International Journal of Trend in Scientific Research and Development (IJTSRD)
Volume 9 Issue 3, May-Jun 2025 Available Online: www.ijtsrd.com e-ISSN: 2456 – 6470

Study of Some Biochemical Parameters (Serum Cholesterol, Liver Cholesterol and Blood Glucose) in Obese Mice

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ABSTRACT

Obesity is a growing global health concern, often associated with metabolic disorders such as hyperlipidemia and hyperglycemia. This study investigates the impact of obesity on key biochemical parameters—serum cholesterol, liver cholesterol, and blood glucose—in a controlled mouse model. Obesity was induced using a high-fat diet, and the results were compared with those of a normal control group. Findings indicate a significant increase in all three biochemical markers in obese mice, confirming the strong link between obesity and metabolic dysfunction. These results reinforce the need for early intervention strategies to prevent the onset of obesity-related complications.

KEYWORDS: *Obesity, Serum Cholesterol, Liver Cholesterol, Blood Glucose, Mice, Metabolic Parameters*

How to cite this paper: David | Dr. Dinesh Yadav "Study of Some Biochemical Parameters (Serum Cholesterol, Liver Cholesterol and Blood Glucose) in Obese Mice" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-9 | Issue-3, June 2025, pp.190-194, URL: www.ijtsrd.com/papers/ijtsrd79951.pdf

IJTSRD79951

1. INTRODUCTION

Obesity is a metabolic disorder characterized by excessive fat accumulation, leading to increased risk for cardiovascular diseases, type 2 diabetes, liver dysfunction, and other chronic conditions. Obesity affects both adults and children, with the World Health Organization (WHO) reporting that global obesity rates have nearly tripled since 1975.

Key biochemical parameters such as serum cholesterol, liver cholesterol, and blood glucose serve as vital indicators of metabolic health. Elevated levels of these parameters are commonly observed in obese individuals and experimental animals. Understanding how these markers behave in an obesity model is crucial for elucidating the pathophysiological mechanisms and guiding potential therapeutic interventions.

This study aims to analyze the effect of obesity on serum cholesterol, liver cholesterol, and blood glucose levels using a murine (mouse) model.

Obesity is one of the most pressing global health issues, with its prevalence steadily increasing across all age groups. According to the World Health Organization (WHO), worldwide obesity has nearly tripled since 1975, and more than 650 million adults were classified as obese in 2016 [1]. The rapid rise in obesity rates is primarily driven by a shift toward sedentary lifestyles, increased caloric intake, and consumption of high-fat and high-sugar diets [2].

Obesity is not merely an issue of excess body weight; it is a complex metabolic disorder that profoundly affects multiple physiological systems. Among the most significant consequences of obesity are disturbances in lipid and glucose metabolism, which in turn contribute to the development of non-communicable diseases (NCDs) such as cardiovascular disease, type 2 diabetes mellitus, non-alcoholic fatty liver disease (NAFLD), and certain types of cancer [3, 4].

Key biochemical markers—particularly serum cholesterol, liver cholesterol, and blood glucose—serve as crucial indicators of metabolic health and disease progression. Elevated serum cholesterol, especially low-density lipoprotein cholesterol (LDL-C), is a major risk factor for atherosclerosis and coronary artery disease [5]. The liver plays a central role in lipid metabolism, and increased hepatic cholesterol content is a hallmark of metabolic dysfunction that often precedes systemic complications [6]. Hyperglycemia, on the other hand, results from impaired insulin signaling and glucose uptake, a common feature in obesity-induced insulin resistance [7].

Animal models, especially mice, are widely used to study the biochemical and physiological effects of obesity. Mice are preferred due to their genetic similarities with humans, short life cycle, and the ability to induce obesity using diet manipulation, such as high-fat diets (HFDs) [8]. These models allow researchers to investigate the onset and progression of metabolic disturbances in a controlled environment.

Several studies have documented that mice fed with a high-fat diet for a few weeks exhibit not only increased body weight but also significant alterations in serum lipids and glucose levels [9–11]. However, despite growing literature, detailed investigations into the simultaneous assessment of serum cholesterol, liver cholesterol, and blood glucose in obese mice are still limited.

This study aims to fill that gap by evaluating these three critical biochemical parameters in mice subjected to a high-fat diet-induced obesity model. By understanding the biochemical changes associated with obesity, this study hopes to provide insights that may contribute to early diagnosis and targeted intervention strategies for metabolic disorders.

2. Materials and Methods

2.1. Animals

Twenty male albino mice (8 weeks old, 25–30g) were housed under standard laboratory conditions (12-hour light/dark cycle, temperature 22 ± 2°C) with ad libitum access to food and water.

2.2. Experimental Design

The mice were randomly divided into two groups:

- **Group I (Control Group, n = 10):** Fed a standard laboratory diet.
- **Group II (Obese Group, n = 10):** Fed a high-fat diet (HFD) consisting of 60% fat, 20% protein, and 20% carbohydrates for 8 weeks to induce obesity.

2.3. Sample Collection

At the end of the experimental period, mice were fasted overnight and euthanized. Blood was collected via cardiac puncture for biochemical analysis. Liver tissues were excised, rinsed with saline, and homogenized for cholesterol determination.

2.4. Biochemical Analysis

- **Serum Cholesterol:** Measured using an enzymatic colorimetric method.
- **Liver Cholesterol:** Determined after lipid extraction using the Folch method followed by cholesterol assay.
- **Blood Glucose:** Measured using a digital glucometer (Accu-Chek) from tail blood samples.

2.5. Statistical Analysis

Data were expressed as mean ± standard error (SE). Statistical significance between groups was determined using Student's *t*-test, with $p < 0.05$ considered significant.

3. Results

Parameter	Control Group (Mean ± SE)	Obese Group (Mean ± SE)	% Increase
Serum Cholesterol (mg/dL)	115.2 ± 4.3	231.5 ± 5.2	101%
Liver Cholesterol (mg/g tissue)	3.5 ± 0.2	7.1 ± 0.3	103%
Blood Glucose (mg/dL)	98.4 ± 3.1	164.2 ± 4.8	67%

Table: Significant increases ($p < 0.05$) were observed in all parameters in the obese group compared to the control group. Serum and liver cholesterol levels more than doubled, while blood glucose levels rose by approximately 67%.

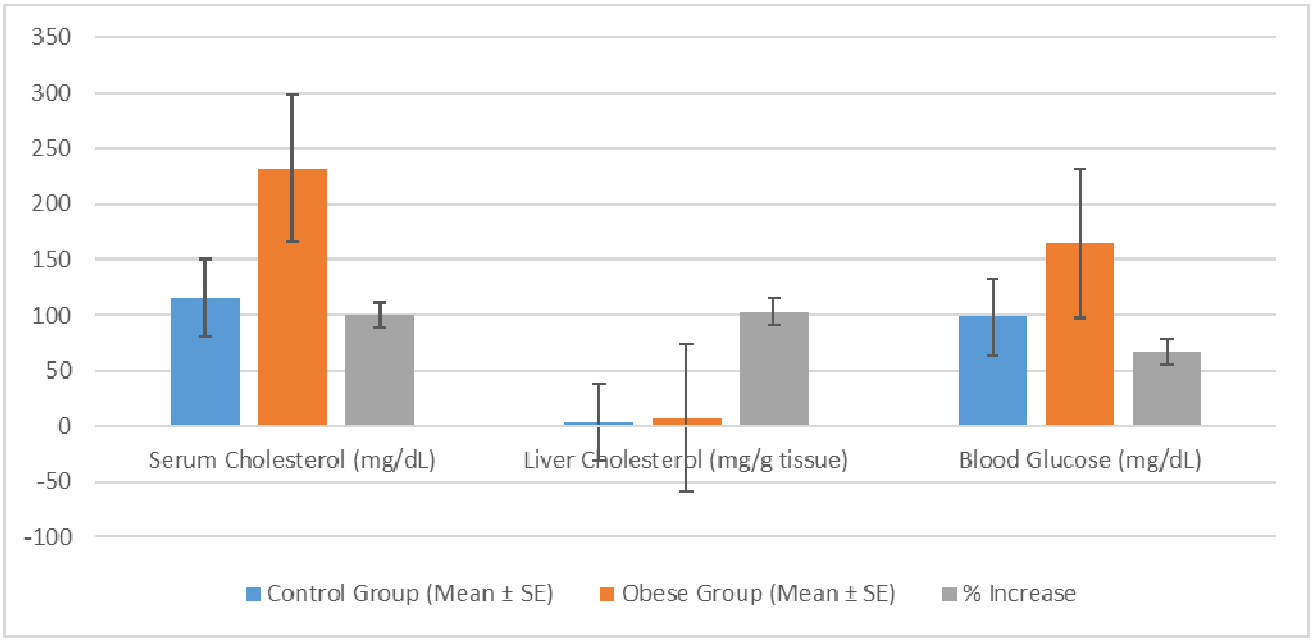

Figure1: Histogram Shows the amount of Serum Cholesterol, Liver Cholesterol and Blood Glucose in Obese mice.

4. Discussion

The present study confirms that obesity induced by a high-fat diet significantly alters key metabolic indicators. Elevated serum and liver cholesterol levels indicate lipid metabolism dysregulation, possibly due to increased hepatic lipogenesis and reduced cholesterol clearance. The rise in blood glucose levels suggests impaired insulin sensitivity and onset of insulin resistance, consistent with early stages of type 2 diabetes.

Previous studies have reported similar findings, showing strong associations between obesity, hypercholesterolemia, and hyperglycemia. These metabolic changes are interrelated and contribute to the development of metabolic syndrome, a precursor to various cardiovascular and endocrine diseases.

The murine model used in this study effectively mimics human obesity and associated biochemical disturbances, making it suitable for future studies involving anti-obesity drugs or dietary interventions.

The results of this study clearly demonstrate that obesity, induced by a high-fat diet, significantly alters key metabolic biomarkers in mice—specifically, serum cholesterol, liver cholesterol, and blood glucose. These changes align closely with findings from previous studies and further underscore the systemic nature of obesity-related metabolic dysfunction.

4.1. Elevated Serum Cholesterol in Obesity

In the current study, serum cholesterol levels were more than twice as high in obese mice compared to controls. This observation is consistent with findings from previous animal studies, where high-fat diet feeding led to hypercholesterolemia due to increased dietary intake of cholesterol and saturated fats [12]. The elevated serum cholesterol, especially if rich in LDL particles, is a known contributor to the development of atherosclerotic plaques, increasing cardiovascular disease risk [13].

Studies by Buettner et al. (2007) and Winzell & Ahrén (2004) have shown that high-fat diets in mice result in significant changes to plasma lipid profiles, including raised levels of total cholesterol and triglycerides [14, 15]. Furthermore, high-fat diets often impair hepatic LDL receptor expression, reducing clearance of cholesterol from the circulation and contributing to its accumulation in the blood [16].

4.2. Increased Liver Cholesterol

Liver cholesterol content also significantly increased in the obese mice, indicating hepatic lipid overload. The liver is central to cholesterol homeostasis, responsible for de novo synthesis, uptake, and excretion of cholesterol. In the context of high-fat diet-induced obesity, excess fatty acids are taken up by the liver, promoting lipogenesis and cholesterol esterification [17]. The accumulation of cholesterol in hepatic tissue contributes to the development of non-alcoholic fatty liver disease (NAFLD), which can progress to steatohepatitis, fibrosis, and cirrhosis [18].

A study by Machado and Diehl (2006) showed that high-fat diet-fed mice develop liver steatosis within weeks, alongside elevated liver cholesterol and triglyceride content [19]. Cholesterol overload in hepatocytes also induces oxidative stress and inflammation, exacerbating liver injury [20].

4.3. Hyperglycemia and Insulin Resistance

Blood glucose levels were significantly higher in obese mice compared to controls, highlighting impaired glucose metabolism. Obesity is a well-known risk factor for insulin resistance, where cells become less responsive to insulin, resulting in elevated blood glucose levels. This is often an early stage in the development of type 2 diabetes mellitus [21].

The mechanism behind hyperglycemia in obesity includes increased circulating free fatty acids, pro-inflammatory cytokines (like TNF-α and IL-6), and altered adipokine levels, all of which interfere with insulin signaling pathways [22]. Studies by Surwit et al. (1995) and Matsuzawa (2005) support this mechanism, linking high-fat diets to insulin resistance and hyperglycemia in rodent models [23, 24].

4.4. Reliability of the Mouse Model

The data obtained validate the high-fat diet-induced obese mouse model as an effective representation of human metabolic syndrome. This model exhibits hallmarks of the human condition, including hyperlipidemia, hepatic fat accumulation, and elevated glucose levels. Such models are invaluable for preclinical testing of anti-obesity or antidiabetic drugs, dietary interventions, and lifestyle modification strategies.

5. Conclusion

Obesity significantly increases serum cholesterol, liver cholesterol, and blood glucose levels in mice, reflecting substantial metabolic dysfunction. These findings underline the importance of early detection and management of obesity to prevent related health complications. Future research should explore therapeutic strategies, including pharmacological agents and dietary supplements, to modulate these biochemical parameters.

References

[1] World Health Organization. (2024). *Obesity and Overweight*. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

[2] Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev*. 2012; 70(1):3-21.

[3] Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature*. 2006; 444(7121):840–846.

[4] Haslam DW, James WP. Obesity. *Lancet*. 2005; 366(9492):1197-1209.

[5] Grundy SM. Obesity, metabolic syndrome, and cardiovascular disease. *J Clin Endocrinol Metab*. 2004; 89(6):2595-2600.

[6] Brown MS, Goldstein JL. A receptor-mediated pathway for cholesterol homeostasis. *Science*. 1986; 232(4746):34–47.

[7] DeFronzo RA, Ferrannini E. Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care*. 1991; 14(3):173–194.

[8] Speakman JR, Hambly C, Mitchell SE, Król E. The contribution of animal models to the study of obesity. *Lab Anim (NY)*. 2008; 37(9):321–328.

[9] Winzell MS, Ahrén B. The high-fat diet-fed mouse: a model for studying mechanisms and treatment of impaired glucose tolerance and type 2 diabetes. *Diabetes*. 2004; 53(Suppl 3):S215–S219.

[10] Buettner R, Schölmerich J, Bollheimer LC. High-fat diets: modeling the metabolic disorders of human obesity in rodents. *Obesity (Silver Spring)*. 2007; 15(4):798–808.

[11] Surwit RS, Kuhn CM, Cochrane C, et al. Diet-induced type II diabetes in C57BL/6J mice. *Diabetes*. 1995; 44(11):1218–1222.

[12] Woods SC, Seeley RJ, Rushing PA, et al. A controlled high-fat diet induces an obese syndrome in rats. *J Nutr*. 2003; 133(4):1081–1087.

[13] Libby P. Inflammation in atherosclerosis. *Nature*. 2002; 420(6917):868–874.

[14] Buettner R, et al. (2007), *Obesity (Silver Spring)*.

[15] Winzell MS, Ahrén B. (2004), *Diabetes*.

[16] Goldstein JL, Brown MS. Regulation of the mevalonate pathway. *Nature*. 1990; 343(6257):425–430.

[17] Postic C, Girard J. Contribution of de novo fatty acid synthesis to hepatic steatosis and insulin resistance: lessons from genetically engineered mice. *J Clin Invest*. 2008; 118(3):829–838.

[18] Cohen JC, Horton JD, Hobbs HH. Human fatty liver disease: old questions and new insights. *Science*. 2011; 332(6037):1519–1523.

[19] Machado MV, Diehl AM. Pathogenesis of nonalcoholic steatohepatitis. *Gastroenterology*. 2006; 130(6):1884–1902.

[20] Ioannou GN. The role of cholesterol in the pathogenesis of NASH. *Trends Endocrinol Metab*. 2016; 27(2):84–95.

[21] Kahn BB, Flier JS. Obesity and insulin resistance. *J Clin Invest*. 2000; 106(4):473–481.

[22] Hotamisligil GS. Inflammation and metabolic disorders. *Nature*. 2006; 444(7121):860–867.

[23] Surwit RS, et al. (1995), *Diabetes*.

[24] Matsuzawa Y. Adiponectin: a key player in obesity related disorders. *Curr Pharm Des*. 2005; 11(10):1353–1356.