

## Research Journal of Pharmaceutical, Biological and Chemical

## Sciences

# Evaluation The Impact of Three Important Fat Sources in Egyptian Diets on Rats.

### Medhat M Abozid<sup>1</sup>\*, and Reham A Moustafa Mariah<sup>2,3</sup>.

<sup>1</sup>Biochemistry Department, Faculty of Agriculture, Menofia University, Shibin El-Kom, Egypt.

<sup>2</sup>Medical Biochemistry Department, Tanta University, Egypt.

<sup>3</sup>Department of Biochemistry and Molecular Medicine, Taibah University, Al-Madinah Al-Munawwarah, Kingdom of Saudi Arabia.

#### ABSTRACT

The objective of the present study was to evaluate three important fats (buffalo, cow and sheep tail) effects on rats. Fatty acids (FAs) were assessed in all fats. GLC analysis of FAs revealed the presence of palmitic, oleic and stearic acids fatty acid more than 67% from total fatty acids in three fats.Twenty-four male albino rats were used over a 45 day period. The animals were divided into 4 groups, where group 1 represents the negative control which was a fed basal diet, while group 2 received 20% buffalo fat in diet, group 3 was given 20% cow fat in diet and group 4 received 20% sheep tail fat in diet. The plasma lipid profile, liver function, catalase and malondialdehyde in the different groups were determined at the end of experiment.Feeding high fat diets (buffalo, cow and sheep tail fats) produced a significant increase in plasma total cholesterol, triglycerides, low density lipoprotein cholesterol, ALT activity, AST activity, catalase activity and malondialdehyde. A higher hyperlipidemic effect of buffalo and cow fats as compared to that of sheep tail fat may be due to the fact that the ratio of saturated to unsaturated fatty acids is lower for sheep tail fat (1.2) than for cow fat (1.4) and buffalo fat(1.7).

**Keywords:** buffalo fat - Cow fat - sheep tail fat – hyperlipidemia - fatty acids - cholesterol.



\*Corresponding author

7(1)



#### INTRODUCTION

Animal fats are acquirable in large quantities from the meat-packing industry [1] main application of edible fats is as a food cooking medium, but fats have recently lost most of the market share to vegetable oils due to the fact that animal fat contains too much SFAs, which may cause health problems [1].

Hyperlipidemia is a predominant risk factor for cardiovascular diseases (CVD) which remains as one of the leading causes of death all over the world [2]. Populations that consume a diet high in saturated fats and cholesterol tend to have higher incidence of coronary heart disease. The high levels of plasma LDL (low density lipoprotein) or other atherogenic lipoproteins are a prerequisite for most forms of atherosclerosis [3].

Cardiovascular diseases (CVD) represent major challenges for basic science and clinical research. It is obvious that appropriate animals models are crucial for studies on the pathogenesis and therapy of this complex metabolic disorder, but it is less clear how exactly to define the term "appropriate." From a scientific and an ethical point of view, it is reasonable to require that not only the phenotype but also the pathogenesis of the animal's condition resembles the human disease examined [4].

For this reason, researchers have been using fat-enriched, so-called high-fat (HF) diets, to generate obese rodent models [5]. A multitude of different HF diets have been used with relative fat fractions between 20% and 60% energy as fat, and the basic fat component varies between animal-derived fats, e.g., lard or beef tallow, and plant oils, e.g., corn or safflower oil [6,7].

Different surveys in Egypt concluded that the primary nutritional problem for many Egyptian adults is a tendency towards obesity [8].

In Egypt the major fat sources in human diets are buffalo, cow and sheep fats, so the aim of this study is focus on these types of fats and its effects on lipid profile, liver functions and antioxidant status in rats feed on these fats.

#### MATERIALSANDMETHODS

#### Fat sources

Buffalo, cow and sheep tail fats were purchased from the local market (Giza, Egypt).

#### Gas chromatography (GC) analysis of fatty acid methyl esters (FAMEs)

Saturated, unsaturated and total fatty acids were determined in fats by using methyl esters boron trifluoride method [9]. FAMEs were identified on a Agilent Technologies 7890A GC equipped with flame ionization detector (PE Auto System XL) with auto sampler and Ezchrom integration system. Carrier gas (He); ca. 25 Psi – air 450 ml/min – Hydrogen 45 ml – split 100 ml/min. Oven temperature 200°C injector and detector 250°C.

#### Animal and experimental design

The work was carried out at the Biochemistry Department, Faculty of Agriculture, Menofia University (Egypt). Male albino rats (n = 24) weighted 120–140 g were obtained from the Research Institute of Ophthalmology (Giza, Egypt). Rats were fed *ad lipitum* on a basal diet and water for 15 days as an adaptation period. They were housed individually in stainless steel cages and randomly dived into four groups of six. Rats in control group (Con) were fed with basal diet, while the others were fed with high-fat diet (20% fat source and the remaining 80% normal diet) for 6 consecutive weeks as follow:

Buffalo fat group (20% buffalo fat and the remaining 80% normal diet) Cow fat group (20% cow fat and the remaining 80% normal diet) Sheep tail group (20% sheep tail fat and the remaining 80% normal diet)



#### **Blood sampling and analysis**

Blood samples were collected after 45 days in tubes containing heparin as an anticoagulant from the eye plexuses under diethyl ether anesthesia and then centrifuged at 3000 rpm for 20 min to obtain plasma, which was kept frozen until analysis.

The total cholesterol was analyzed according to [10]. HDL-C was determined according to [11]. According to [12] LDL-C was calculated. The triglycerides were analyzed according to [13]. Alanineaminotransferase (ALT) and aspartateaminotransferase (AST) activities were measured according to the method described by [14]. ALP was determined according to the method of [15]. Total protein was determined according to [16]. Albumin was determined according to [17].Plasma malondialdehyde (MDA) was assessed as an indicator of lipid peroxidation [18] while catalase enzyme activity was determined according to [19].

#### **Statistical analysis**

The results of the animal experiments were expressed as the mean  $\pm$ SD and they were analyzed statistically using the one-way analysis of variance ANOVA followed by Duncan's test. In all cases *p*<0.01 was used as the criterion of statistical significance.

#### **RESULTS AND DISCUSSIONS**

#### Fatty acids profile of buffalo, cow and sheep tail fats

The fatty acids composition of buffalo, cow and sheep tail fats is presented in Table 1. According to the results shown, eight fatty acids were identified in three fats, while the analysis of FAMEs gave the proportion of palmitic, oleic and stearic as the major fatty acids which comprised together more than 65% of the total identified FAME in three fats.

Ret. Time	Fatty acids	Common name	Buffalo fat	Cow fat	Sheep tail fat
7.7	C12:0	Lauric	1.54	1.66	0.1
9.43	C14:0	Myristic	7.88	7.1	3.04
11.54	C16:0	Palmitic	38.14	29.36	22.04
14.18	C18:0	Stearic	9.56	11.23	22.38
11.91	C16:1 (n-9)	Palmitooleic	1.56	1.56	2.47
14.58	C18:1 (n-9)	Oleic	26.15	27.4	34.11
15.33	C18:2 (n-6)	Linoleic	4.56	3.92	2.82
16.4	C18:3 (n-3)	α Linolenic	1.52	1.13	0.5
Total identified fatty acids			90.91	83.36	87.46
Total identified saturated fatty acids			57.12	49.35	47.56
Total identified unsaturated fatty acids			33.79	34.01	39.9
Saturated/unsaturated ratio			1.7	1.4	1.2

#### Table 1: Fatty acids in buffalo, cow and sheep tail fats.

In buffalo fat the major fatty acid was palmitic (38.14%), followed by oleic (26.15%) and stearic (9.56%), this results are in line with [20]. In cow fat the major fatty acid was palmitic (29.36%), followed by oleic (27.4%) and stearic (11.32%), this results are agree with [21]. In sheep tail fat the major fatty acid was oleic (34.11%), followed by stearic (22.38%) and palmitic (22.04%), which is very close to the result of [22].

#### Impact of buffalo, cow and sheep tail fats feeding on lipid profile

By the end of the 45 days there was a significant increase in blood levels of total triglycerides, total cholesterol, LDL-cholesterol with no significant changes of HDL-cholesterol of high fat diet fed rats; (buffalo, cow and sheep tail groups) compared to the control group, (Table 2); a finding in accordance with that of [23,24].

7(1)



Groups	HDL-cholesterol mg/dl	Triglecerides mg/dl	T. cholesterol mg/dl	LDL-cholesterol mg/dl
Buffalo fat	30 ± 3.16 a	154.2 ± 2.6 d	146 ± 2.5 c	85.16 ± 3.7 c
Cow fat	28 ± 1.58 a	142 ± 3.3 c	135.6 ± 2.9 c	79.2 ± 1.8 c
Sheep tail fat	28.2 ± 1.3 a	125.4 ± 3.8 b	115.2 ± 3.7 b	61.92 ± 4.37 b
Control	28.4 ± 1.5 a	104 ± 4.6 a	91 ± 2.2 a	42.2 ± 3.9 a

#### Table 2: Statistical comparison between all studied groups as regards to lipid profile

The hyperlipidemic and particularly the hypercholesterolemic effects of the animal fats may be due to the higher percentage of saturated fatty acids (47.56% - 57.12%) in them; on the other hand three fats (buffalo, cow and sheep tail fats) contain a high amount of cholesterol [25, 26].

Dyslipidemic changes may be due to the increased triacylglycerol, content of the liver due to increased influx of excess non-esterified fatty acids (NEFAs) into the liver [27].

Our results showed that buffalo and cow fat diets elevated triglycerides, total cholesterol and LDLcholesterol levels more than this parameters in sheep tail fat diet, may be to high saturated/unsaturated fatty acid ratio (Table 1) and also high content of total cholesterol compared with sheep tail fat [25, 26].

#### Impact of buffalo, cow and sheep tail fats feeding on liver functions

Table 3 shows the effect of three fat types on the liver functions of rats. The transaminases AST and ALT merely give a crude estimate of the degree of liver damage. Hypercholesterolemia was characterized by a significant increase in ALT, AST and ALP.

#### Table 3: Statistical comparison between all studied groups as regards to liver functions

Groups	ALT	AST	AIP	Albumin	T.Protein
	0/1	0/1	0/1	g/ui	g/ui
Buffalo fat	32.83 ± 3 b	102.16 ± 5.67 b	281.5 ± 8.14 b	4.15 ± 0.27 a	7.05 ± 0.1 a
Cow fat	31.5 ± 2.8 b	100.16 ± 6.4 b	362.5 ± 34.58 c	4.183 ± 0.23 a	7 ± 0.32 a
Sheep tail fat	36.5 ± 1.7 b	110.16 ± 4.9 b	336.83 ± 24.28 c	4.25 ± 0.25 a	7.216 ± 0.54 a
Control	25.16 ± 0.98 a	59.5 ± 6.8 a	243.3 ± 26.19 a	4.17 ± 0.13 a	7.175 ± 0.13 a

#### Table 4: Statistical comparison between all studied groups as regards to lipid peroxidase and catalase

Groups	lipid peroxidase	catalse
	mmol/ml	U/ml
Buffalo fat	19.25 ± 2.11 c	317.5 ± 14.26 c
Cow fat	19.13 ± 0.628 c	271.3 ± 22.82 b
Sheep tail fat	15.46 ± 1.65 b	259.6 ± 23.34 b
Control	10.48 ± 1.25 a	181.83 ± 12.75 a

It is observed that diets containing high fat ratio (buffalo, cow and sheep tail fats) increased significantly ALT, AST and ALP activities compared with control group.

The highest level of ALT was 36.5 U/l in the sheep tail fat group, followed by buffalo fat group (32.83 U/l) and cow fat group (31.5 U/l), while highest level of ALP was 362.5 U/l in cow fat group, followed by sheep tail fat group (336.83 U/l) and buffalo fat group (281.5 U/l).

On the other hand the highest level of AST was 110.16 U/l in the sheep tail fat group, followed by buffalo fat group (102.16 U/l) and cow fat group (100.16 U/l).

This increase of ALT and ALP activities in blood indicates hepatic injury, while the increase of blood AST activity was more specific for cardiac injury. These data were in agreement with those of [26, 28].

January–February 2016



#### Impact of buffalo, cow and sheep tail fats feeding on oxidative statues

The Plasma lipid peroxidase level was increased significantly in the rats given high fat diets (buffalo, cow and sheep tail fats) by 83.7, 82.5and 47.5 %, respectively compared to the control rats, also and catalase activity was increased significantly in the rats given high fat diets (buffalo, cow and sheep tail fats) by 74.9, 48.1 and 42.77%, respectively compared to the control rats.

Hypercholesterolemia induces oxidative stress by causing a reduction in the antioxidant enzymatic defense potential of tissues and the generation of oxygen free radicals. As a result of these metabolic events, peroxidation reactions are accelerated, leading to cellular injury and atherosclerosis [29]. The elevation in oxidative stress in high fat rats (buffalo, cow and sheep tail fats) in the current study was reflected by a elevation in plasma catalse activity and the elevation of MDA as indicators of lipid peroxidation.

#### CONCLUSION

In the current study, despite the difference in the contents of FAs of buffalo, cow and sheep tail fats showed significant increase in plasma lipid profile, liver enzymes and lipid peroxidation parameters compared to the control rat group.

It can be concluded that three fats produced an increase in plasma lipid profile, liver enzymes and lipid peroxidation. The hybelipidemic effect of animal fats may be due to the presence of a high percentage of saturated fatty acid determined in the present study in addition to total cholesterol described previously.

#### REFERENCES

- [1] Santos DKF, Rufino RD, Luna JM, Santos VA, Salgueiro AA. J Petroleum Sci Eng 2013; 105: 43-50.
- [2] Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K. Lancet 2012; 380: 2224–2260.
- [3] Carmena R, Duriez P, Fruchart JC. Circulation 2004; 109: III2–III7.
- [4] Buettner R, Jurgen S, Bollheimer C. Obesity 2007; 15: 798–808.
- [5] Oakes ND, Cooney GJ, Camilleri S, Chisholm DJ, Kraegen EW. Diabetes 1997; 46: 1768–1774.
- [6] Ahren B, Gudbjartsson T, Al Amin AN. Pancreas 1999; 18: 75–83.
- [7] Lingohr MK, Buettner R, Rhodes CJ. Trends Mol Med 2002; 8: 375–84.
- [8] Food Agriculture Organization, 2015. http://www.fao.org/ag/agn/nutrition/egy\_en.stm.
- [9] Willim H. Official Methods of Analysis of the Association of Official Analytical Chemists 2000, pp 19-20.
- [10] Richmond W. Clin Chem 1973; 19: 1350-1356.
- [11] Lopez MF, Stone S, Ellis S, Collwell JA. Clin Chem 1977; 23: 882-886.
- [12] Demacker AG, Hijmans BG, Brenninkmeijer AP, Jansen JS. Clin Chem 1984; 30: 1797-1800.
- [13] Fossati P, Prencipe L. Clin Chem 1982; 28: 2077–2080.
- [14] Retiman S, Frankel S. Am J Clinic Path 1957; 28: 56-59.
- [15] King EJ, Armstrong AR. Practical Clinical Biochemistry, 1980, pp. 850.
- [16] Tietz NW. Fundamentals of Clinical Chemistry. Philadelphia, Saunders WB, 1976, pp. 299.
- [17] Doumas BT, Watson WA, Biggs HG. Clin Chim Acta 1971; 31: 87-96.
- [18] Satoh K. Clinic Chim Acta 1978; 90: 37–43.
- [19] Sumner JB, Somers GF. Chemistry and methods of enzymes. New York, 1947, pp. 209.
- [20] Varricchio ML, Di Francia A, Masucci F, Romano R, Proto V. Ital J Anim Sci 2007; 6: 509-511.
- [21] Soyeurt H, Dardenne P, Gillon A, Croquet C, Vanderick S, Mayeres P, Bertozzi C, Gengler N. J Dairy Sci 2006; 89: 4858–4865.
- [22] Xiong D, Zhang H, Xie Y, Tang N, Berenjian A, Song Y. 2015; 11 (2): 57-65.
- [23] Woo MN, Bok SH, Lee MK, Kim HJ, Jeon SM, Do GM, Shin SK, Ha TY, Choi MS. J Med Food 2008; 11: 169-78.
- [24] Kamal AA, Mohamed AN. Diabet Metab Synd 2009; 1: 1-17.
- [25] Zicarelli L. Vet Res Communications 2004; 28: 127-135.
- [26] Ramadan MF, Zayed R, Abozid MM, Asker MMS. Grasas Y Aceites 2011; 62 (4): 443-452.
- [27] Grundy SM. J Clin Endo Metab 2004; 89(6): 2595-2600.
- [28] Daher CF, Baroody FG, Baroody MG. Fitoterapia 2006; 77: 183-188.
- [29] Gokkusu C, Mostafazadeh T. Clin Chim Acta 2003; 328: 155–161.

January–February

2016

RJPBCS

7(1)

Page No. 646