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Histological Changes and Lipid Profile Analysis in Male Rats Exposed High Fat Diet

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Abstract

Background: High Fat Diet (HFD) is associated with development of cardiovascular, metabolic, liver and central nervous system diseases. The study is aimed at evaluating the effects of high fat diets on lipid profile and selected organs in male wistar rats.
Methodology: 24 rats were randomly assigned into 4 groups. Group A received Normal Rat Chow diet + Distilled Water (DW), Group B fed 25% HFD + DW, Group C fed with 50% HFD + DW and Group D fed 100% HFD + DW. Animal's weights were measured pre and post-treatment. Rats were anaesthetized (diethylether) and sacrificed after 12 hours fast. The blood samples taken from the orbital sinus, centrifuged and serum obtained was used for the assay of lipoprotein A, Total cholesterol (TC), triglycerides (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL). The brain, liver and heart were excised and processed for histology.
Results: Result showed the presence of a significant dose dependent increase ($p < 0.05$) in the body weight, concentrations of TC, TAG, LDL, lipoprotein A and decrease in the concentrations of HDL in HFD groups compared with the control. Histological results from HFD treated rats have showed no histological abnormalities of the liver, brain and heart tissues.
Conclusion: Intake of high fat diets in this study altered serum lipid profile with significant weight gain - a risk factors for cardiovascular and metabolic diseases.

Keywords: High Fat Diet, Lipid profile, Lipoprotein A, Histological Changes

1.0 Introduction

Lipids are water insoluble organic compounds, which are essential for many normal functions of living organisms. The principal functions of lipids are to act as energy stores and to serve as important structural component of cells [1]. The high saturated fatty acids intake increases the Low-Density Lipoprotein cholesterol (LDL-c) and reduces the High-Density Lipoprotein cholesterol (HDL-c) in the bloodstream [2]. A balance of unsaturated fatty acids is important when selecting food sources and those rich in monounsaturated and polyunsaturated fatty acids; particularly, the

long-chain n-3 polyunsatured fatty acids should be used preferentially since they reduce the risk for atherosclerosis [3]. Lipoprotein A is a Low Density Lipoprotein (LDL) that contains a lipid core and an Apo lipoprotein B subunit. Lipoprotein A differs from LDL, as it also contains an Apo lipoprotein A subunit, Apo protein (a) [4]. Lipoprotein A is a Low Density Lipoprotein (LDL) that contains a lipid core and an Apo lipoprotein B subunit. Lipoprotein A differs from LDL, as it also contains an Apo lipoprotein A subunit, Apo protein A [4]. Lipoprotein A is produced predominantly (or perhaps only) by the liver [5]. Lipoprotein A levels are thought to be largely controlled by genetic factors. However, several reports have indicated that the type of fat consumed can alter Lipoprotein A levels [5]. Consumption of High Fat Diet directly affects the serum Lipid Profile and the fatty acids composition, which is an important factor in the modulation of lipid metabolism [6]. High-Fat Diets are known to lead to a positive fat balance and consequently to adipose mass accumulation [6]. The increase in adipose tissue causes the clinical problems associated with obesity, Central Nervous System (CNS) diseases and other related diseases. Several studies have shown that High Fatty Diet may be related to an overproduction of Reactive Oxygen Species (ROS) and an increase in lipid peroxidation, leading to an oxidative imbalance in different tissues [7]. The abysmal elevation in the concentrations of lipid components seen in this study is of greater concerns. However, despite the elevation histological findings revealed no significant damages to the organs.

2.0 Materials and Methods

2.1 Chemicals and Reagents All chemical used in this study were standard and of analytical grade

2.2 Experimental animals

Twenty five (25) healthy male rats weighing 150 g – 180 g were used in this study. They were housed under standard laboratory conditions of light, temperature and relative humidity. The animals were given standard rat chow diet and water *ad libitum*. All the experimental procedures were carried out in accordance with the recommendation of National Institute of Health Guide for the care and use of Laboratory animals (NIH, 1985) as well as Ethical Guidelines for the use of Laboratory Animals in Kwara State University, Nigeria.

2.3 Test Diets

The normal and the high-fat diets (Table 1) were processed and prepared by an animal science nutritionist in the Department of Animal Science and Nutrition, Bowen University, Iwo, Osun State, Nigeria with the following feed compositions (% per 100 g of feed):

Table 1: Percentage Compositions of normal and experimental diets.

Ingredients	HFD (Qty/kg)	Normal Diet (Qty/kg)
Vegetable oil	3.00	0.06
Animal fat (Tallow)	20.00	0.00
Corn (yellow)	4.00	7.00
Soya full fat 48%	5.00	0.06
Di-methionine	0.20	1.20
Limestone	1.60	1.00
Vitamin-mineral premix	1.00	1.00
Fish meal	3.00	0.06
Milk + butter	4.00	1.00
Wheat bran	2.00	5.00
Lysine	0.06	2.00
Salt	0.50	0.50
PKC-DOC	0.01	2.00

2.4 Experimental design

Twenty four (24) from already acclimatized rats were randomly assigned into 4 groups. Each of the 3 4 group consisted of six rats. Group I received Normal Rat Chow diet + Distilled Water (DW), Group II fed with 25% HFD + DW, Group III fed with 50% HFD + DW and Group IV fed 100% HFD + DW. The animals were administered for six weeks.

High fat diet was prepared as previously described [8].

2.5 Animal sacrifice and blood sample collection

At the end of 6 weeks of treatments, animals were sacrificed under anesthesia (diethylether) after overnight fasting and blood samples taken from the orbital sinus, centrifuged at 300 rpm. Serum obtained used for biochemical analysis.

2.6 Assay of lipid profile

The concentration of lipoprotein A, total cholesterol (TC), triglyceride (TAG), high density lipoprotein (HDL), low density lipoprotein (LDL) & very low density lipoprotein (VLDL). The concentration of cholesterol was measured according to method [9], the determination of the concentration of HDL was done by enzymatic method [10]. While LDL was measured according to the Fried Ewald Equation. VLDL concentration can be calculated by dividing the value of TAG by 5. The concentration of triglycerides by enzymatic method was estimated according to the method. [11]

2.7 Histology

The portion of the brain, liver and heart were excised and stained with H & E stain for histological examination, according to the method of histological technique described.[12]

2.8 Statistical analysis

Data obtained are expressed as Mean ± Standard Error of Mean (SEM) of 6 determinations. For statistical analysis, data were subjected to one-analysis of variance (ANOVA) and followed by post hoc test. The data were considered statistically significant at P values of < 0.05.

3.0 Results

3.1 Body weight

As shown in Table 1 below, the mean body weights of the rats were almost the same in all groups at the onset of the study. At the time of sacrifice, the mean body weight increased significantly (P< 0.05) in groups III and IV fed compared with the baseline weights. Percentage increase in mean body weights were 41.3% and 60.5% respectively .while no significant differences were observed in baselines and post treatment of group II and the normal control.

Table 2: The Effects of high fat- diet on the animal's weight.

Groups	Pre-Treatment Weight (g)	Post-Treatment weight (g)
Group I	186.23 ± 17.3	184.75 ± 15.02
Group II	182.23 ± 17.3	201.49 ± 16.93*
Group III	172.66 ± 14.2	23n8.09 ± 17.38*
Group IV	170.65 ± 19.6	273.87 ± 26.29*

Values are expressed as Mean ± SEM (n = 6).* P <0 05, statistically significant from the normal control.

3.2 Lipid Profile

Table 2 below shows the comparative effects of the test diets on the lipid profile of the experimental and control rats. After 6 weeks of diet, levels of the constituting parameters of the lipid profile increased significantly (P< 0.05) in dose dependent manner across all groups except HDL with reduction in groups III and IV.

Table 3. The Effect of high fat- diet on serum lipid profile in male Wistar rat.

Groups	TC (mmol/l)	TG (mmol/l)	HDL (mmol/l)	LDL (mmol/l)
Group I	2.58 ± .05	1.26 ± .12	.80 ± .05	.60 ± .05
Group II	3.12 ± .09*	1.60 ± .07*	.90 ± .07	.66 ± .05
Group III	3.6 ± .07*	1.76± .07*	.74 ± .09	.62 ± .04
Group IV	4.08 ± .07*	2.04 ± .05*	.68 ± .07	1.74 ± .08*

Values are expressed as Mean ± SEM ($n = 6$).* $P < 0$ 05, statistically significant from the normal control.

3.3 Lipoprotein A

The concentration of the serum lipoprotein A were observed to be significantly (P< 0.05) increased in dose dependent manner among the rat groups treated with high fat diet when compared with the normal control as shown in Figure 1.

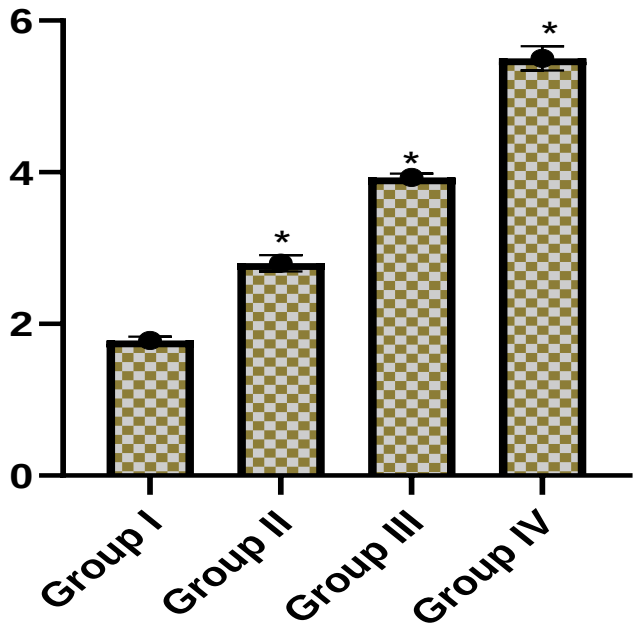

Figure 1. The Effect of high fat- diet on serum lipoprotein A in male albino rats. Values are presented as means ± SEM. Mean values with (*) are significantly different ($p < 0$ 05)

3.4 Histological Analysis

Under high power magnification light microscopic examination, of the liver, heart and brain tissues of the experimental and normal rats were closely examined. Liver, heart and brain cells from grouped rats and control shows normal histoarchitecture as shown below (Plates 1-3).

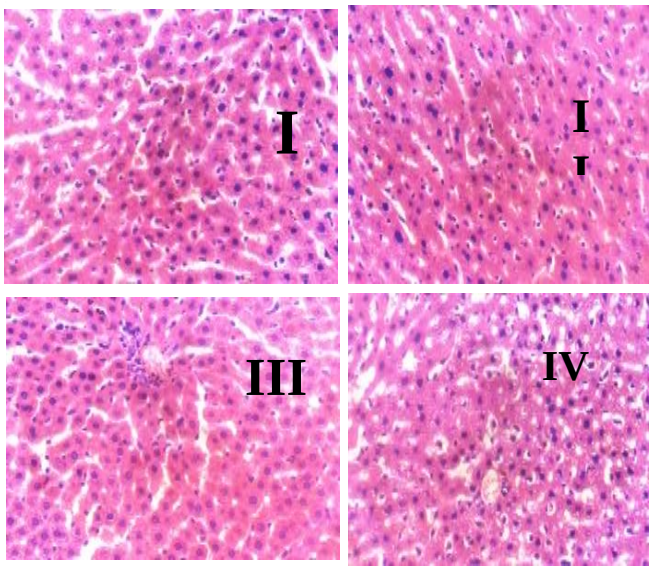

Plate 1: Photomicrographs of the liver (H & E, x 20). showing preserved liver architecture with normal hepatocytes and portal tracts (I)normal control (II) 25% HFD (III) 50% HFD, (IV) 100% HFD.

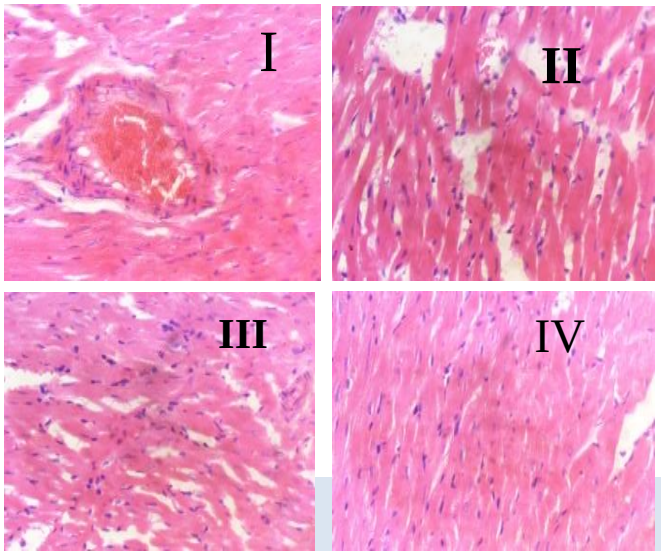

Plate 2: Photomicrographs of the heart tissues (H & E, x 20). Showing preserved branching bundles of myocytes, patent coronary vessels and mild inflammation (I) normal control (II) 25% HFD (III) 50% HFD, (IV) 100% HFD.

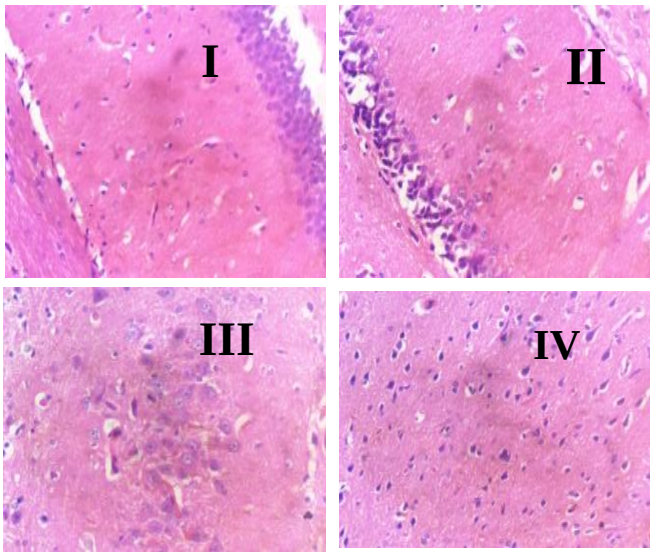

Plate 3: Photomicrographs of the brain tissues (H & E, x 20). showing preserved neuronal bodies surrounded by glial matrix in the cerebral and hippocampal cortices (I) normal control (II) 25% HFD (III) 50% HFD, (IV) 100% HFD.

4.0 Discussion

High fat diet intake in this study significantly *(P* <0 05) increases the total body weight compared to the normal control as shown in (Table 2). The observed increase in the body weight agrees with the findings of few studies [13],[14] which suggested that total dietary fat intake is linked to an increased risk of obesity and diabetes. The Result of table 3 showed the presence of a significant elevation (p <0.05) in the concentrations of total cholesterol, triglycerides and low density lipoprotein levels in high-fat fed experimental groups in a dose dependent pattern; and a decrease in the concentrations of high density lipoprotein in groups III and IV compared with the normal control group**.** as shown in figure 1 there were observed significant *(P* <0 05) dose dependent increment in the concentrations of lipoprotein A. This hyperlipidemia observed in this study supports the impact of high fat diets on blood lipid chemistry as previously by results of other studies [15],[16]. The findings from histological examinations of the liver, brain and heart tissues revealed normal cellular histoarchitecture. These reports were not in line with previous study [17], The differences may be due to short treatment duration (6 weeks) as against the 14 weeks of feeding in the study. Consumption of a high fat diet, has been viewed as a significant modifiable risk factor for some diseases such as Type 2 diabetes, cardiovascular disease and certain types of cancer (Yehul *et al*., 2018). However, the mechanisms by which HFDs exert adverse effects on human health remains poorly understood. This abnormal increase in the levels of lipid components calls for reasonable caution.

5.0 Conclusion

This study evaluated the effects of high intake of dietary fat on body weight, lipid profile and liver, heart and brain histology. The findings obtained from the study revealed increased cardiovascular and metabolic risks associated with consumption of high dietary fat as evidenced by significant weight gained and hyperlipidemia.

Conflicts of interest

The authors declare no conflicts of interest

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