand, the RBC significantly decreased by (18.5%) in the PG group compared with the control. The RBC increased by (8.5%) in the EG group compared with control group, but the increase was not significant. The increase in WBC in PG group was (11.4%) comparing with control group (Table 4). Robbins *et al.* (2012) mentioned that the increase of leucocytes may be caused by inflammation.

Also, the results in Table (4) show a decrease in HT concentration of plant ghee (44.9%) compared with control group (53.4%). Earlier studies showed a decrease in HT concentration of thermoxidized ghee fed rabbits (Zeb, and Uddin, 2017).

Kidney functions

Data present in Table (5) shows the changes in plasma total urea, and creatinine. The data indicates that PG group had the highest plasma total urea, and creatinine (63. mg/dl) and (0.466 mg/dl), respectively, when compared to control group.

Table 3. Body weight and percentage of body weight gain of rats

Body weight / groups	Initial weight (g)	Final weight (g)	% body weight Gain	Feed intake (g/day)
Control	136±15.5[a]	193±35.28[a]	41.91[a]	18±0.95[a]
EG	138±15.6[a]	193±21.96[a]	39.86[b]	18±1.34[a]
PG	134±15.5[a]	195±25.49[a]	45.52[a]	20±1.92[a]

Mean values within a row not sharing a common superscript are significantly different, P< .05
EG: Egyptian ghee (samna) PG: plant ghee

Table 4. The effect of Egyptian ghee and plant ghee on hematological parameters

Groups	RBC ($\times 10^6$/ml)	WBC ($\times 10^3$/ml)	Platelets PLT	HB g/d	HT %
Control	6.82±0.372[a]	11.4±0.78[a]	340±106[b]	14.9±0.48[a]	53.4±4.55[a]
EG	7.40±0.187[a]	11.2±1.47[a]	361±39.5[b]	14.1±0.59[a]	49.0±2.45[a]
PG	5.56±1.374[b]	12.7±0.35[a]	462±47.2[a]	12.6±3.57[a]	44.9±12.14[a]

Mean values within a row not sharing a common superscript are significantly different, P< .05
EG: Egyptian ghee (samna) PG: plant ghee

Table 5. Effect of Egyptian ghee and plant ghee on kidney function

Parameters	Urea (mg/dl)	Creatinine (mg/dl)
Control	59±19.49[a]	0.380±0.07[a]
EG	55.40±7.50[a]	0.346±0.07[a]
PG	63±7.38[a]	0.466±0.12[a]

Mean values within a row not sharing a common superscript are significantly different, P< .05
EG: Egyptian ghee (samna) PG: vegetable ghee

Whereas treatment with EG caused the lowest changes (55.4mg/dl) and (0.346mg/dl), respectively. Differences are insignificant.

Total lipids, total cholesterol, triglyceride, HDL, LDL and VLDL

Table (6) shows that PG treatment caused significant increases in total lipid TL, triglyceride TG, TC, LDL-c and V-LDL-c concentration in the plasma and a significant decrease in HDL-c level compared to control group. On the other hand the treatment with EG ghee did not cause significant changes in all of the studied blood lipid profile except for HDL(29.5%) compare with control.

The results of lipid profile are in agreement with Nirmala *et al.* (2016) they found significant decreases in total cholesterol, LDL-c, and VLDL-c level (39.4 mg/dl), (21mg/dl), and (13.2mg/dl), respectively, and significant increases in HDl-c and triglycerides (14.6mg/dl), and (71.4mg/dl) respectively, when rats fed with 10% crud ghee for 8 weeks compared with control group.

According to Chinnadurai *et al* (2013), ghee improves lipid profiles by increasing HDL content and reduces lipid peroxidation.

There are a few studies done on animal models, which reveal the exact opposite effect of Ghee consumption on cholesterol levels. In an 8-week study done on Wistar rats that were fed a balanced diet including 2.5% to 10% Ghee, an inverse correlation was found between ghee consumption and cholesterol levels,(Kumer *et al*, 2000). The serum lipid profiles of these animals showed a dose dependent decrease in total cholesterol, low-density lipoproteins and very low-density lipoproteins cholesterol and triglyceride levels when ghee was fed greater than 2.5% in the diet.

Plasma and Liver enzymes (ALT, AST and ALP)

The results in table (7) show significant increases in plasma ALT, AST and ALP activities for PG group. This indicate the hepatotoxicity and loss of structural integrity. However EG group decrease plasma AST and ALP when compared with control group.

In the same Table the data show significant increases in liver ALT ,AST and ALP activities for PG group, and the treatment with EG very closed with control except for ALT plasma and liver. While the differences are significant.

An obvious sign of hepatic injury is the leaking of cellular enzymes such as ALT, AST and ALP into plasma due to the disturbance caused in the transport functions of hepatocytes. ALT is more specific to the liver, and it is a better parameter for analyzing hepatic injury. High levels of AST indicate the cellular leakage as well as loss of functional ability of liver cell membrane. Serum ALP is also related to liver cell damage (Darbar *et al.*, 2011).

The present results are in agreement with Zeb and Ullah, (2015) and Zeb and Uddin, (2017), who stated found an increase in ALT concentration when fed rabbits on oxidized lipid. Earlier studies of Al-Othman *et al*. (2006) also showed that oxidized rancid corn oil increased the serum ALT concentration in rats.

Chaturvedi *et al* ,(2016) reported that normal milk ghee was decreased liver function indices; (ALT), (AST), and (ALP) concentrations in plasma serum after 2 months compared with sunflower oil.

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Table 6: Total lipids, total cholesterol, and triglyceride, HDL, LDL and VLDL

Parameters / Groups	Total Lipids (mg/dl)	Triglyceride (mg/dl)	Total Cholesterol (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Control	248±8.46^{b}	75.02±5.407^{b}	58.28±4.028^{b}	23.14±1.851^{b}	20.13±4.481^{b}	15.00±1.078^{ab}
EG	254±11.01^{b}	69.00±9.964^{b}	60.60±4.231^{b}	29.97±4.653^{a}	16.83±4.308^{b}	13.80±1.991^{b}
PG	282±16.71^{a}	90.33±9.819^{a}	71.17±6.006^{a}	20.71±3.359^{b}	32.40±7.079^{a}	18.06±1.964^{a}

Mean values within a row not sharing a common superscript are significantly different, P< .05

EG: Egyptian ghee (samna) PG: vegetable ghee

Table 7. Plasma and Liver enzymes (ALT, AST and ALP)

Parameters / Treatment	ALT U/L		AST U/L		ALP IU/I	
	Plasma	Liver	Plasma	Liver	Plasma	Liver
Control	21.01±02.117^{c}	83.8±1.23^{c}	40.20±0.890^{b}	96.4±0.57^{b}	28.17±2.148^{b}	18.26±3.955^{b}
EG	24.80±01.483^{b}	86.8±1.45^{b}	39.50±3.505^{b}	95.1±2.95^{b}	27.96±0.775^{b}	19.19±4.414^{b}
PG	29.25±01.660^{a}	90.3±0.95^{a}	44.81±0.289^{a}	99.9±2.66^{a}	31.89±2.173^{a}	26.77±1.043^{a}

Mean values within a row not sharing a common superscript are significantly different, P< .05

EG: Egyptian ghee (samna) PG: plant ghee

Oxidative stress marker

A marked decrease in TBARS levels was found in the hepatic tissue homogenate of EG (18.83%) and increase in PG (3.47%). On the other hand, a decrease in GSH (11.4%) was obvious when compared to the control group (Table 8). However, GSH activities were very closed in the Egyptian ghee group as well as control group. The presence of EG group partially minimized the toxic effect of (TBARS) compared to PG group. Generally, treatment with ghee increased the activities of GSH and decrease the levels of TBARS in liver compared to plant ghee group. The free radicals are thought to react instantaneously with membrane lipids to cause lipid peroxidation and cell death (Sharma *et al*., 2008). Reactive oxygen species (ROS) are mainly generated in the mitochondria, leading to serious damage to cellular macromolecules, including protein dysfunction, lipid peroxidation, DNA damage, and oxidative stress (Djordjević, 2004; Galati e*t al*., 2002).

"Activities of antioxidative enzyme in tissues have always been used as a marker for tissue damage" (Tsai-Turton *et al.,* 2007). Liver enzyme like SOD, CAT, and GST were dramatically reduced in PG group compared to control group (Table 8). However, treatment with EG significantly increased SOD, and CAT, compared to control group. But GST activities were very closed for the Egyptian ghee group and control group.The presence of EG increased the activities of antioxidant enzymes and partially minimized the toxic effect of EG compared to PG. Chaturvedi *et al* ,2016 reported that rats received normal milk ghee showed no oxidative stress after 2 months compared with sunflower oil .

Rani and Kansal, (2012) compared the cancer protective effects of diet supplementation with milk Ghee G and Soybean oil on two groups of rats, they found that ghee had a protective effect against carcinogen induced mammary cancer in rats, and down regulates the enzyme activities responsible for carcinogen activation in liver and upregulates carcinogenic detoxification activities in liver and mammary tissues.

In the present study, it was probable that treatment with EG boosted the antioxidant system, The reduced oxidative stress and lipid peroxidation observed in the treated animals may be attributed to the important role of Egyptian ghee.

Table 8. Activities of antioxidant enzymes in liver

Parameters	SOD U/ml	CAT (U/ml)	GSH (U/ml)	GST (µmol/hr)	TBARS (nmol/ml)
Control	15.98±1.088^{b}	127±1.79^{b}	24.80±1.516^{a}	6.89±1.418^{a}	45.25±6.342^{ab}
EG	19.15±2.774^{a}	133±3.31^{a}	24.93±0.562^{a}	6.16±3.110^{a}	36.73±3.140^{b}
PG	15.25±1.058^{b}	98.6±6.00^{c}	19.92±0.580^{b}	4.06±1.186^{b}	46.82±9.680^{a}

Mean values within a row not sharing a common superscript are significantly different, P< .05

EG: Egyptian ghee (samna) PG: vegetable ghee

Glutathione (GSH) content, as a marker of non-enzyme antioxidant defense system, was also evaluated. GSH is a small thiol-containing molecule which represents the main nonenzymatic intracellular defense system reducing different ROS, such as hydroperoxides, peroxides, etc. In this respect, the decreased GSH content reflects the processes of cell oxidation and lipid peroxidation (Tzankova *et al*., 2017).

CONCLUSION

The study concluded that the health impact of traditional Egyptian ghee is better than plant ghee, however further research on the health effect of using Egyptian ghee in cooking are needed, to identify the effect of heat on oxidation of the fatty acids.

REFERENCES

Aboudonia, S.A. and S.I. AIagamy. 1993. Ghee ncyclopdia of Food Scince, Food Technology and Nutrition, (ds. R. Macra, R. K. Robinson & M. J. Sadlr) Acadmic Prss, London. 6:399.

Afsanh., A. Morshdi, Hossinpour and A, Mina. 2016. Invstigation of quality, advantags and disadvantags, procssing and charactristics of ghee: a review paper. Indian J. Fundam. Appl. Lif Sci. 6(S2): 1-7.

Ahmadias, l .N., M.R. Alipour, S. Andalib and, H, Ebrahimi. 2008.Effect of ghee oil on blood fat profile and passive avoidance learning in male rats. Medical Journal Tabriz University of Medical Sciences;30(3):7–10.

Al-Othman, A.M., F, Ahmad, S,Al-Orf, K.S.Al-Murshed and Z,Arif. 2006. "Effect of dietary supplementation of Ellataria cardamomum and Nigella sativa on the toxicity of rancid corn oil in rats," International Journal of Pharmacology, vol. 2, no. 1,pp:60–65.

Atta, M.M., A. A. Ahmed and A.Y. Girgis. 2013. Determination of trans fatty acids in animal fats and their blends with vegetable oil. J. Biol. Chem. Environ. Sci.,Vol.8(1): 333-350.

Besten,G.d., A. Bleeker., A. Gerding, K,Karen van Eunen, R. Havinga, T.H. Dijk, M.H. Oosterveer, J.W. Jonker, A.K.Groen, D. Reijngoud and B.M.Bakker.2015**.**Short-Chain Fatty Acids Protect Against High-Fat Diet–Induced Obesity via a PPARγ-Dependent Switch From Lipogenesis to Fat Oxidation. Diabetes. 64(7): 2398-408**.**

Blankson, H., J.A. Stakkestad, H. Fagertun, E. Thom, J.Wadstein and O. Gudmundsen. 2000. Conjugated linoleic acid reduces body fat mass in overweight and obese humans. The Journal of nutrition, 130(12): 2943-2948**.**

Bucolo,G. and H.Davi. 1973.Quantitative determination of serum triglycerides by use of enzymes.Clin Chem. (19): 476-482.

Burstein, M., H.R. Scholnick and R.Morfin.1980.Rapid method for isolation of lipoproteins from human serum by precipitation with polyanions.Scand J Clin Lab Invest. (40):583-595.

Chaturvedi, P., P. and K. B. Moseki., Mazunga. 2016. Effects of used sunflower oil and ghee (clarified butter) on lipid profile and antioxidants in SD rats. Ejbps. 3. 10:59-64.

Childs, A.C., S.L. Phaneuf, A.J.Dirks, T.Phillips and C.Leeuwenburgh. 2002. Doxorubicin treatment in vivo causes cytochrome C release and cardiomyocyte apoptosis, as well as increased mitochondrial efficiency, superoxide dismutase activity, and Bcl-2: Bax ratio. Cancer Res. (62): 4592-4598.

Chinnadurai, K., H.K,Kanwal, A.K,Tyagi, C,Stanton and P. Ross. 2013**.** High conjugated linoleic acid enriched ghee (clarified butter) increase the antioxidant and antiathrognic potency in female Wistar rats. Lipids in halth and disas.12(1). 121.

Darbar, S., A, Bhattacharya and SChattopadhyay. 2011. Antihepatoprotective of livina, a polyherbal preparation on paracetamol induced hep- atotoxicity: a comparison with silymarin, Asian J. Pharm. Clin. Res. 4 (1): 72–77.

Djordjević,V.B. 2004. Free radicals in cell biology. Int. Rev. Cytol. 237:57–89.

Dorni,C., P. Sharma, G. Saikia and T. Longvah. 2018. Fatty acid profile of edible oils and fats consumed in India. Food chemistry. 238:9-15.

Deosarkar,S.S. and C.D. Khedkar. 2015. Ghee. in caballero, finglas, & toldrá (eds.),encyclopedia of food and health. Academic Press.36(5): 483-496.

FAOSTAT. 2014**.** Food and Agriculture Organization of the United Nations (FAO) statistical database. Accessed Jun. 21. 2016.http://faostat3.fao.org.

Fossati, P. and L.Prencipe. 1982.Serum triglycerides determined calorimetrically with an enzyme that produces hydrogen peroxide. Clin Chem. (28): 2077-2080**.**

Galati,G**.,** S. Tafazoli, O.Sabzevari, T.S. Chan and P.J. O'Brien. 2002. Idiosyncratic NSAID drug induced oxidative stress. Chem. Biol. Interact. 142:25–41.

Grove,T.H. 1979.The effect of reagent pH on determination of high-density lipoprotein cholesterol by precipitation of with sodium phosphotungstate-magnesium.Clin. Chem., (25): 560-564.

Habig,W.H., M.J.Pabst, and W.B. Jakoby. 1974. Glutathione S-transferases the first enzymatic step in mercapturic acid formation. Journal of biological Chemistry, 249(22): 7130-7139.

IOOC .2001 .Method of Analysis of the International Olive Oil Concil. Preparation of fatty acid methyl esters from olive oil and olive-pomace oil.COI/T 20/Doc. No.24.

Iqbal, M.P.2014. Trans fatty acids – A risk factor for cardiovascular 456 disease. Pakistan Journal of Medical Sciences. 30(1):194-197.

Jollow, D.J., J.R. Michell. Zampaglionic and J.R.Gillete.1974. Bromoibenzene-induced Liver necrosis: Protective role of glutathione and evidence for 3. 4- Bromobenzene oxide as hepatotoxic metabolite. Pharmacology. 1: 151-169.

Kind, P.R.N and E.J.King. 1954**.** Estimation of plasma phosphatase by determination of hydrolyzed phenol with amino-anti-purine. Journal of Clinical Pathology.7: 322-326.

Kumar, M.V., K.Sambaiah and B.R.Lokesh. 2000. Hypocholesterolemic effect of anhydrous milk fat ghee is mediated by increasing the secretion of biliary lipids. J Nutr Biochem.11(2): 69-75.

Lei, E., K.Vacy and W.C,Boon,. 2016. Fatty acids and their therapeutic potential in neurological disorders. Neurochemistry International. 95: 75–84.

Luck,H.1974. Catalase. In: M.V,Bergmayer. (Ed.), Method of Enzymatic Analysis. Verlag Chemic. Academic Press.New York. p: 885.

Malinska, H., M. Hüttl, O. Oliyarnyk, M. Bratova and L. Kazdova. 2015. Conjugated linoleic acid reduces visceral and ectopic lipid accumulation and insulin resistance in chronic severe hypertriacylglycerolemia. Nutrition. 31(7-8): 1045-51.

May,C.Y. and K.Nesaretnam. 2014.Research advancements in palm oil nutrition. European journal of lipid science and technology.116(10):1301-1315.

Mishra, H.P. and I. Fridovich. 1972. The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. The Journal of Biological Chemistry.247:3170-3175.

Morsi, M. S., M. Galal, K. Abd El-Rahman and A. Katry. 2014. Palm kernel oil increases the risk of coronary heart disease in rats compared with ghee. IOSR Journal of Pharmacy and Biological Sciences (IOSR-JPBS) 9 (1):34-40.

Nirmala, K.S., S. Manjula and H. Sahajananda. 2016. Effect of two types of dietary ghee on serum lipid levels in rats. J. Evolution Med. Dent. Sci2016.5(49):3240-44.

Pegolo, S., G. Stocco, M. Mele, S. Schiavon, G. Bittante and A. Cecchinato. 2017. Factors affecting variations in the detailed fatty acid profile of Mediterranean buffalo milk determined by 2-dimensional gas chromatography. *Journal of dairy science*. *100*(4):2564-2576.

Rani, R., V.K. Kansal, D. Kaushal and S. De. 2011. Dietary intervention of cow ghee and soybean oil on expression of cell cycle and apoptosis related genes in normal and carcinogen treated rat mammary gland. Mol Biol Rep. 38(5): 3299-307.

Rani, R. and V.K. Kansal. 2012**.** Effects of cow ghee (clarified butter oil) & soybean oil on carcinogen–metabolizing enzymes in rats. Indian J Med Res.136(3):460-5.

Reitman,S. and S. Frankel. 1957. A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. American Journal of Clinical Pathology. (26): 56-63.

Robbins,S.L., V. Kumar, A.K,Abbas.,and, J.C. Aster.2012. Robbins Basic Pathology. 9th edn, Philadeiphia: Elsevier Health Sciences.

Sharma, A., P. Kaur, B.Kumar, S. Prabhakar and K.D.Gill.2008**.** Plasma lipid peroxidation and antioxidant status of Parkinson's disease patients in the Indian population, Parkinsonism Relat. Disord. 14: 52–57.

St-Onge, M. P. and P.J.H. Jones. 2002.Physiological Effects of Medium-Chain Triglycerides: Potential Agents in the Prevention of Obesity. *J Nutr 1132*(3): 329-332.

Tappel,A.L. and H. Zalkin. 1959. Inhibition of lipide peroxidation in mitochondria by vitamin E. Archives of Biochemistry and Biophysics. 80(2):333-336.

Tsai-Turton, M., B.T. Luong, Y. Tan and U. Luderer. 2007. Cyclophosphamide-Induced Apoptosis in COV434 Human Granulosa Cells Involves Oxidative Stress and Glutathione Depletion. Toxicol. Scince. 98:216-230.

Tzankova,V., D. Aluani, M. Kondeva-Burdina,Y. Yordanov, F. Odzhakov, A. Apostolov and K.Yoncheva. 2017. Hepatoprotective and antioxidant activity of quercetin loaded chitosan/alginate particles in vitro and in vivo in a model of paracetamol-induced toxicity. Biomedicine & Pharmacotherapy. 92. (2017) :569–579.

Walter,C.L. 1981. The exposure of humans to nitrite.Fd. Scince Technology. (Abs). (13): 385.

Walters,M. and H.Gerade.1970**.**Ultramicromethod for the determination of conjugated and total bilirubin in serum or plasma .Microchem.J15:231.

Zeb, A. and I. Uddin. 2017**.** The Coadministration of Unoxidized and Oxidized Desi Ghee Ameliorates the Toxic Effects of Thermally Oxidized Ghee in Rabbits. Nutrition and Metabolism:1-7.

Zeb, A. and S. Ullah. 2015. Sea buckthorn seed oil protects against the oxidative stress produced by thermally oxidized lipids. Food Chemistr. vol. 186. Pp: 6–12.

Zollner, N. and K. Kirsch. 1962. Ueber die quantitative bestimnung von lipoiden (mikromethod) mittels dervielennaturlichellipoiden (allenbekannten plasmalipoiden) gemeisamensulphophospho-vanillin reaction.Z.ges. exp. Med. (135):545-561

الملخص العربي

التأثير الصحى للسمن البلدى المصرى مقارنة بالسمن النباتى فى حيوانات التجارب

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تهدف الدراسة إلي مقارنة التأثير الصحى للسمن البلدى المصرى بالسمن النباتى وذلك من خلال تقدير تركيب الأحماض الدهنية وبعض المقاييس البيوكيميائية فى كل من الدم والكبد لعدد ٢١من ذكور فئران التجارب والتى قسمت إلي ثلاث مجموعات، المجموعة الضابطة تغذت على الوجبة القياسية والتى تحتوى على ١٠% زيت ذرة، المجموعة الثانية تم فيها إستبدال زيت الذرة ب١٠% سمن بلدى وفى الثالثة ب١٠% سمن نباتى(زيت نخيل) وذلك لمدة ٦ أسابيع. فى نهاية التجربة تم ذبح الفئران وتقدير صورة الدم و دهون الدم وبعض إنزيمات الدم الكبد، والتي تعتبر مؤشرا للحالة الصحية. عولجت البيانات إحصائياً باستخدام برنامج SPSS.

أظهرت النتائج أن السمن البلدى أدى إلى إنخفاض النسبة المئوية لوزن الجسم المكتسب والإجهاد التأكسدى وله تأثيرات وقائية على الكبد حيث أدى الى إنخفاض ملحوظ فى مستوى الجلسريدات الثلاثية واللليبوبروتينات منخفضة الكثافة، بينما أدى إلى إرتفاع مستوى اللليبوبروتينات مرتفعة الكثافة وأيضا زيادة نشاط الإنزيمات المضادة للأكسدة فى الكبد مقارنة بالمجموعة الضابطة ومجموعة السمن النباتى. وأشارت النتائج أنه فى مجموعة الفئران التى تغذت على السمن النباتى حدث زيادة معنوية فى كل من دهون الدم (الجلسريدات الثلاثية والكولسترول الكلى واللليبوبروتينات منخفضة الكثافة) ومستوى الراديكاليات الحرة وإنزيمات الكبد وحدث إنخفاض فى مستوى اللليبوبروتينات مرتفعة الكثافة ونشاط الإنزيمات المضادة للأكسدة. وخلصت الدراسة إلى أن الأثارالوقائية الصحية للسمن المصرى التقليدى أفضل من السمن النباتى، ويجب إجراء المزيد من البحوث عن الأثار الوقائية الصحية للسمن البلدى فى الطبخ المصرى، للتأكد من تأثير حرارة الطبخ على محتواها من الأحماض الدهنية المختلفة.