



Cardiometabolic Effects of *Sus barbatus* Meat Consumption: Analysis of Blood Cholesterol Levels and Cardiac Histopathological Alterations in *Mus musculus*

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ABSTRACT

Purpose of the study: This study aims to determine the effects of pork (*Sus scrofa domestica*) and beef (*Bos taurus*) consumption on total cholesterol levels and the histopathological structure of the heart in male ICR mice (*Mus musculus*).

Methodology: This study employed an experimental laboratory design using a Completely Randomized Design (CRD). Fifteen male ICR mice (*Mus musculus*), aged 2–3 months and weighing 25–30 g, were divided into control, pork-fed (*Sus barbatus*), and beef-fed (*Bos taurus*) groups. After 30 days of treatment, blood samples were analyzed for total cholesterol levels, and heart tissues were examined histologically using the Hematoxylin-Eosin (HE) staining method. Data were analyzed statistically using SPSS.

Main Findings: Pork consumption increased total cholesterol in male ICR mice to 704 mg/dL. Beef-fed mice showed moderate cholesterol levels (319–321 mg/dL), while the control remained normal (114 mg/dL). Heart weight was highest in the pork group (0.25 g). Histopathology revealed fat cell accumulation only in pork-fed mice, with normal heart structures in both control and beef-fed groups.

Novelty/Originality of this study: This study uniquely investigates the direct impact of pork consumption on total cholesterol levels and cardiac histopathology in male ICR mice. It provides new evidence of fat accumulation in heart tissue due to pork intake, contributing to early detection of dietary-induced cardiac risks and expanding current understanding of food-based cardiovascular effects in animal models.

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1. INTRODUCTION

Food safety is one of the essential aspects that must be considered in daily consumption. The public is becoming increasingly aware of the importance of paying attention to the nutritional content and the long-term effects of the food they consume, particularly in relation to metabolic health and the function of vital organs [1]-[3]. One of the critical issues in food safety is the clarity of ingredient composition, including fat content and the

animal origin of food products [4]-[6]. Several cases involving the discovery of pork fat in food and beverage products highlight the urgent need for stricter supervision of the food industry.

One particular pig species that has drawn attention in the context of food consumption is *Sus barbatus*, commonly known as the bearded pig. This species is known for its high fat content and physiological characteristics that may influence the nutritional quality of its meat [7]-[9]. Although the texture and taste of *Sus barbatus* meat may be appealing to some consumers, its consumption poses potential health risks, particularly related to lipid metabolism [10], [11]. Pork generally contains high levels of saturated fat, which can raise blood cholesterol levels and promote the formation of arterial plaque [12], [13]. This condition increases the risk of cardiovascular diseases, including atherosclerosis, hypertension, and heart attacks..

Meat consumption—especially red meat from terrestrial animals—significantly affects consumers' nutritional status and metabolic health. Saturated fats, which are abundant in animal-based products, can raise total cholesterol and low-density lipoprotein (LDL) levels, both of which play key roles in the development of atherosclerotic plaque [14], [15]. Moreover, a high-fat diet without adequate fiber intake may further worsen blood lipid profiles [16], [17]. Numerous studies have linked red meat consumption with an increased risk of degenerative diseases such as coronary heart disease and dysfunction of vital organs [18], [19].

Cholesterol is one of the biomolecules that plays a crucial role in the pathophysiology of cardiovascular disease [20], [21]. A 2002 World Health Organization (WHO) report estimated that hypercholesterolemia was responsible for approximately 4.4 million deaths annually worldwide [22], [23]. Elevated cholesterol levels can lead to the narrowing and hardening of blood vessels (atherosclerosis), which can severely impact heart function [24], [25]. Previous studies have shown that the consumption of red meat, including from certain pig species, contributes to increased total cholesterol levels and organ dysfunction, either through histopathological alterations or systemic inflammation.

Research by Jahan et al. [26] showed that consumption of fat from beef significantly increased total cholesterol levels, LDL, and caused histological changes in the liver of mice. Meanwhile, another study conducted by Morrison et al. [27] confirmed that animal fat from pork and beef exacerbates atherosclerosis and increases inflammatory mediators in the blood. This fact is supported by the findings of Li et al. [28], which shows that red meat consumption not only affects lipid metabolism but also disrupts the balance of gut microbiota, which has systemic effects on health.

Studies on the health effects of *Sus barbatus* meat consumption remain very limited, particularly in the context of experimental trials using animal models. The novelty of this study lies in its specific focus on directly evaluating the impact of *Sus barbatus* meat consumption on total cholesterol levels and the histological structure of the heart in male ICR strain mice (*Mus musculus*). By utilizing both biochemical and histopathological parameters, this research aims to provide relevant and in-depth empirical data regarding the potential cardiometabolic risks associated with consuming bearded pig meat.

The urgency of this study continues to grow in line with the increasing need for scientific evidence to support decision-making in food safety, as well as strategies for preventing degenerative diseases through better regulation of animal protein consumption. This research contributes to the existing body of knowledge by addressing a specific gap in the literature and by providing a foundation for further investigations into the health implications of consuming meat from non-conventional animal sources.

2. RESEARCH METHOD

This study is a qualitative research with an experimental approach, which applies the principles of control over variables that influence the course of the experiment [29], [30]. The research was conducted at the Biopharmaceutical Laboratory, Faculty of Pharmacy. The experimental animals used were male ICR mice (*Mus musculus*), aged 2–3 months and weighing 25–30 grams. The variables in this study consisted of two types: the independent variable (types of meat: *Sus barbatus* and *Bos taurus*) and the dependent variables (lipoprotein levels and cardiac histopathology of the mice).

The data collection method used in this study was a laboratory experiment. The tools used included mouse cages, nipples, feeding containers, gloves, masks, a digital scale, surgical instruments such as dissection boards, petri dishes, tweezers, glass slides, scalpels, plastic, 1 cc syringes, venoject tubes, a centrifuge, a microtome, and a digital camera. The materials used included standard AD1 feed, husks, water, 15 adult male mice, pork (*Sus barbatus*) and beef (*Bos taurus*) meat, ether, 10% formalin, absolute alcohol I, II, III, acid alcohol, xylol I, II, canada balsam, egg albumin, paraffin I, II, Hematoxylin-Eosin (HE) staining solution, and distilled water.

The research began with a preparation phase, which involved obtaining pork and beef from a traditional market. The meat was diced and boiled for five minutes before being fed to the mice. Ten male ICR mice (*Mus musculus*), weighing 25–30 grams and aged 2–3 months, were acclimatized for one week with standard feed. The mice were then randomly divided into three treatment groups: a control group (standard feed only), a group fed with pork, and a group fed with beef. Each mouse received 5 grams of meat per day. On the 30th day, blood was

collected via the orbital sinus to obtain serum samples, which were then analyzed for total cholesterol levels using an autoanalyzer.

The next phase involved the preparation of heart histopathology slides, performed at the anatomical pathology laboratory using the Kiernan method. The process included tissue fixation, dehydration, paraffin infiltration, sectioning with a microtome, and staining using the Hematoxylin-Eosin (HE) method. The prepared slides were examined under a microscope at 100x and 400x magnification to observe histopathological changes such as fatty degeneration (vacuolization) and necrosis. The experimental data were analyzed using a Completely Randomized Design (CRD) and processed with SPSS software to determine significant differences between treatment groups.

3. RESULTS AND DISCUSSION

This study investigated the effect of *Sus barbatus* (bearded pig) meat consumption on total cholesterol levels and the histopathological profile of the heart in mice (*Mus musculus*). The experimental animals used in this study were 15 male mice (*Mus musculus*), each weighing between 24–25 grams. The animals underwent a 7-day acclimatization period before treatment began.

Following acclimatization, treatment was initiated on the 8th day using a Completely Randomized Design (CRD) consisting of three treatment groups:

- P0 (control): received standard feed only
 - P1: received *Sus barbatus* (bearded pig) meat
 - P2: received *Bos taurus* (beef) meat
- Each group consisted of five mice.

3.1. Body Weight Measurement of Male ICR Mice (*Mus musculus*)

Mice exhibit a growth curve characterized by data points representing body weight over time. The growth pattern of mice follows a sigmoid curve. There are two distinct phases in mouse growth: the first phase is the rapid growth phase, during which the rate of body weight gain increases sharply; the second phase is the slow growth phase, where the rate of weight gain decreases until it reaches zero, indicating that the animal has reached full maturity. The point between these two phases is referred to as the transition point. The average initial and final body weights of the mice are presented in Table 1.

Table 1. Average Initial and Final Body Weight of Male ICR Mice (*Mus musculus*)

Treatment	Mean \pm Std. Deviation	
	Initial Body Weight (g)	Final Body Weight (g)
P0 (Control)	28.78 \pm 0.94	32.64 \pm 0.45
P1 (Pork Meat)	26.30 \pm 0.00	28.92 \pm 2.60
P2 (Beef Meat)	25.38 \pm 1.88	33.98 \pm 0.93

The results presented in Table 1 show that the control group (P0) had a relatively low initial body weight of 28.78 grams, which increased to 32.64 grams. This result is comparable to that of the P2 treatment group (fed with beef), which had an even lower initial body weight of 25.38 grams but showed a significant increase to 33.98 grams. In contrast, the P1 group (fed with pork) exhibited unstable weight gain, with an initial weight of 26.3 grams and a final weight of only 28.92 grams. Thus, the average weight gain in the P0 and P2 groups was greater than in the P1 group.

The increase in mice body weight was influenced by the level of feed consumption. Mice in the P0 (control) and P2 (beef) groups showed higher consumption levels as they were able to consume the feed more optimally. Conversely, mice in the P1 (pork) group showed lower feed intake, resulting in less stable weight gain. This is presumed to be due to the pork hardening more quickly, making it more difficult for the mice to consume. A decrease in appetite caused by the tough texture of pork may lead to reduced weight gain and potentially affect animal health.

Previous studies support these findings, indicating that pork consumption in mice may induce liver inflammation [31], [32]. During the 21-day maintenance period, it was observed that mice in the pork-fed group consistently left more feed residues each day, whereas in the beef-fed group, there were no significant feed leftovers (as shown in Table 2).

Table 2. Residual feed from pork consumption

Treatment Day	Residual Consumption (grams)
1	-
2	12.1
3	15.12
4	10.08
5	14.0
6	15.29
7	13.29
8	13.29
9	12.32
10	15.32
11	15.02
12	15.0
13	12.29
14	12.30
15	13.26
16	11.22
17	12.0
18	12.21
19	11.42
20	-
21	13.29

Following the body weight measurements, the mice were sacrificed to collect their hearts. The heart organs were weighed to assess differences in heart weight across the treatment groups. These results are presented in Table 3.

Table 3. Heart Organ Weight of Male ICR Mice (*Mus musculus*)

Treatment	Mean \pm Std. Deviation
Heart Weight	
P0 (Control)	0.09
P1 (Pork)	0.25
P2 (Beef)	0.18

The results of the heart organ weighing presented in Table 3 show that the average heart weight varied among treatment groups. In the control group (P0), the heart weight was 0.09 grams. The P1 treatment group (fed with pork) had a heart weight of 0.25 grams, while the P2 group (fed with beef) had a heart weight of 0.18 grams.

These differences indicate that the heart weight of mice in the control group (P0) remained within the normal range. In contrast, the P1 and P2 groups showed cardiac enlargement beyond normal limits. This enlargement is presumed to result from the addition of cardiac muscle tissue, particularly due to thickening of the left ventricular wall. Ventricular wall thickening reflects a muscular response to increased cardiac workload and potential obstruction of blood flow, which may ultimately impair optimal cardiac filling [33], [34].

This condition is referred to as cardiac hypertrophy, which represents a compensatory response to pressure overload or volume overload, leading to increased wall tension in the cardiac muscle. Left ventricular hypertrophy begins with an increase in myocardial contractility, influenced by the activation of the adrenergic nervous system as part of a neurohumoral response. This is followed by an increase in venous return due to peripheral vasoconstriction and fluid retention by the kidneys.

The resulting increase in blood volume within the vascular system raises the workload of the heart. Subsequently, myocardial contraction diminishes due to reduced blood supply from the coronary arteries, which is caused by arteriosclerosis and a decline in coronary flow reserve. As peripheral resistance and systolic load on the left ventricle increase, the heart undergoes hypertrophy as a result of sympathetic nervous system activation aimed at enhancing myocardial contractility [35].

The differences among the three treatment groups are considerable. From the data, it can be concluded that the treatment with the most significant effect on increasing heart weight was P1, the group fed with *Sus barbatus* meat.

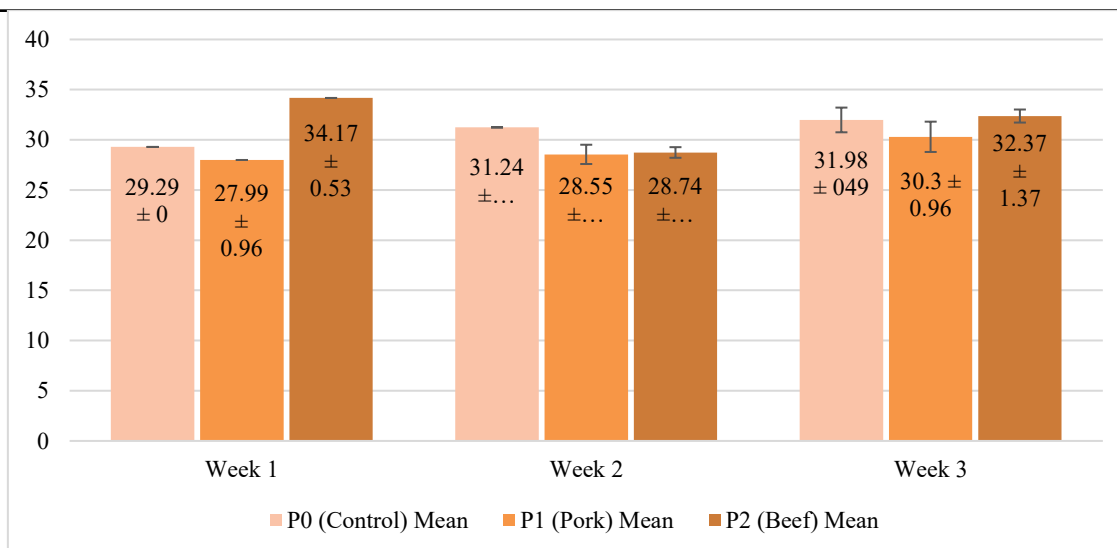


Figure 1. Body Weight Chart of Mice Subjected to Treatment Over 21 Days

Figure 1 illustrates the average body weight of male ICR mice that received different dietary treatments (P0 = control, P1 = pork, and P2 = beef) over a 21-day period. In Week 1, the P2 group (beef) showed the highest mean body weight (34.17 ± 0.53 g), followed by the P0 control group (29.29 ± 0 g), and the P1 group (pork) with the lowest weight (27.99 ± 0.96 g). By Week 2, the P0 group continued to show an increase (31.24 ± 1.23 g), whereas the P1 and P2 groups decreased slightly to 28.55 ± 1.51 g and 28.74 ± 0.56 g, respectively. However, by Week 3, all groups showed weight gain. The P2 group increased to 32.37 ± 1.37 g, the P0 group reached 31.98 ± 0.49 g, and the P1 group also improved to 30.30 ± 0.96 g. Overall, the P2 group (beef) maintained the highest average weight across the study, suggesting that beef consumption more effectively supports body weight gain in mice compared to pork. Meanwhile, although the P1 group showed an initial decrease, it eventually experienced weight gain by the end of the treatment period.

3.2. Total Cholesterol Level Measurement and Cardiac Histopathology Examination

For the measurement of total cholesterol levels, approximately 1 ml of blood serum is required to detect the presence of lipids in the blood. The lipoprotein level analysis in this study used samples from five mice: P0:M3, P1:M4, P1:M5, P2:M1, and P2:M3. As shown in Table 4.3, lipid presence was detected in one of these samples.

Table 4. Results of Total Blood Cholesterol Test in Male ICR Mice (*Mus musculus*)

Sample	Mean Lipoprotein Level Test Result
P0 (Control)	1.14
P1 (Pork Meat)	7.04
P2 (Beef)	3.19

The results of the lipoprotein level test showed an increase in cholesterol levels in the blood of mice, although not all treatments experienced this increase. Based on the average cholesterol level data in Table 4, it is known that the P0 treatment as a control, which was only fed AD1, did not increase the blood cholesterol levels of mice and was still within the normal range, which was 114 mg/dL. Meanwhile, the highest cholesterol levels were found in the P1 treatment which was fed pork, which was 704 mg/dL. The P2 treatment which was fed beef had an average cholesterol level of 319 mg/dL. The increase in blood cholesterol levels was caused by the consumption of meat that is high in fat and cholesterol. According to data from the USDA (United States Department of Agriculture) [36], The cholesterol content in 100 grams of pork tenderloin is around 79 mg, while lean ground beef contains around 78 mg of cholesterol.

The fat content of pork is also higher than beef. Consuming foods high in fat and cholesterol will increase blood cholesterol levels and LDL levels. High amounts of saturated fat cause the liver to stop taking LDL, so that cholesterol levels in the blood increase. High LDL will cause cholesterol to stick to the walls of blood vessels, forming plaque known as atherosclerosis [37], [38]. This cholesterol buildup narrows blood vessels, increases blood pressure, and increases the risk of coronary heart disease [39].

Fat intake exceeding 30% of total daily energy needs can disrupt fat metabolism in the blood. Therefore, it is recommended to consume fat no more than 30% of total daily energy needs [40]. In addition to food consumption, cholesterol levels are also affected by physical activity. Lack of physical activity or a sedentary lifestyle can increase the risk of coronary heart disease [41], [42].

Food consumed will be processed through digestion of carbohydrates, proteins, and lipids into glucose, amino acids, and fatty acids and glycerol. These products are processed into acetyl CoA and oxidized through the citric acid cycle [43], [44]. Lipids consist of triglycerides, cholesterol, and phospholipids. Triglycerides are the most abundant type of lipid. Saturated fats are found in animal products, while unsaturated fats are found in grains, nuts, and vegetable oils [45], [46]. Lipids function as a source of energy, heat insulators, energy reserves (triglycerides), precursors of adrenal hormones and gonadal steroids, and raw materials for bile acids [47], [48]. Because lipids are hydrophobic, a solvent in the form of apoproteins in the form of spherical particles called lipoproteins is needed to transport lipids in the blood [49].

Histopathological examination was conducted to assess myocardial cell damage in the heart. For this examination, three heart organ samples were taken from each treatment group: P0, P1, and P2. Observations were made after staining the heart tissue sections with Hematoxylin-Eosin (HE) and examining five different fields of view within a single tissue section at 400x magnification. The results of the histopathological examination are presented in Table 5.

Table 5. Histopathological Examination Results of the Heart in Male Mice (*Mus musculus*) ICR

No	Organ	Code	Description
1	Heart	Control (P0)	No accumulation of fat cells observed
2	Heart	Pork (P1)	Accumulation of fat cells observed
3	Heart	Beef (P3)	No accumulation of fat cells observed

Based on Table 5, the results of histopathological examination show that in the P0 (control) and P2 (beef) treatments, no changes in the structure of the heart tissue or the presence of fat cells in the adipose tissue were found. In contrast, in the P1 (pork) treatment, there was tissue damage in the form of fat cell infiltration in the adipose tissue. Figure 4 shows changes in the histopathological structure indicating cell degeneration characterized by fat accumulation. Fat accumulation in cells is one of the characteristics of the degeneration process, where cells experience enlargement of fat droplets that push the cell nucleus to the edge or appear like small droplets in the cytoplasm.

The fat content in pork was recorded at 8.25%, higher than beef which only contains 4.5% fat. Excessive intake of unsaturated fat in mice can disrupt fat metabolism. Unlike carbohydrates and proteins that are directly metabolized in the liver, fat is first transported to adipose tissue and muscle, which contain the enzyme lipoprotein lipase to hydrolyze fat into free fatty acids. These free fatty acids will then be carried to the liver for further metabolic processes [50]. Cell damage due to fat infiltration causes penetration of fat cells into the cell membrane and accumulation of intracellular fat, including between cardiac parenchymal cells.

This is thought to be a result of the transformation of interstitial tissue cells into fat cells. The free fatty acids formed can undergo esterification into triacylglycerols, both in the liver, adipose tissue, and muscles. Excessive fat intake causes accumulation of triacylglycerols in the muscles, including the heart muscle. This accumulation occurs through the transport of triacylglycerol by VLDL from the liver to the circulation, then to peripheral tissues or muscles that have the enzyme lipoprotein lipase, thereby increasing the accumulation of fat in myocardial cells—a condition known as myocardial fatty acid.

Free fatty acids (FA) can enter myocardial cells through three main pathways. First, FA are derived from the hydrolysis of triglycerides (TG) in adipose tissue by hormone-sensitive lipase and circulate bound to albumin. Second, FA are produced from the intracellular hydrolysis of TG in the lipoprotein core. Third, FA are derived from the local hydrolysis of chylomicron TG and VLDL by the enzyme lipoprotein lipase (LPL) in local capillaries. Although the concentration of FA in TG lipoproteins is higher, it is generally believed that FA bound to albumin is the main source of energy for myocardial cells. Images of the heart tissues from groups P0, P1, and P2 are shown below.

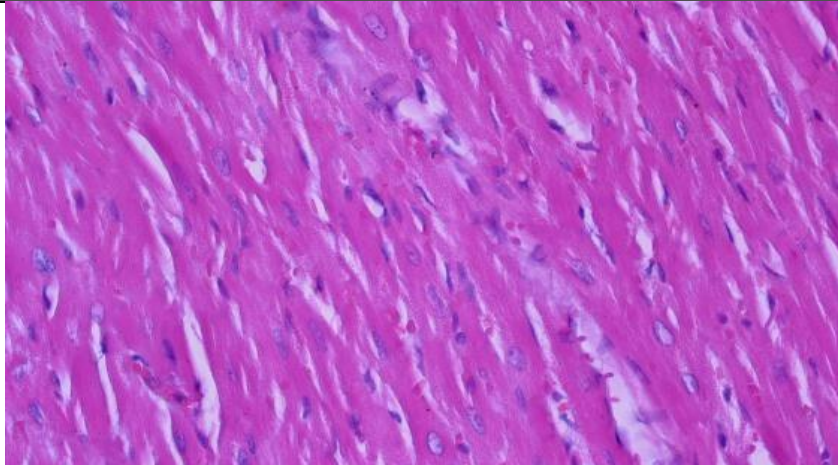


Figure 2. Histopathology of the Heart in Male ICR Mice (*Mus musculus*) Under Control Treatment (P0)

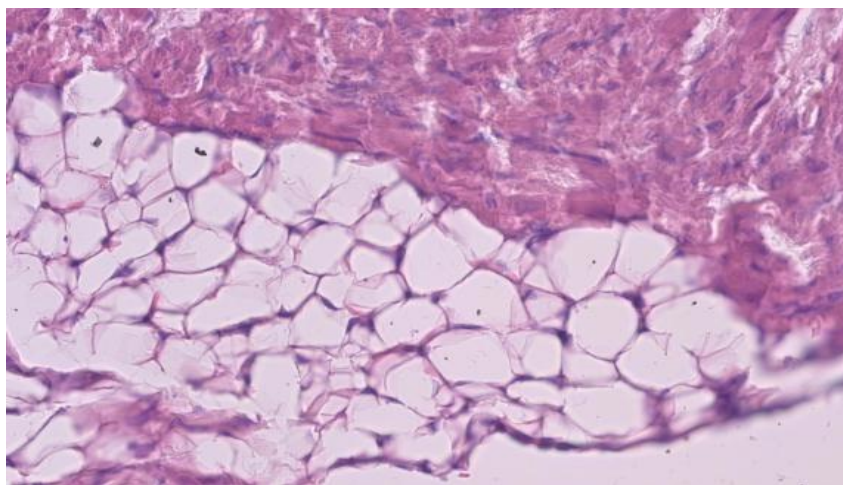


Figure 3. Histopathology of the Heart of ICR Mice (*Mus musculus*) with pork treatment (P1)

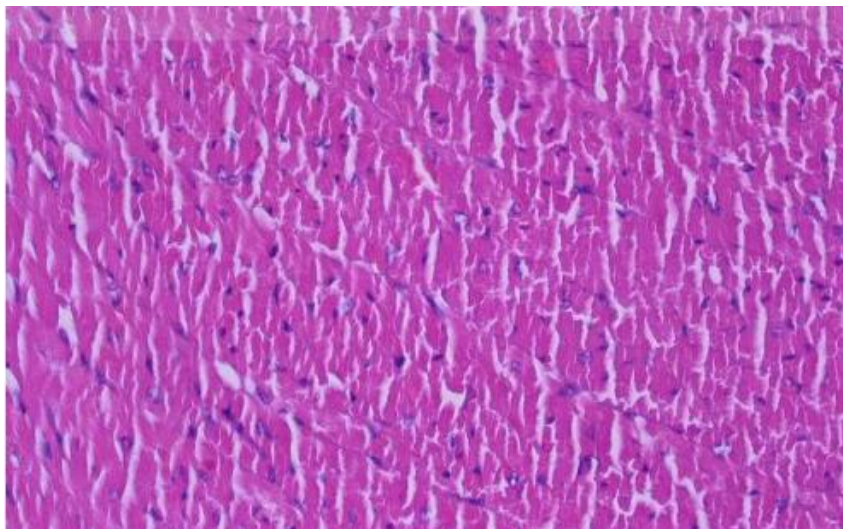


Figure 4. Histopathology of the Heart of Male ICR Mice (*Mus musculus*) with beef treatment (P2)

The image above is an image of the heart tissue resulting from histopathological examination of the mouse heart, where in Figure 2 with sample P0 it looks normal, there are no fat cell deposits in the adipose, and for Figure 3 with sample P1 with pork treatment there are visible fat cell deposits in the organ tissue, while in Figure 4 which was given beef treatment, the mouse heart organ is also normal, no fat cell deposits are visible.

This study provides novel evidence on the physiological impacts of *Sus barbatus* (bearded pig) meat consumption compared to *Bos taurus* (beef) in mice, specifically highlighting its significant effect on increasing

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blood cholesterol levels and inducing histopathological changes in cardiac tissue. The findings underscore that pork consumption, particularly from *Sus barbatus*, leads to higher residual feed, unstable body weight gain, cardiac hypertrophy, and fat infiltration in myocardial cells, which are early markers of cardiovascular risk. These results have important implications for public health and nutritional science, particularly in regions where exotic or wild meats are traditionally consumed without comprehensive understanding of their metabolic consequences. However, the study is limited by its small sample size and the short duration of intervention, which may not fully capture long-term physiological adaptations or cumulative organ damage. Future studies with larger cohorts, longer observation periods, and broader biochemical assessments are warranted to validate and expand upon these preliminary findings.

4. CONCLUSION

Based on the findings of this study, it can be concluded that the consumption of *Sus barbatus* (bearded pig) meat significantly increases total blood cholesterol levels and causes adverse histopathological changes in cardiac tissue, such as myocardial fat infiltration and cardiac hypertrophy, in male ICR mice. In contrast, mice fed with *Bos taurus* (beef) exhibited better weight gain, lower cholesterol levels, and normal heart histology, similar to the control group. These results indicate that the high fat and cholesterol content in *Sus barbatus* meat poses a greater risk to cardiovascular health. Therefore, it is recommended that future studies be conducted using a larger sample size, extended observation period, and include additional biochemical markers (e.g., LDL, HDL, triglycerides, inflammatory cytokines) to better understand the systemic impacts. The implications of this research are particularly relevant for dietary health policies and public awareness in communities where wild or exotic meats are part of traditional diets, emphasizing the need for caution and nutritional evaluation before regular consumption.

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AUTHOR CONTRIBUTIONS

Conceptualization, K.S. and J.M.; Methodology, K.S. and J.M.; Software, K.S.; Validation, K.S., J.M. and K.K.; Formal Analysis, K.S.; Investigation, K.S.; Resources, J.M. and K.K.; Data Curation, K.S.; Writing – Original Draft Preparation, K.S.; Writing – Review & Editing, K.S., J.M. and K.K.; Visualization, K.S.; Supervision, J.M. and K.K.; Project Administration, J.M.; Funding Acquisition, K.K.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

USE OF ARTIFICIAL INTELLIGENCE (AI)-ASSISTED TECHNOLOGY

Not applicable.

REFERENCES

- [1] D. Ambroselli *et al.*, “New advances in metabolic syndrome, from prevention to treatment: the role of diet and food,” *Nutrients*, vol. 15, no. 3, 2023, doi: 10.3390/nu15030640.
- [2] S. I. S. Rattan and G. Kaur, “Nutrition, food and diet in health and longevity: we eat what we are,” *Nutrients*, vol. 14, no. 24, pp. 1–7, 2022, doi: 10.3390/nu14245376.
- [3] N. E. Marshall *et al.*, “The importance of nutrition in pregnancy and lactation: lifelong consequences,” *Am. J. Obstet. Gynecol.*, vol. 226, no. 5, pp. 607–632, May 2022, doi: 10.1016/j.ajog.2021.12.035.
- [4] M. Esposito *et al.*, “Insoluble residues from soybean, rice, oat and almond -based beverage: Landscape of the product category, chemical characterisation and valorization in the food industry,” *Trends Food Sci. Technol.*, vol. 152, p. 104669, 2024, doi: 10.1016/j.tifs.2024.104669.
- [5] P. W. Harlina *et al.*, “Lipidomics: a comprehensive review in navigating the functional quality of animal and fish products,” *Int. J. Food Prop.*, vol. 26, no. 2, pp. 3115–3136, 2023, doi: 10.1080/10942912.2023.2252622.
- [6] C. O. R. Okpala and M. Korzeniowska, “Understanding the relevance of quality management in agro-food product industry: From ethical considerations to assuring food hygiene quality safety standards and its associated processes,” *Food Rev. Int.*, vol. 39, no. 4, pp. 1879–1952, 2023, doi: 10.1080/87559129.2021.1938600.
- [7] G. Siemiński *et al.*, “Meat nutritional value of puławska fattening pigs, polish large white × puławska crossbreeds and hybrids of danbred,” *Animals*, vol. 13, no. 15, pp. 1–11, 2023, doi: 10.3390/ani13152408.
- [8] I. Chernukha *et al.*, “Pork fat and meat: a balance between consumer expectations and nutrient composition of four pig

- breeds,” *Foods*, vol. 12, no. 4, 2023, doi: 10.3390/foods12040690.
- [9] A. Brumm, “Pigs as pets: early human relations with the sulawesi warty pig (*Sus celebensis*),” *Animals*, vol. 13, no. 1, 2023, doi: 10.3390/ani13010048.
- [10] S. S. Q. Rodrigues, L. Vasconcelos, A. Leite, I. Ferreira, E. Pereira, and A. Teixeira, “Novel approaches to improve meat products’ healthy characteristics: a review on lipids, salts, and nitrites,” *Foods*, vol. 12, no. 15, pp. 1–18, 2023, doi: 10.3390/foods12152962.
- [11] A. D. Carboni, T. Di Renzo, S. Nazzaro, P. Marena, M. C. Puppo, and A. Reale, “A comprehensive review of edible flowers with a focus on microbiological, nutritional, and potential health aspects,” *Foods*, vol. 14, no. 10, 2025, doi: 10.3390/foods14101719.
- [12] R. Barthwal, S. Jain, P. Maurya, and M. K. P., “Meat Products with Low Cholesterol BT - Healthier Meat Products,” A. Kumar Pathera, H. Kumar, and S. Yadav, Eds., Cham: Springer Nature Switzerland, 2025, pp. 313–329. doi: 10.1007/978-3-031-78215-2_15.
- [13] C. R. D. Joseph Mercola, “Linoleic acid : a narrative review of the effects of increased chronic disease,” *Nutrients*, vol. 15, no. 3129, 2023, doi: 10.3390/nu15143129.
- [14] S. U. Islam, M. B. Ahmed, H. Ahsan, and Y. S. Lee, “Recent molecular mechanisms and beneficial effects of phytochemicals and plant-based whole foods in reducing ldl-c and preventing cardiovascular disease,” *Antioxidants*, vol. 10, no. 5, 2021, doi: 10.3390/antiox10050784.
- [15] R. S. Najjar, “The impacts of animal-based diets in cardiovascular disease development: a cellular and physiological overview,” *J. Cardiovasc. Dev. Dis.*, vol. 10, no. 7, 2023, doi: 10.3390/jcdd10070282.
- [16] H. Liang *et al.*, “A high-fat diet and high-fat and high-cholesterol diet may affect glucose and lipid metabolism differentially through gut microbiota in mice,” *Exp. Anim.*, vol. 70, no. 1, pp. 73–83, 2021, doi: 10.1538/expanim.20-0094.
- [17] Y. Nie and F. Luo, “Dietary fiber: an opportunity for a global control of hyperlipidemia,” *Oxid. Med. Cell. Longev.*, vol. 2021, 2021, doi: 10.1155/2021/5542342.
- [18] C. Giromini and D. I. Givens, “Benefits and risks associated with meat consumption during key life processes and in relation to the risk of chronic diseases,” 2022. doi: 10.3390/foods11142063.
- [19] C. Caiati, A. Stanca, and M. E. Lepera, “Free radicals and obesity-related chronic inflammation contrasted by antioxidants: a new perspective in coronary artery disease,” *Metabolites*, vol. 13, no. 6, 2023, doi: 10.3390/metabo13060712.
- [20] S. Sood, N. Mittal, T. G. Singh, and S. Devi, “Pathogenesis of obesity-associated cardiovascular diseases: key role of biomolecules,” *Heal. Sci. Rev.*, vol. 7, p. 100098, 2023, doi: 10.1016/j.hsr.2023.100098.
- [21] R. Kumar, N. Chhillar, D. S. Gupta, G. Kaur, S. Singhal, and T. Chauhan, “Cholesterol homeostasis, mechanisms of molecular pathways, and cardiac health: a current outlook,” *Curr. Probl. Cardiol.*, vol. 49, no. 1, Part B, p. 102081, 2024, doi: 10.1016/j.cpcardiol.2023.102081.
- [22] N. W. S. Chew *et al.*, “The global burden of metabolic disease: data from 2000 to 2019,” *Cell Metab.*, vol. 35, no. 3, pp. 414–428.e3, Mar. 2023, doi: 10.1016/j.cmet.2023.02.003.
- [23] D. Mohajan and H. K. Mohajan, “Obesity and its related diseases: a new escalating alarming in global aealth,” *J. Innov. Med. Res.*, vol. 2, no. 3, pp. 12–23, 2023, doi: 10.56397/jimr/2023.03.04.
- [24] E. Gusev and A. Sarapultsev, “Atherosclerosis and inflammation: insights from the theory of general pathological processes,” *Int. J. Mol. Sci.*, vol. 24, no. 9, 2023, doi: 10.3390/ijms24097910.
- [25] R. K. Upadhyay, “High cholesterol disorders, myocardial infarction and its therapeutics,” *World J. Cardiovasc. Dis.*, vol. 13, no. 08, pp. 433–469, 2023, doi: 10.4236/wjcd.2023.138040.
- [26] M. S. Jahan, M. I. Haque, M. Gautam, and M. E. R. Bhuiyan, “Comparative analysis of high-fat diets: effects of mutton, beef, and vegetable fats on body weight, biochemical profiles, and liver histology in mice,” *Heliyon*, vol. 10, no. 20, p. e39349, 2024, doi: 10.1016/j.heliyon.2024.e39349.
- [27] M. C. Morrison *et al.*, “Differential effects of plant and animal fats on obesity-induced dyslipidemia and atherosclerosis in Ldlr-/-Leiden mice,” *FASEB J. Off. Publ. Fed. Am. Soc. Exp. Biol.*, vol. 37, no. 8, p. e23096, Aug. 2023, doi: 10.1096/fj.202300585R.
- [28] J. Li *et al.*, “Differential effects of dietary white meat and red meat on NAFLD progression by modulating gut microbiota and metabolites in rats,” *Oxid. Med. Cell. Longev.*, vol. 2022, p. 6908934, 2022, doi: 10.1155/2022/6908934.
- [29] B. C. Yang, T. Chen, Y. F. Feng, H. Shi, and H. Zhao, “Research on experimental design method of engine system based on D-optimal design,” *Powertrain Syst. net-zero Transp. - Proc. 2021 Powertrain Syst. Net-zero Transp. Conf. 2021*, pp. 303–314, 2022, doi: 10.1201/9781003219217-17.
- [30] C. G. Thomas, “Experimental Research BT - Research Methodology and Scientific Writing,” C. G. Thomas, Ed., Cham: Springer International Publishing, 2021, pp. 93–133. doi: 10.1007/978-3-030-64865-7_5.
- [31] W. Zhu *et al.*, “Effects of dietary pork fat cooked using different methods on glucose and lipid metabolism, liver inflammation and gut microbiota in rats,” *Foods*, vol. 10, no. 12, p. 3030, Dec. 2021, doi: 10.3390/foods10123030.
- [32] M. Hussain *et al.*, “Gut inflammation exacerbates hepatic injury in C57BL/6J mice via gut-vascular barrier dysfunction with high-fat-incorporated meat protein diets,” *Food Funct.*, vol. 11, no. 10, pp. 9168–9176, 2020, doi: 10.1039/D0FO02153A.
- [33] A. Caturano *et al.*, “Cardiac hypertrophy: from pathophysiological mechanisms to heart failure development,” *RCM*, vol. 23, no. 5, pp. 165–null, 2022.
- [34] M. Yildiz, A. A. Oktay, M. H. Stewart, R. V. Milani, H. O. Ventura, and C. J. Lavie, “Left ventricular hypertrophy and hypertension,” *Prog. Cardiovasc. Dis.*, vol. 63, no. 1, pp. 10–21, 2020, doi: 10.1016/j.pcad.2019.11.009.
- [35] B. H. Lorell and B. A. Carabello, “Left Ventricular Hypertrophy,” *Circulation*, vol. 102, no. 4, pp. 470–479, Jul. 2000, doi: 10.1161/01.CIR.102.4.470.

- [36] USDA (United States Department of Agriculture), "Nutrients : Cholesterol (mg)," 2018.
- [37] S. Jebari-Benslaïman *et al.*, "Pathophysiology of atherosclerosis," *Int. J. Mol. Sci.*, vol. 23, no. 6, pp. 1–38, 2022, doi: 10.3390/ijms23063346.
- [38] A. Gąsecka, S. Rogula, Ł. Szarpak, and K. J. Filipiak, "LDL-cholesterol and platelets: Insights into their interactions in atherosclerosis," *Life*, vol. 11, no. 1, pp. 1–13, 2021, doi: 10.3390/life11010039.
- [39] W. Hollander, "Role of hypertension in atherosclerosis and cardiovascular disease.," *Am. J. Cardiol.*, vol. 38, no. 6, pp. 786–800, Nov. 1976, doi: 10.1016/0002-9149(76)90357-x.
- [40] A. Nikparast *et al.*, "The association between total, animal, and plant protein intake and metabolic dysfunction-associated fatty liver disease in overweight and obese children and adolescents," *Nutr. J.*, vol. 24, no. 1, 2025, doi: 10.1186/s12937-025-01142-4.
- [41] F. Battista, F. Duregon, M. Vecchiato, A. Ermolao, and D. Neunhaeuserer, "Sedentary lifestyle and physical inactivity: A mutual interplay with early and overt frailty," *Nutr. Metab. Cardiovasc. Dis.*, vol. 35, no. 6, p. 103971, 2025, doi: 10.1016/j.numecd.2025.103971.
- [42] Y. Sun, Z. Peng, and H. Liang, "Role of physical activity in cardiovascular disease prevention: impact of epigenetic modifications," *Front. Cardiovasc. Med.*, vol. 12, no. January, 2025, doi: 10.3389/fcvm.2025.1511222.
- [43] H. Cai *et al.*, "Lactate activates trained immunity by fueling the tricarboxylic acid cycle and regulating histone lactylation," *Nat. Commun.*, vol. 16, no. 1, pp. 1–15, 2025, doi: 10.1038/s41467-025-58563-2.
- [44] E. Vásquez Castro, Ö. Ata, M. G. Steiger, T. Causon, and D. Mattanovich, "Engineering komagataella phaffii for citric acid production through carbon-conserving supply of acetyl-CoA," *Metab. Eng.*, vol. 89, pp. 47–59, 2025, doi: 10.1016/j.ymben.2025.02.005.
- [45] L. R. de Souza, D. R. da Silva Honorato, E. E. N. Carvalho, E. V. de Barros Vilas Boas, and O. C. Neto, "Additives used as fat substitutes in meat products: sensory and technological impacts, nutritional aspects and health benefits," *Eur. Food Res. Technol.*, 2025, doi: 10.1007/s00217-025-04772-4.
- [46] Z. Chen *et al.*, "Monounsaturated fatty acids from plant or animal sources and risk of type 2 diabetes in three large prospective cohorts of men and women," *Diabetologia*, vol. 68, no. 4, pp. 801–814, 2025, doi: 10.1007/s00125-024-06353-8.
- [47] B. A. A. Sai Kumar, "Hormonal regulation of metabolism, water, and minerals BT - textbook of veterinary physiology," P. K. Das, V. Sejian, J. Mukherjee, and D. Banerjee, Eds., Singapore: Springer Nature Singapore, 2023, pp. 391–415. doi: 10.1007/978-981-19-9410-4_16.
- [48] D. Zhang *et al.*, "Important hormones regulating lipid metabolism," *Molecules*, vol. 27, no. 20, pp. 1–20, 2022, doi: 10.3390/molecules27207052.
- [49] L. Di and A. Maiseyeu, "Low-density lipoprotein nanomedicines: mechanisms of targeting, biology, and theranostic potential," *Drug Deliv.*, vol. 28, no. 1, pp. 408–421, 2021, doi: 10.1080/10717544.2021.1886199.
- [50] J. Heeren and L. Scheja, "Metabolic-associated fatty liver disease and lipoprotein metabolism," *Mol. Metab.*, vol. 50, p. 101238, 2021, doi: 10.1016/j.molmet.2021.101238.