



# Preparation of High Fat Diets and their Impact on the Development of Obesity, Hyperlipidemia and Hyperglycemia in Albino Rats

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## KEYWORDS

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metabolic syndrome,  
obesity,  
dyslipidemia

## ABSTRACT:

**Introduction:** The rising prevalence of type 2 diabetes has drawn attention to dietary factors contributing to insulin resistance, a hallmark of the disease. High-fat diets (HFDs) have been consistently associated with the development of hyperglycemia and insulin resistance in experimental animal models.

**Objectives:** This study aimed to investigate the effects of different fat sources in HFDs on the induction of insulin resistance in rats. Five types of HFDs were formulated using the fat sources i.e. branded vegetable fat i.e. Dalda (hydrogenated fat), Animal fat (lard), Margarine, Coconut oil, and Palm oil

**Methods:** All diets were otherwise identical in composition, consisting of Bengal gram flour, soybean, sugar, mineral salts, cholesterol, and albumin, yielding a nutritional profile of around 25% carbohydrates, 25 % protein, 20 % fat, 4 % fiber, and approximately 1.0 % of salts and minerals.

**Results** The ingredients were thoroughly mixed, formed into dough, pelletized, and stored at -20°C to minimize fat oxidation prior to use.

**Conclusions:** The results demonstrated that none of these prepared high fat diets could induce either obesity, hyperlipidemia and hyperglycemia in Sprague Dawley strain of albino rats.

## 1. Introduction

The metabolic syndrome includes a number of metabolic problems, including insulin resistance, central obesity, high blood pressure, and dyslipidemia. It raises the risk of getting type II diabetes mellitus and atherosclerotic heart diseases (Swarup et al., 2024). Concerning to note that most of the population today suffers from metabolic syndrome, whose prevalence has been increased dramatically in recent decades in tandem with the global rise in obesity rates (Larruy et al., 2024). Person who are obese and more likely to display symptoms of metabolic syndrome has a clustering of phenotypes associated with higher cardiovascular risk. Diabetes mellitus is a metabolic imbalance that is not physiological. About 7% of adults around the world suffer from diabetes mellitus (DM), which is a global health issue (Yameny et al., 2024). Diabetes is a

prevalent chronic disorder of glucose metabolism caused by inadequate synthesis or use of insulin, resulting in hyperglycemia and glycosuria. Among the most prevalent signs of diabetes are elevated blood glucose and problems with reproduction, which put a person at risk for several complications (Abel et al., 2024). Type 2 diabetes accounts for 85–95% of all occurrences of the disease, and its widespread occurrence has a significant negative impact on health care (Ma et al., 2024). There are currently 41 million diabetics in India, and by 2025, there should be 70 million, estimated 380 million people worldwide are expected to have diabetes by 2025. (Kumar et al., 2024). Another disorder which associated with metabolic syndrome is dyslipidemia, an important risk factor for cardiovascular (CV) disorders is dyslipidemia, which is defined as abnormal blood lipid levels (Pappan et al., 2024). The next disorder that is



associated with metabolic syndrome is dyslipidemia, an important risk factor for cardiovascular (CV) disorders, which is defined as abnormal blood lipid levels. Triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL) cholesterol, and cholesterol are important contributions to these pathways. It has been shown that excessive consumption of fats increases fat-mediated oxidative stress and reduces the activity of antioxidative enzymes, which may lead to many risk factors like dyslipidemia and hypertension (Yang et al., 2024).

Several rodent models have been employed to investigate the pathogenesis of metabolic syndrome. Rodent models fed a high-fat (HF) diet have made a substantial contribution to the study of the pathophysiology of insulin resistance syndrome. In 1959, Masek and Fabry described the first nutritional intervention that used a "high-fat diet" to cause obesity (Buettner et al., 2024). The term "high-fat diet" is used to describe a variety of diets with wildly disparate fatty acid contents, as the research makes clear. This has unavoidably caused a great deal of variation in the reported results. In contrast to the regular chow diet, the majority of research has only used one high-fat formula and has not examined the impact of the particular fat component in the model. It is generally accepted that diets based on saturated fatty acids cause the typical high-fat diet phenotype, while diets containing polyunsaturated  $\omega$ -3 fatty acids have positive effects on body composition and insulin action, based on the limited data comparing various high-fat diets with regard to their metabolic effects (Varra et al., 2024).

The present study is an attempt to prepare five different types of diets supplemented with saturated fats such as hydrogenated vegetable fats (commercial product: Dalda), margarine, coconut oil, and palm oil, animal fat (lard), in the hope that feeding these to experimental animals may create symptoms of metabolic syndrome.

## 2. Methods

### 2.1: Procurement of Rats and Normal Pellet Diet.

Thirty male Sprague Dawley (SD) rats weighing about 120-150 g (6 to 8 weeks old) were procured from the animal colony of CSIR-Central Drug Research Institute, Lucknow. These were acclimatized in the room where

these standard conditions are being maintained i.e. Light 50 to 50 lux, temperature  $25 \pm 2$  °C, air changes 10 to 12 per hour, and light-dark cycle 12/12 hours. The animals were randomly divided and five animals were kept in one polypropylene cages of the size i.e. 30 x 22 x 14 cm). They were fed ad lib normal pellet diet and had access to tap water until the start of experiment. The normal pellet diet was procured from Hylasco Bio Technology (India) Pvt Ltd. India.

### 2.2: Preparation of High Fat Diets

The ingredients used in the preparation of high fat diets (HFD) were procured from local market include gram flour, soya bean, table sugar, mixture of salts, Dalda, Margarine (Delicious table margarine), Palm Oil (Natraj super refined palmolein), Coconut Oil (KPL Shudhi coconut oil) and animal fat. The other chemical ingredients were procured from Sisco Research Laboratories, Mumbai. These were cholesterol, egg albumin and monosodium glutamate (MSG). Five different diets were prepared by mixing the ingredients. Each diet has different type of fat. The composition of these high fat diets has been shown in Table 1.

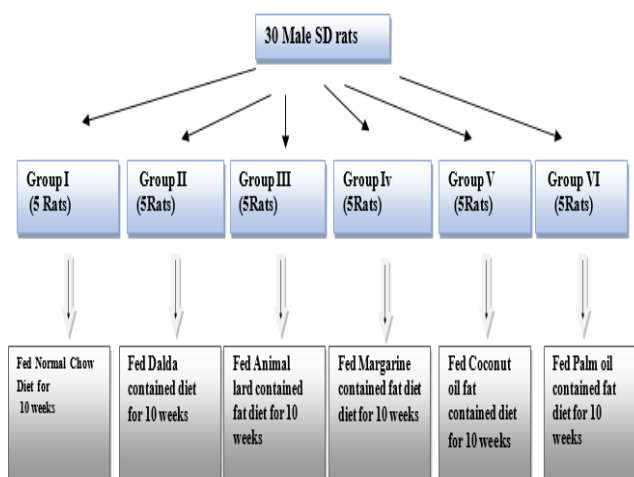
**Table 1. Composition of High-Fat Diets**

<i>Ingredients</i>	<i>Quantity gm/kg</i>
Gram Flour	250
Soyabean	200
Sugar	100
<b>Dalda, lard, margarine, coconut oil and palm oil</b>	250
Cholesterol	10
Albumin	50
Mineral Salt	10
MSG	10

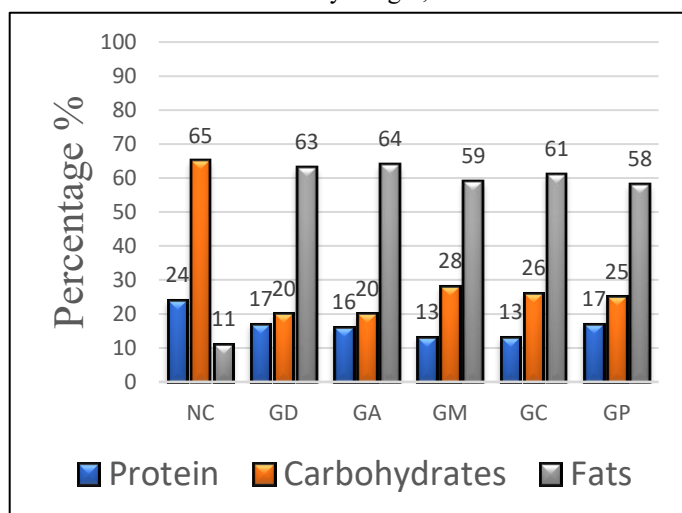


### 2.3: Grouping of animals and feeding the diets

Post acclimatization, the animals were randomly divided into six groups each having five animals. The groups were termed as I to VI. The animals of Group-I were kept on normal pellet diet, group-II was kept on prepared diet having Dalda as the source of fat, group-III was kept on prepared diet having animal lard as the source of fat, group-IV was kept on prepared diet having margarine as a source of fat and group-V was kept on prepared diet having coconut oil as a source of fat and group-VI was kept on prepared diet having palm oil as the source of fat. However, the animals received 10 % fructose in their drinking water throughout the experiment. Fig 1 shows the diagrammatic representation.



### 2.4: Measurement of body weight, food and water intake



Each day the animals were weighed on single pan balance. Food and water intake of each group was determined by the difference in weight and volume of food kept in the cage and water filled in graduated bottle and weight of food and water remained in the cage and bottle.

### 2.5: Estimation of Blood glucose, Oral Glucose Tolerance and Serum lipid profile.

On day 30, 60 and 90th post feeding with the HFDs, the oral glucose tolerance of each animal was determined as follows. The animals were starved for 6 to 8 hours and the fasted animals were given a glucose load (10 g/kg), and blood glucose was monitored in tail snips every 30 min post glucose load till 120 min. Blood glucose was monitored by glucostrips with the help of Glucometer. The blood of each animal was collected from jugular vein before the sacrifice of the animals on day 70th post start of the experiment. The serum was prepared by centrifuging the blood after keeping for 2 hours at room temperature. The serum samples were analyzed for lipid profile i.e. Triglycerides (TG). Total Cholesterol (TC), LDL, and HDL-cholesterol on Semi Auto Analyzer using assay kits and instructions as detailed by the manufacturer Erba Biochem.

### 2.6: Analysis of Data

The data of all the five animals in each group was expressed as mean  $\pm$  standard deviation (SD). The differences of ratios of indexes were analyzed using Dunett's test Prism software Version 5, and inter-group comparisons were made using the Multivariate General Linear Model.

## 3. Results and Discussion

Fig 1 shows the percent content of carbohydrate, protein and fats in the five prepared diets and normal chow diet. Diet containing dalda as the source of fat has around 20 % carbohydrate content, around 17 % protein content and around 63 % fat content. Diet containing animal lard has around 64 % fat content, around 20 % carbohydrate content and around 17 % protein content. Diet containing margarine as the source of fat contains around 59 % fat, 28 % carbohydrate and 13 % protein. Coconut oil containing diet showed around 61 % fat, 26 % carbohydrate and 13 % protein contents. Palm oil



containing diet showed around 25 % carbohydrate, 17 % protein and 58 %. Analysis of commercially obtained normal chow diet showed around 24 % protein, 65 % carbohydrate and 11 % fat content.

Figure 2 shows the changes in body weight of rats during the study. The mean body weight ( $\pm$  SD) of the experimental groups was significantly lower compared to the normal control group ( $284.00 \pm 75.99$  g). The values were  $181.10 \pm 16.00$  g in Group-II,  $160.50 \pm 4.79$  g in Group-III,  $164.10 \pm 6.63$  g in Group-IV,  $186.10 \pm 15.83$  g in Group-V, and  $200.70 \pm 23.39$  g in Group-VI

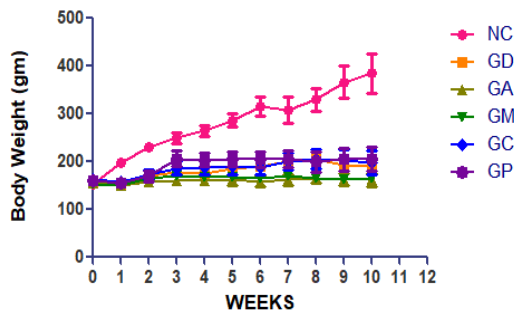
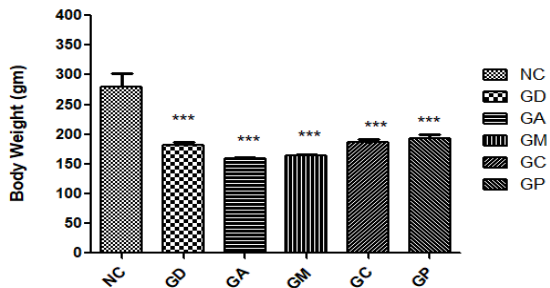


Figure 2: Graphical representation of Body Weight

Figure 3 shows the daily diet consumption of all the group. The average food consumption was recorded as 25.00 g/day in normal chow diet fed group (Group-I). Whereas, the food consumption in other groups were as follows i.e. around 11.00 g/day in group-II, around 9.00 g/day in group-III, 10.00 g/day in group-IV, around 12.00 g/day in group-V, and around 11.00 g/day in group-VI. It is evident from the results that food consumption of rats kept on the prepared diets was significantly lower ( $p < 0.05$ ) than normal control group.

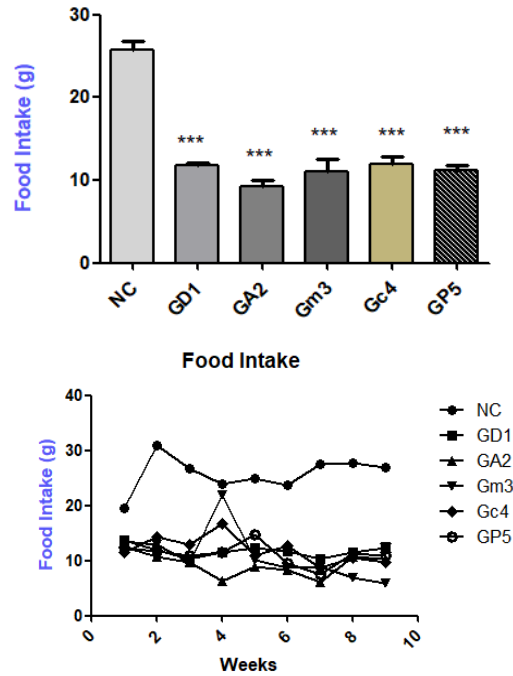
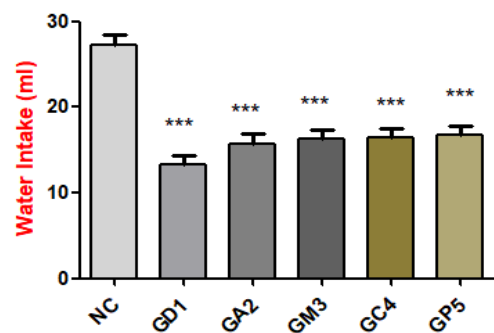
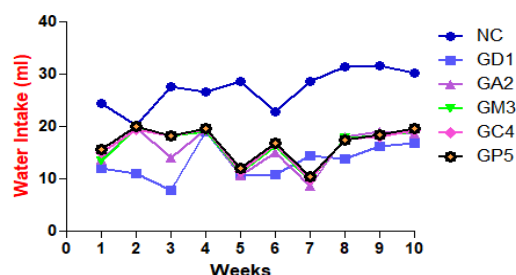


Figure 3. Graphical representation of Food Intake

Figure 4 shows the daily water intake of all groups. 10% fructose in drinking water was given to the experimental group, and normal drinking water was given to the normal control group. The normal diet group (Group-I) drank an average of 20 ml/day. In comparison, water intake was around 13 ml/day in Group-II, around 15 ml/day in Group-III, around 11 ml/day in Group-IV, around 14 ml/day in Group-V, and around 10 ml/day in Group-VI. These results showed that rats fed on the prepared high fat diets consumed much less water compared to the normal control group ( $p < 0.05$ ).





**Figure 4: Graphical representation of Water Intake**

Table 1 and 2. show the Oral Glucose Tolerance Test (OGTT) post glucose load. Comparing the Area Under Curve (0 to 120 min) between the groups, there was found no significant difference in either of the high fat diet fed groups to normal diet group on day 30th post feeding of respective diets. Similarly, there was found no significant difference in either of the high fat diet groups compared to normal diet fed group.

**Table 1. Oral Glucose Tolerance Test (OGTT) of rats on day 30th post feeding of High Fat Diets**

Group	Blood Glucose mg/dl					AUC
	0 min	30 min	60 min	90 min	120 min	
Group -I	96.20 ± 7.78	128.00 ± 11.11	138.00 ± 6.72	129.00 ± 2.00	112.20 ± 25.76	14982
Group -II	86.80 ± 3.54	117.60 ± 9.56	128.20 ± 2.31	123.20 ± 7.46	102.20 ± 6.85	13905 <sup>ns</sup>
Group -III	85.00 ± 5.09	118.00 ± 13.82	130.40 ± 13.15	112.60 ± 16.42	101.00 ± 8.76	13602 <sup>ns</sup>
Group -IV	86.60 ± 4.12	121.00 ± 14.91	129.40 ± 14.06	113.20 ± 6.77	101.40 ± 8.23	13728 <sup>ns</sup>
Group -V	86.60 ± 4.12	121.00 ± 14.91	129.40 ± 14.06	113.20 ± 16.77	101.40 ± 8.23	13698 <sup>ns</sup>
Group -VI	81.00 ± 4.69	114.00 ± 15.50	119.50 ± 6.34	113.00 ± 10.63	100.75 ± 4.91	13121 <sup>ns</sup>

All experimental groups were compared with normal control group (NC), Each value is presented in mean ±

Standard Error of Mean, (\*p <0.05; p>0.05 and ns= not significant).

**Table 2. Oral Glucose Tolerance Test (OGTT) of rats on day 60th post feeding of High Fat Diets**

Group	Blood Glucose mg/dl					AUC
	0 min	30 min	60 min	90 min	120 min	
Group -I	89.8 ± 7.60	139.80 ± 14.27	138.60 ± 2.57	139.60 ± 17.56	131.42 ± 13.97	15861
Group -II	96.20 ± 2.63	127.8 ± 10.53	150.4 ± 3.07	153.4 ± 23.90	122.6 ± 15.86	16230 <sup>ns</sup>
Group -III	83.80 ± 7.13	134.60 ± 25.31	143.20 ± 24.92	137.80 ± 22.85	119.00 ± 9.31	15516 <sup>ns</sup>
Group -IV	82.80 ± 7.8	131.00 ± 23.22	143.40 ± 25.43	131.20 ± 22.31	120.40 ± 9.58	16180 <sup>ns</sup>
Group -V	94.20 ± 3.18	130.40 ± 10.32	150.80 ± 2.85	151.20 ± 21.76	122.80 ± 15.15	15432 <sup>ns</sup>
Group -VI	104.20 ± 4.33	146.60 ± 24.42	156.00 ± 29.42	152.40 ± 10.53	117.70 ± 17.16	16905 <sup>ns</sup>

All experimental groups were compared with normal control group (NC), Each value is presented in mean ± Standard Error of Mean, (\*p <0.05; p>0.05 and ns= not significant)

Figure 5 a to d shows the serum lipid profile of rats of Group I to VI. It can be seen from the bar diagram that triglyceride levels showed increase in animals of Group IV whereas the animals of Group II showed significantly low triglyceride levels.

The animals of Group-V had slightly lower triglyceride level compared to normal diet control Group-I, whereas the other groups i.e. Group-III, Group- V and Group-VI have average mean values of 56.94, 54.82, 34.50, and

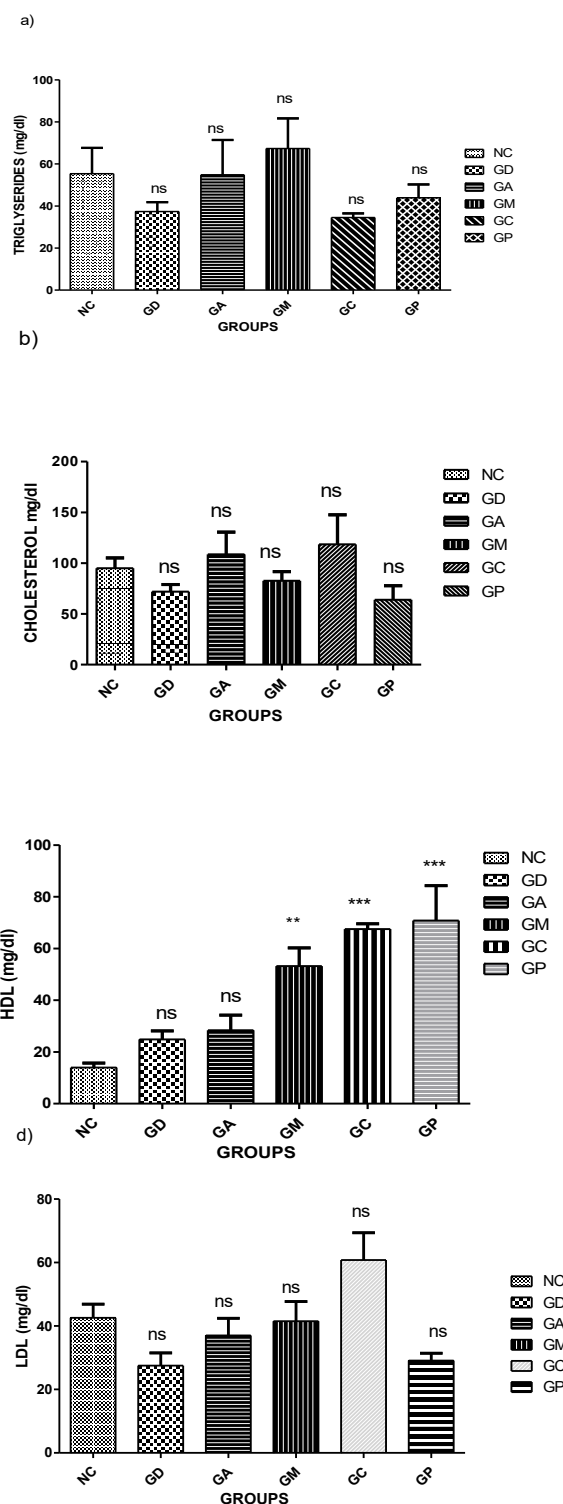


43.86. (fig. 6a). There was found no significant change in cholesterol levels among the experimental groups compared to the normal control one (fig. 6b). However, the serum HDL level is significantly in high animals of Group-IV, Group-V and VI compared to animals of Group-I i.e. normal control ones (Fig. 6c). The serum LDL level in the animals of group-I was around 100 mg/dl. Group-II, IV and VI exhibited slightly lower LDL levels and rats of Group-III and V had slightly elevated levels. Comparing to HDL/LDL and HDL/cholesterol ratios (Table 3.), there was found mean value around  $0.33 \pm 0.07$  and  $0.15 \pm 0.05$  in Group-I, whereas all the high fat diet fed rats showed increase i.e.  $0.97 \pm 0.28$  and  $0.36 \pm 0.14$  in Group-II,  $0.88 \pm 0.48$  and  $0.26 \pm 0.06$  in Group-III,  $1.41 \pm 0.51$  and  $0.65 \pm 0.15$  in Group-IV,  $1.19 \pm 0.33$  and  $0.69 \pm 0.28$  in group-V,  $2.51 \pm 0.92$  and  $1.45 \pm 0.95$  in group-VI . The increasing pattern of HDL/LDL and HDL/cholesterol ratios in all the high fat diet groups (significant in Group IV and VI) advocates dysfunction of lipid metabolism. The exact reason for these changes may not be explainable at the moment except when a high-fat diet is consumed, even when it contains saturated fats, HDL (reverse cholesterol transport) can improve the outflow of cholesterol from tissues back to the liver.

**Table 3. Means and standard deviations of HDL/LDL ratio and HDL/ Cholesterol ratio.**

GROUP	HDL/LDL RATIO	HDL/CHOLESTEROL RATIO
	Mean $\pm$ SD	Mean $\pm$ SD
Group I	$0.33 \pm 0.07$	$0.15 \pm 0.05$
Group II	$0.97 \pm 0.28$	$0.36 \pm 0.14$
Group III	$0.88 \pm 0.48$	$0.26 \pm 0.06$
Group IV	$1.41 \pm 0.51^*$	$0.65 \pm 0.15^*$
Group V	$1.19 \pm 0.33$	$0.69 \pm 0.28$
Group VI	$2.51 \pm 0.92^{**}$	$1.45 \pm 0.95^*$

Each value is presented as mean  $\pm$  SD of 5 rats. Significance  $p < 0.05$



**Figure 5. Serum cholesterol, Serum triglycerides, LDL and HDL cholesterol levels in normal control group and High Fat Diet fed experimental groups**



## Discussion

The present study was an attempt to replace the expensive commercially available diets with handmade high-fat diets for inducing hyperlipidemia as well as type 2 diabetes in albino rats. High-fat diet induced hyperlipidemia and type 2 diabetes in albino rats closely resemble the human metabolic syndrome. Since several years animals with obesity, dyslipidemia, and insulin resistance have been modeled using diets high in fats and have been reviewed by Kurhaluk et al., (2025). According to earlier studies, the local high-fat diet may change lipid and glucose metabolism, likely by altering insulin functions, that cause obesity, dyslipidemia, and hyperglycemia associated with glucose intolerance (possibility of insulin resistance (Suryaningtyas et al., (2025). Shinji Ikemoto et al., reported that many factors influence oral glucose tolerance tests, including the absorption rate in the intestine, insulin secretion, glucose accumulation in muscle tissues, and glucose output from the liver. Mice fed with palm oil may have experienced insulin resistance; though, due to defective insulin function, similarly, palm oil having high-fat diet showed though not very promising results. The study by Buettner et al. (2006) demonstrated that animal lard can be recommended as one of the standard fats for creating a valid rat model of metabolic changes linked to obesity. This present study showed that animal lard, as a fat source, is not associated with metabolic disorders. These observations could be helpful for future research that aims to use homemade high-fat diets to induce metabolic syndrome in rats.

In this study, the purpose was to determine whether homemade high-fat meals may induce diabetes in albino rats, but no discernible changes in metabolism were found with any of prepared diets. The exact reason for this can be advocated at this moment that the animals were not eating properly and because of less consumption, such changes occurred. The findings emphasize the significance of choosing the right fat sources and compositions, adding some tasty substances in the diet for inducing metabolic changes.

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## Conflict of Interest

The authors of this paper affirm that they have no conflicts of interest with regard to its publication.

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