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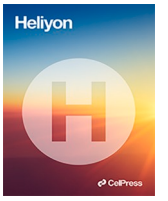

Research article

Comparative analysis of high-fat diets: Effects of mutton, beef, and vegetable fats on body weight, biochemical profiles, and liver histology in mice

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ABSTRACT

Background: High-fat diets are associated with metabolic syndrome, cardiovascular diseases, and liver disorders. Beef and mutton, both widely consumed meats, are significant sources of animal fat, while soybean oil, a commonly used cooking oil, is a prominent source of plant-derived fat. This study aimed to compare the effects of regular consumption of beef fat, mutton fat, and soybean oil in mice to assess potential health risks.

Methods: Sixty Swiss albino male mice were divided into four groups: a control group (Group A) fed a standard mice pellet, and three treatment groups (Groups B, C, D) receiving 10 % dietary fat from mutton, beef, and soybean oil, respectively. Parameters such as body weight, caloric intake, serum markers, and liver histopathology were studied.

Results: Consumption of mutton fat, beef fat, or soybean oil supplemented diet in groups B, C, and D led to higher caloric intake and body weight compared to control group A, which received a standard diet. These diets also caused elevated serum glucose, impaired glucose tolerance, and increased triglycerides, cholesterol, LDL-C, and reduced HDL-C. Elevated AST and ALT levels in the high-fat diet groups, indicated liver damage and fat accumulation. Histological analysis confirmed steatosis, hepatocyte ballooning, and inflammation in all three high-fat diet groups, while the control group had normal liver histology.

Conclusion: High-fat diets, whether plant- or animal-based, led to weight gain in mice and resulted, poor glucose tolerance, dyslipidemia, liver damage and steatohepatitis. Further research is needed to explore the mechanisms behind these effects and improve understanding and management of high-fat diet consequences.

1. Introduction

Fats play a pivotal role in human nutrition, serving as essential components for energy balance, vitamin absorption, and insulation. Essential fatty acids, crucial for various bodily functions, are derived from sources such as vegetable oils, dairy products, meat, grains,

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fish, and fish oil [1]. The composition of fatty acids varies among these sources, particularly in polyunsaturated fatty acids (PUFA), which are classified into omega-3 and omega-6 families.

While PUFA, especially omega-3 and omega-6, are essential, meats like beef and mutton contain higher proportions of saturated fatty acids (SFA) compared to PUFA [2,3]. The saturated fat content in beef and mutton exceeds 40 %. Conversely, soybean oil, a plant-based source, is rich in PUFA [4,5].

High-fat diets have been associated with various health concerns, including metabolic syndrome, cardiovascular diseases, and liver disorders [6–8]. In Bangladesh, a developing nation experiencing rapid urbanization, the prevalence of chronic diseases is rising [9, 10]. This surge can be attributed to shifts in dietary patterns, increased access to unhealthy food choices, irregular eating habits, reduced physical activity, and elevated stress levels [10,11]. The prevalence of metabolic syndrome in Bangladesh is 32,000 per 100, 000 females and 25,000 per 100,000 [12]. Additionally, the incidence of cardiovascular disease (CVD) is reported at 8,000 per 100,000 in urban areas and 2,000 per 100,000 in rural regions [12]. Furthermore, hypertension affects 13,700 per 100,000 of the population, with a corresponding prevalence of 6,700 per 100,000 for type-II diabetes in Bangladesh [13]. Urban areas exhibit higher rates of these conditions compared to rural areas.

A range of health conditions, including obesity, insulin resistance, hypertension, and dyslipidemia, impact 20–40 % of the population in developing countries, and this trend is on the rise [14]. Obesity, closely linked to high-fat diet consumption, underscores the pivotal role of dietary fat, cholesterol, and fatty acids in dyslipidemia [6]. Unhealthy food habits are primary contributors to conditions resembling metabolic syndrome. In Bangladesh, beef and mutton are common meat choices, while soybean oil is predominantly used for cooking [15–17]. These dietary preferences can significantly influence population health outcomes. Limited information exists on the comparative effects of high-fat diets based on soybean oil, mutton fat, and beef fat under Bangladeshi conditions using mice models. This research aims to fill this gap by investigating the impacts of these high-fat diets on body weight, lipid profiles, blood glucose, glucose tolerance, and liver histopathology in male mice.

2. Materials and methods

2.1. Experimental mice

Sixty male Swiss Albino mice (*Mus musculus*), aged five weeks and weighing 25 ± 2 g, were obtained from the International Centre for Diarrheal Disease Research, Bangladesh (ICDDR'B), Dhaka. Upon acquisition, the mice underwent a two-week acclimatization period in a well-ventilated laboratory maintained at 28 ± 2 °C with 70–80 % humidity and a 12-h light/dark cycle. During the acclimatization period, the mice were provided with ad libitum access to water and a standard pellet diet (5 g per mouse per day) supplied by ICDDR'B. The study consisted of four experimental groups, and each experiment was conducted in biological triplicates. In each replicate, five mice were assigned to each group, resulting in a total of fifteen mice per group across the three replicates, with sixty mice used in total.

2.2. Diets

The standard ICDDR'B mice pellet consisted of 53.85 % carbohydrates, 19.7 % protein, 15.75 % fat, 4.2 % fiber, and 6.5 % ash, providing an energy content of 444.35 kcal per 100 g. To prepare the experimental diets used in this study, fresh beef and mutton fat, excluding any meat, were sourced from the local KR market and subsequently heated to extract the oils to facilitate the mixing process. Rupchanda fortified soybean oil (Bangladesh Edible Oil Limited) was purchased commercially. The extracted mutton and beef fat oils, along with the soybean oil, each provided an energy content of 9 kcal/ml. These oils were then blended with the standard mice pellets to formulate the experimental diets as follows:

- Group A (Control): Standard mice pellet (100 %), providing 444.35 kcal/100 g.
- Group B: Standard mice pellet (90 %) + 10 % mutton fat oil by weight, providing 490.5 kcal/100 g.
- Group C: Standard mice pellet (90 %) + 10 % beef fat oil by weight, providing 490.5 kcal/100 g.
- Group D: Standard mice pellet (90 %) + 10 % soybean oil by weight, providing 490.5 kcal/100 g.

To prevent rancidity and maintain the palatability of the experimental diets, they were prepared twice weekly, packaged in small airtight zipper bags (25 g per bag), and stored in a refrigerator at 4 °C until use.

2.2. Ethical approval

The research methodology adhered to the guidelines set by the Animal Welfare and Experimentation Ethics Committee (AWEEC) of Bangladesh Agricultural University, Mymensingh, Bangladesh [AWEEC/BAU/2022-84].

2.3. Blood sample collection

After the 10-week experimental period was completed, each mouse was placed in a desiccator saturated with cotton soaked in diethyl ether to induce unconsciousness, facilitating the sacrificial process. Subsequently, the abdominal and thoracic cavities were meticulously opened, and blood samples were directly collected from the heart using a 3 ml syringe.

2.4. Preparation of serum for biochemical analysis

Following the collection of blood from the heart, samples were transferred into the Eppendorf tubes without any anticoagulant and allowed to stand at room temperature, inclined at approximately 45°, for 2 h. Subsequently, the Eppendorf tubes were refrigerated overnight. The resultant serum was carefully separated and transferred into fresh Eppendorf tubes, after which it underwent centrifugation to eliminate excess cells. This centrifugation process was conducted using the Wise Spin R centrifuge machine, Model- VM 10, Daihan Scientific Co., Ltd, Korea, employing low-speed settings at 2000 revolutions per minute (rpm) for 20 min. Following centrifugation, the serum was transferred to another fresh tube and stored at freezing temperatures until subsequent analysis.

2.5. Biochemical tests

Serum concentrations of cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) were measured using a Cobas C311 biochemistry analyzer (Roche®, Switzerland), following manufacturer's instruction as described in published research [18].

2.6. Intraperitoneal glucose tolerance test (ipGTT)

The IPGTT was conducted following established protocols [19–21].After a 12-h fast, animals were weighed, and baseline blood glucose levels were measured. A glucose solution was then injected directly into the peritoneal cavity at a dosage of 2 g per kilogram of body weight. Blood samples were collected at specified time points: 0, 30, 60, and 120 min post-injection, using tail vein puncture. Glucose concentrations in the samples were measured using the Finetest glucometer from OSANG Healthcare, Korea.

2.7. Sample collection for histopathology

For histological examination, the livers from the experimental mice were meticulously harvested. Each liver was carefully washed in a Petri dish using normal saline and subsequently transferred into an organ tube containing a 15 % formalin solution, with each specimen treated individually. The specimens were allowed to rest in ambient room conditions until the commencement of the histological procedures.

2.8. Sample preparation and staining of liver tissue

The liver tissues were processed in the laboratory following standard histological procedures. Initially, the tissues were separated, cut, and fixed in Bouin's fluid for 24 h. Subsequent dehydration was performed using ascending concentrations of alcohol (70 %, 80 %, 90 %, 95 %, 100 % I, 100 % II, and 100 % III), each for 2 h. The tissues were then cleared in three changes of xylene, followed by infiltration with paraffin. After embedding, 5-μm thick sections were cut using a sliding microtome, floated on a lukewarm water bath, and mounted on slides coated with egg albumin. The slides were dried for 12 h before staining. The sections were stained using Hematoxylin and Eosin (H&E), following the protocol described by Cardiff et al. [22]. Finally, the stained sections were examined under an Olympus photomicroscope (CX43) at 10× objective for histological analysis.

2.9. The non-alcoholic fatty liver disease activity score (NAS) for histological assessment of liver

NAS scoring was conducted following the protocol used in published work on mice [23,24]. This method assigns a score from 0 to 8 to histological hallmarks, allowing for the simultaneous evaluation of multiple independent features. Steatosis severity was scored based on the percentage of affected hepatocytes: 0 for less than 5 %, 1 for 5%–33 %, 2 for 34%–66 %, and 3 for more than 66 %. Lobular inflammation is scored as 0 for no foci, 1 for 1–2 foci per field, 2 for 2–4 foci per field, and 3 for more than 4 foci. Hepatocyte ballooning, indicating cell swelling, is scored as 0 for no ballooning, 1 for few ballooned cells, and 2 for many or prominent ballooning. These three scores were combined per slide to calculate NAS score, with five slides evaluated from each experimental group.

2.10. Statistical analysis

The data were presented as mean ± S.E. for each group, and statistical analyses were performed using GraphPad Prism Version 9.3.1 (GraphPad Software, San Diego, CA). A one-way ANOVA followed by Tukey's multiple comparisons test was employed to assess body weight, lipid profile, the area under the curve (AUC) for glucose, and liver function. For evaluating glucose tolerance, weekly calorie intake, and weekly feed intake, a two-way ANOVA followed by Tukey's multiple comparisons test was applied. A significance level of $P < 0.05$ was established as the criterion for statistical significance. Additionally, one-way ANOVA followed by Tukey's multiple comparisons test was utilized to evaluate hepatic steatosis, inflammation, hepatocyte ballooning, and NAS.

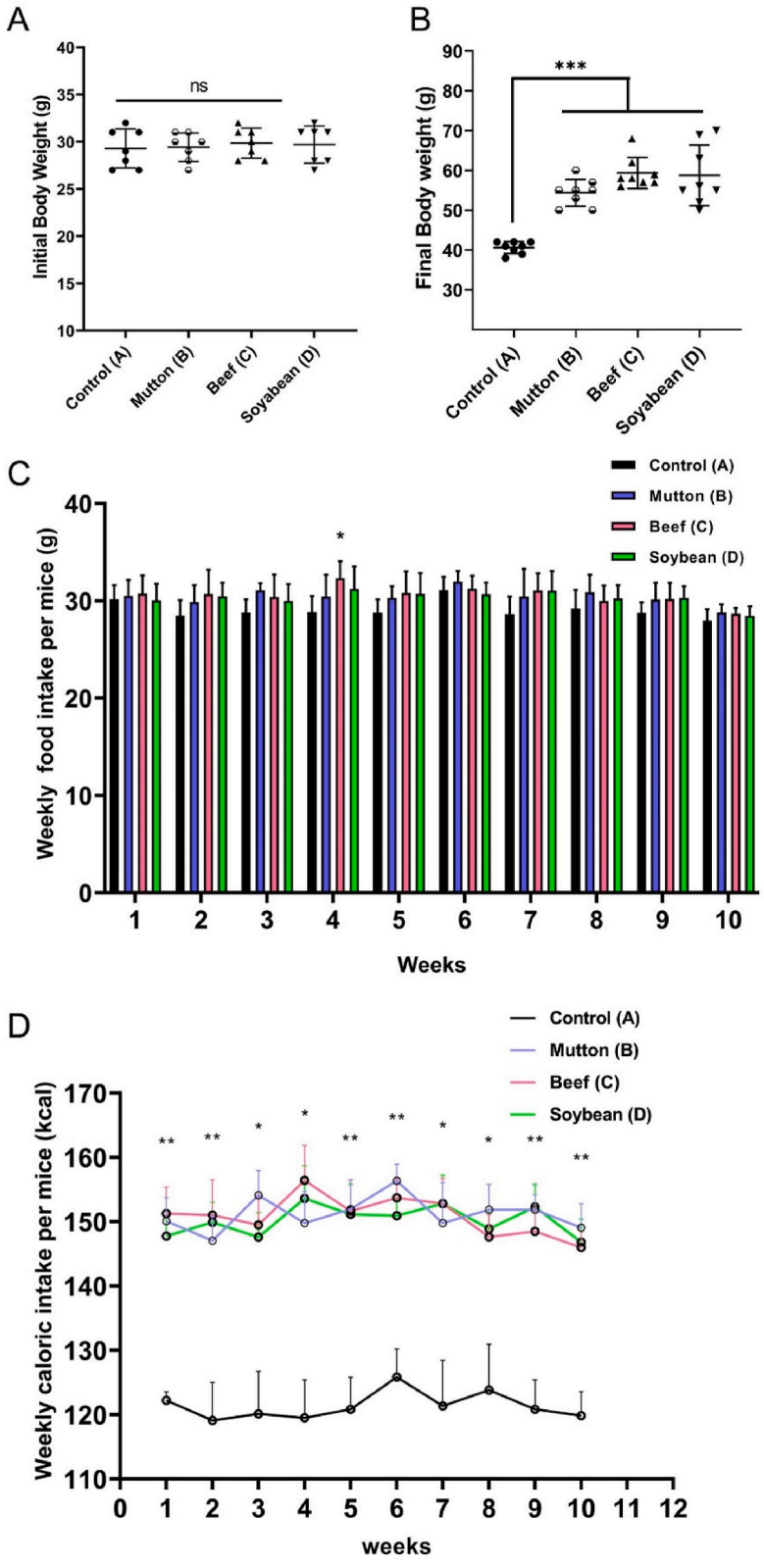

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Fig. 1. Body weight and dietary intake analysis in experimental groups
(A) The initial body weight of the experimental groups showed no significant differences, while the final body weight after 10 weeks on high-fat diets. (B) A significant increase in body weight compared to the control group is observed. Weekly food intake per mouse (C) was measured in grams across different groups throughout the experimental period, along with weekly calorie consumption per mouse (D) in kilocalories (kcal). Data are presented as mean ± SE, with statistical significance indicated by *P < 0.05, **P < 0.01, ***P < 0.001, and ns denoting statistically non-significant differences.

3. Results

3.1. Effects of high-fat diets on body weight

The experiment commenced with four distinct groups of mice, designated as Group A (control), Group B (mutton fat-mixed diet), Group C (beef fat-mixed diet), and Group D (soybean oil-mixed diet), all initially possessing comparable mean body weights of around 30 g (Fig. 1A). After a ten-week trial of various high-fat diets, the treatment groups (Groups B, C, and D) demonstrated a significant increase in body weight compared to the control group (Fig. 1B). We also evaluated whether there were any differences in weekly feed intake. The weekly feed intake (g) per mouse did not differ significantly between the beef fat, mutton fat, and soybean-supplemented groups and the control group (Fig. 1C). However, there was a significant difference in weekly calorie intake per mouse throughout the experiment (Fig. 1D), attributed to the additional calories provided by the beef fat, mutton fat, and soybean oil diets compared to the control group, which received a standard diet. The weekly calorie intake remained consistent among the high-fat diet groups.

3.2. Effects of high-fat diets on blood glucose and glucose tolerance

Serum glucose concentrations are depicted in Fig. 2A. Mice on high-fat diets had significantly elevated serum glucose levels ($P < 0.001$) compared to controls, with no significant differences among the high-fat diet groups. Subsequently, we postulated that high-fat diets might disrupt normal glucose metabolism in the experimental groups. To investigate this hypothesis, we conducted an intraperitoneal glucose tolerance test (ipGTT) (Fig. 2B) and calculated the area under the curve (AUC) (Fig. 2C) to assess potential disturbances in glucose metabolism. Notably, glucose levels in all high-fat diet groups, measured at various time intervals following a 2 g/kg body weight intraperitoneal glucose administration after a 12-h fasting period, were significantly elevated ($P < 0.01$) compared to the control group. The AUC analysis further revealed a significantly higher ($P < 0.01$) mean value in all three fat-supplemented groups compared to the control (Fig. 2C). These findings strongly indicate that regular consumption of all three high-fat diets adversely impacts glucose tolerance.

3.3. Effects of high-fat diets on lipid profile

Fig. 3A illustrates the impact of different diets on the serum cholesterol levels of mice. Significant increases ($P < 0.001$) in cholesterol levels were observed in all treatment groups compared to the control. Notably, no significant differences were found in cholesterol levels among mice fed mutton fat-based, beef fat-based, and soybean oil-based diets.

Similarly, as shown in Fig. 3B, the mean triglyceride concentrations in all three treatment groups were significantly higher ($P < 0.001$) than in the control group. Fig. 3C demonstrates a significant decrease ($P < 0.05$) in HDL-C levels in groups B, C, and D compared to the control. For LDL-C levels, all three treated groups exhibited significantly higher ($P < 0.05$) levels compared to the control, although the soybean oil group had the lowest concentration among the treated groups, as depicted in Fig. 3D.

3.4. Effects of high-fat diets on liver function

To evaluate the impact of high-fat diets on liver function, AST and ALT levels were measured, as shown in Fig. 4. The mean ALT value was highest in the soybean fat group, followed by the beef and mutton fat groups, while the control group exhibited significantly lower ALT values ($P < 0.001$), as depicted in Fig. 4A. In Fig. 4B, the beef fat group had the highest AST level, closely followed by the mutton fat and soybean fat groups. Mean AST values increased significantly ($P < 0.01$) in the three high-fat diet groups compared to the control, with no significant differences among the high-fat diet groups.

3.5. Liver histopathology

Following the observation of elevated AST and ALT values in the high-fat diet groups, we conducted liver histopathology to investigate potential histological alterations. The histopathological analysis of liver tissues is depicted in Fig. 5. Hematoxylin and eosin (H&E) stained sections (Fig. 5A–D) were examined to assess the extent of fat accumulation using steatosis scoring (Fig. 5E), which revealed a significant increase ($P < 0.001$) in lipid droplet accumulation in all three high-fat diet groups compared to the control group. Furthermore, the number of inflammatory cell clusters per image was quantified across the different treatment groups to assign inflammation scores (Fig. 5F), revealing a significant difference ($P < 0.05$) between the high-fat diet groups and the control. Hepatocellular hypertrophy was also assessed using hepatocyte ballooning scores (Fig. 5G), which indicated a significantly higher prevalence of ballooned hepatocytes in the mutton fat, beef fat, and soybean oil-supplemented groups compared to the control. These three

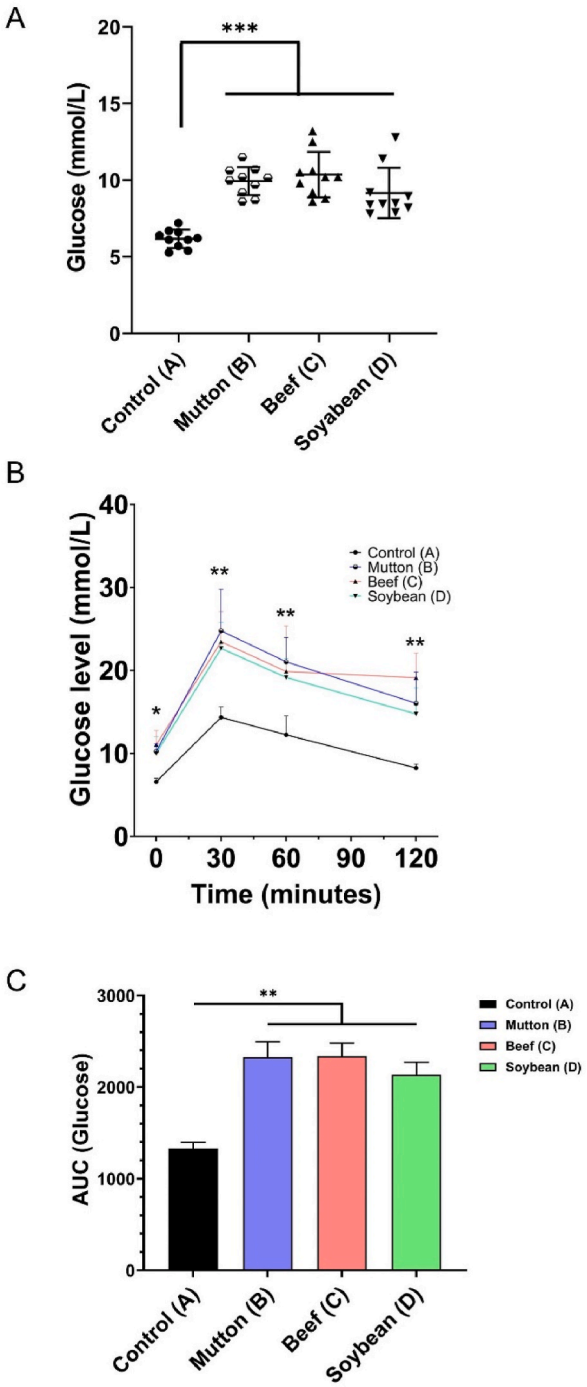

Fig. 2. Effects of high-fat diets on blood glucose and glucose tolerance. (A) Blood glucose levels across various experimental groups. (B) Glucose concentrations during intraperitoneal glucose tolerance test (ipGTT) at different time points for each group. (C) Area under the curve (AUC) analysis for all groups. Data are presented as mean ± SE. Significance is indicated by *P < 0.05, **P < 0.01, ***P < 0.001.

scores were then combined to calculate the NAS score for all groups (Fig. 5H). The significantly higher NAS scores ($P < 0.01$) in all three fat-supplemented groups strongly suggest that they exhibited mild to borderline nonalcoholic steatohepatitis (NASH), whereas the control group did not develop NASH.

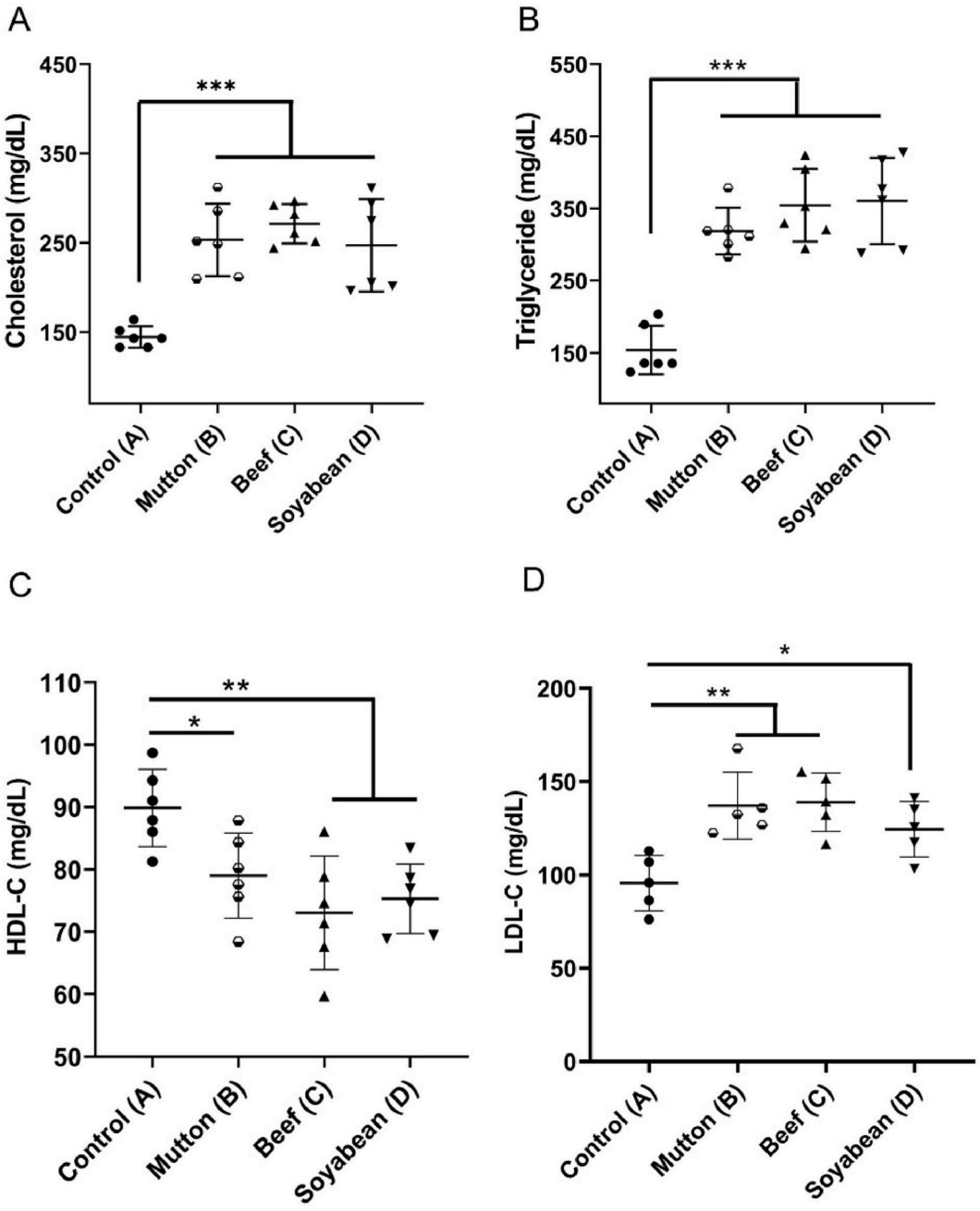

Fig. 3. Effects of high-fat diets on lipid profile. (A) Serum cholesterol levels across different groups. (B) Serum triglyceride levels across different groups. (C) HDL-C levels in the experimental groups. (D) LDL-C levels in the experimental groups. Data are presented as mean ± SE. Significance is indicated by *P < 0.05, **P < 0.01, ***P < 0.001.

4. Discussion

In this experiment, a 10-week feeding regimen of high-fat diets—both animal-based (beef, mutton) and plant-based (soybean)—resulted in a significantly higher average final body weight compared to the control group. This outcome was anticipated, as increased fat consumption does not stimulate fat oxidation, which relies on sympathetic nervous system (SNS) activation [25,26]. We observed a significant difference in calorie intake between all three high-fat diet groups and the control group, despite similar feed intake across all groups. The additional 10 % fat in the high-fat diets provided more calories, leading to increased body weight as the excess energy was stored as fat in various tissues, contributing to obesity [27,28]. These findings are consistent with previous studies [29,30], which also documented significant weight gain in mice fed high-fat diets. Both beef and mutton fats are rich in saturated fatty acids (SFAs),

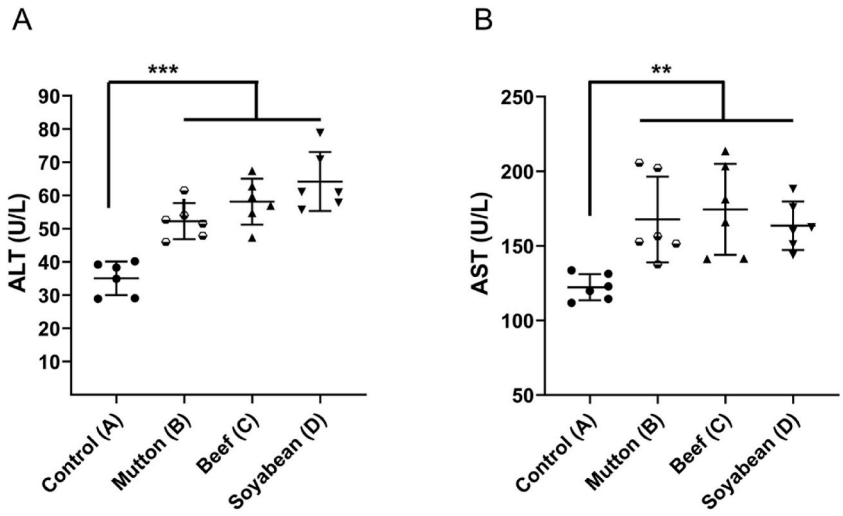

Fig. 4. Effects of high-fat diets on liver function. (A) shows ALT and (B) shows AST concentrations in different groups. Both ALT and AST values increased significantly in all high-fat groups compared to the control. Data are presented as mean ± SE. **P < 0.01, ***P < 0.001.

particularly palmitic and stearic acids [31]. These acids are associated with increased body weight due to their high caloric density and their ability to promote fat storage. In contrast, soybean oil is primarily composed of polyunsaturated fatty acids (PUFAs), mainly linoleic and oleic acids [31]. Although PUFAs are generally linked to a lower risk of weight gain, an imbalance between oleic and linoleic acid content could still promote fat storage, leading to weight gain [32]. Therefore, it can be inferred that regular consumption of diets enriched with beef fat, mutton fat, or soybean oil may contribute to obesity in mice.

We also observed significantly elevated serum glucose levels ($P < 0.05$) in mice fed high-fat diets from mutton fat, beef fat, and soybean oil compared to the control group. This aligns with findings from Winzell and Ahren [33] and Huang et al. [34], who also reported increased blood glucose levels in mice on high-fat diets. An intraperitoneal glucose tolerance test (ipGTT) confirmed that glucose levels remained elevated across all high-fat diet groups, regardless of fat source, at different time points tested compared to controls. These elevated glucose levels suggest impaired glucose clearance, indicative of conditions such as diabetes, metabolic syndrome, or impaired glucose tolerance. High-fat diets can reduce glucose uptake in adipocytes, contributing to elevated blood glucose levels [35]. High fat intake may also increase free fatty acids (FFAs), which reduce GLUT4 translocation, impairing insulin sensitivity and raising plasma glucose concentrations [36]. Additionally, high-fat diets increase fasting hepatic glucose production [37] and induce insulin resistance in muscles and the liver due to elevated FFAs [38]. Although the polyunsaturated fatty acids (PUFAs) in soybean oil have the potential to improve glucose tolerance by enhancing insulin sensitivity, the poor glucose tolerance observed in our study following a soybean oil-supplemented diet could be attributed to the excessive intake of omega-6 relative to omega-3 [39]. This imbalance may trigger inflammatory responses and activate the endoplasmic reticulum (ER) stress pathway, ultimately leading to insulin resistance and elevated blood glucose levels [40].

In our experiment, treated groups had significantly higher triglycerides, total cholesterol, and LDL, and lower HDL levels compared to the control group. These findings align with studies on mice fed high-fat diets [41,42]. Dietary cholesterol, including animal fats and vegetable oils, has been associated with elevated cholesterol levels [43,44]. Other studies [41,42] also reported elevated serum triglycerides due to excess calorie intake and the conversion of fatty acids into triglycerides [38,45].

For HDL-C, some studies observed an increase [41,46], while others noted a decrease [42]. In our study, the reduced HDL-C levels may be attributed to high-fat diets altering lipoprotein metabolism, which reduces HDL-C clearance and affects cholesterol transport [47].

Our findings of increased LDL-C are consistent with [48] but contrast with [46]. High-fat diets enhance LDL synthesis, upregulate cholesterol biosynthesis genes, and promote, and modulate enzymes like HMG-CoA reductase, increasing LDL levels and atherosclerosis risk [49]. Further investigation is required whether these mechanisms are involved in our experiment.

High levels of ALT and AST observed in response to high-fat diet consumption in our study suggest potential liver dysfunction and metabolic disturbances. High-fat diets, especially those abundant in saturated fats, have been linked to the development of hepatic steatosis, characterized by the accumulation of triglycerides within liver cells. This build-up of lipids can impair liver function and lead to hepatocellular injury, consequently releasing ALT and AST into the bloodstream [50,51]. Furthermore, high-fat diets may exacerbate oxidative stress and inflammation in the liver [52–54], further contributing to hepatocellular damage and elevated transaminase levels.

We observed borderline non-alcoholic steatohepatitis in all three high-fat groups. The observed histological changes in the liver tissues of mice fed high-fat diets can be attributed to several underlying mechanisms. SFAs present in beef and mutton fat can promote excessive triglyceride accumulation within hepatocytes, leading to hepatic steatosis [28,50]. This fat accumulation impairs liver function and contributes to hepatocellular necrosis and inflammatory changes [55,56], as evidenced by histopathological sections and

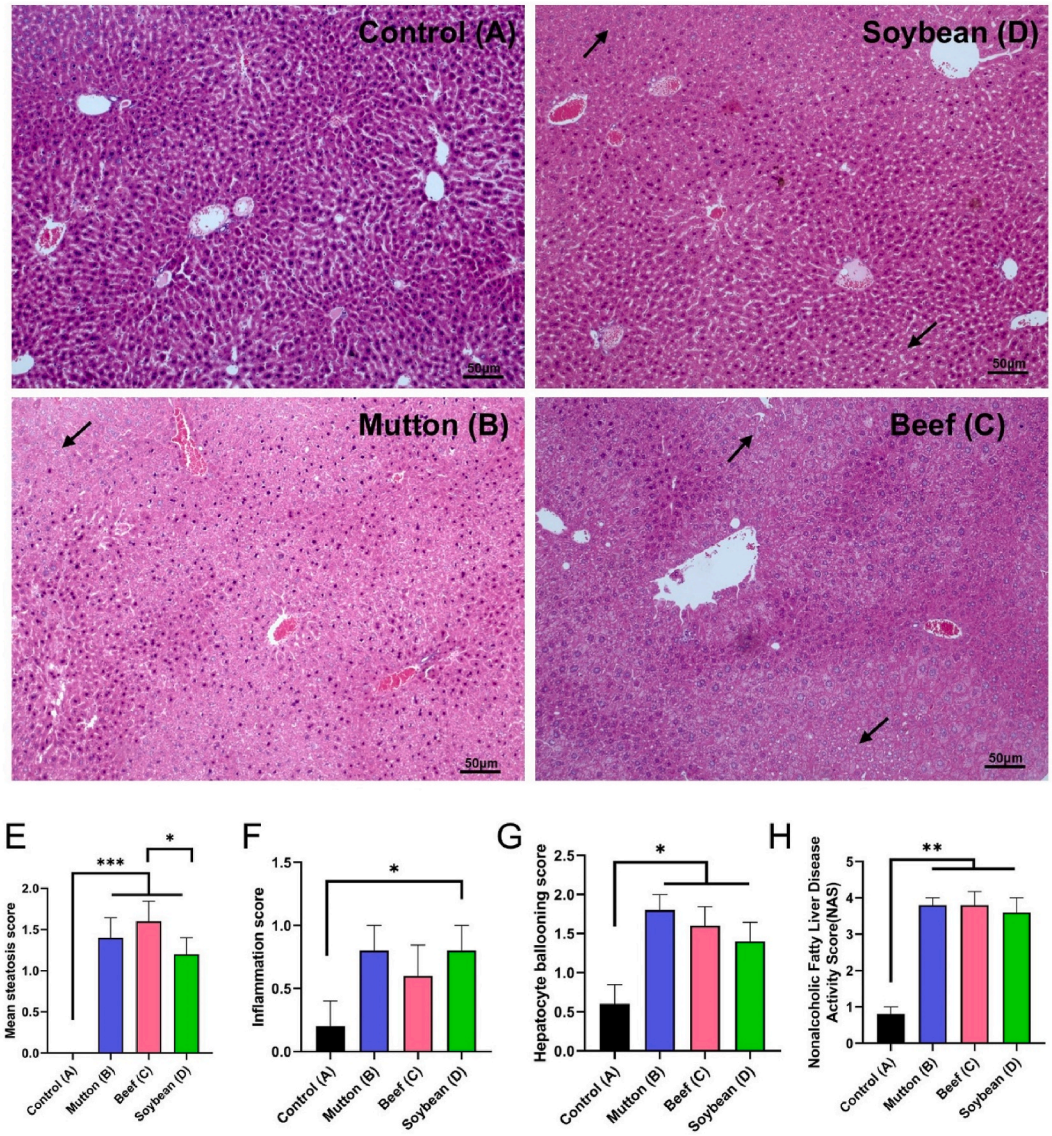

Fig. 5. Histopathological alterations in the liver following high-fat diet consumption. Representative liver tissue sections stained with Hematoxylin and Eosin. Images were captured using a 10× objective lens. (A) The control group exhibits normal liver morphology with no visible lesions. (B) The mutton-fat group, (C) the beef-fat group, and (D) the soybean-fat group all demonstrate the presence of fat droplets (indicated by arrows) and hepatocyte hypertrophy. Images were captured using a 10× objective lens. (E) The mean steatosis score for each group was quantified based on fat droplet accumulation per image. (F) The inflammation score was determined by counting the number of inflammatory cell clusters per image. (G) The extent of hepatocellular ballooning was assessed by counting enlarged hepatocytes per figure in each group (H) The NAS score was calculated by summing the steatosis, inflammation, and hepatocellular ballooning scores for each image.
Data are presented as mean ± SE. Statistical significance is denoted as follows: *P < 0.05, **P < 0.01, ***P < 0.001.

inflammation scores in our experiment. Although some studies have reported that the omega-6 fatty acids present in soybean oil can reduce liver fat accumulation by enhancing lipid oxidation, others have indicated that soybean oil supplementation may cause inflammation and liver damage [31].

Overall, these histological changes, coupled with elevated ALT and AST levels, impaired lipid profiles, and altered glucose metabolism, underscore the intricate relationship between dietary fat intake, metabolic pathways, and liver pathology. However, our study has certain limitations. While our findings provide substantial evidence that regular consumption of high-fat diets, whether of animal or plant origin, can lead to weight gain, impaired glucose tolerance, and dyslipidemia, along with compromised liver health, the study does not explore the underlying mechanisms in detail.

Future research should focus on investigating whether supplementation with beef fat, mutton fat, and soybean oil directly affects metabolism by examining liver-specific lipid metabolism genes and conducting a more comprehensive analysis of serum lipid profiles.

Additionally, it would be valuable to assess the long-term effects of these fat-enriched diets. Prolonged exposure may exacerbate the effects observed in our study, potentially leading to more pronounced metabolic disturbances, further deterioration in glucose tolerance, and more severe hepatic pathology, such as advanced fibrosis or steatosis. Chronic exposure may also reveal cumulative impacts on other metabolic pathways or systemic functions that were not apparent during the 10-week study period. Our research provides a foundation for future investigations into the long-term health implications of different dietary fats and suggests potential directions for future studies.

5. Conclusions

In conclusion, our study emphasises the detrimental impact of high-fat consumption, irrespective of its source, on various physiological parameters in mice. Notably, increased body weight and impaired glucose tolerance were consistently observed across all high-fat groups. Furthermore, adverse lipid profiles, manifested by lower HDL-C and higher LDL-C levels, were evident in high-fat cohorts. Alarmingly, hepatocellular damage, as indicated by elevated AST and ALP values, along with steatohepatitis, was observed across high-fat groups compared to control. These findings collectively emphasize the urgent need for dietary interventions to mitigate the escalating health risks posed by high-fat diets.

CRediT authorship contribution statement

Mst. Sharifa Jahan: Writing – review & editing, Methodology, Investigation, Data curation. **Md. Iqramul Haque:** Writing – original draft, Visualization, Methodology, Investigation, Formal analysis. **Manish Gautam:** Validation, Methodology, Investigation. **Mohammad Eliusur Rahman Bhuiyan:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Funding acquisition, Conceptualization.

Data availability statement

Has data associated with your study been deposited into a publicly available repository?
Response: No.
Please select why. Please note that this statement will be available alongside your article upon publication.
Response: Data will be made available on request.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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