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Impact of a soybean oil-enriched diet on metabolic parameters in a chick model of childhood obesity

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Abstract

Obese and overeating children are at risk of obesity and its complications in adulthood. Research on childhood obesity encounters numerous challenges in mammals. Broiler chicks are a suitable animal model for studying childhood obesity. Genetically, broiler chicks exhibit high growth rates. They are hyperphagic, hyperglycemic, and capable of accumulating abdominal fat, and their diet can be managed from birth. Soybean oil, which is rich in omega-6 (n-6) polyunsaturated fatty acid linoleic acid and low in omega-3 (n-3) polyunsaturated fatty acid (n-6 : n-3 ratio = 7.50), is widely utilized in human nutrition. However, conflicting findings have been reported regarding the efficacy of this oil in humans and rodents. Effects of a soybean oil-enriched diet (4.00% total fat as a control *vs.* 11.00% total fat as a treatment) on metabolic disorders in broiler chicks were evaluated from hatching to 36 days of age. Results showed no changes in food intake, body weight, appetite-regulating neuropeptide mRNA levels, blood triglycerides, or hematological parameters. In contrast, the relative abdominal fat, blood cholesterol, aortic wall thickness, intima layer, and area of fat cells increased significantly in treatment group compared to control group. Signs of liver fat infiltration (steatosis) and changes in the aortic intima layer, including increased distance between elastin fibers, were observed. In conclusion, in middle-term-fed broiler chicks, a model for childhood obesity using soybean oil high in omega-6 polyunsaturated fatty acids leads to early atherosclerosis, fatty liver, adipose dysfunction, and hypercholesterolemia, without impacting body weight or food intake.

Introduction

There is strong evidence that replacing poly-unsaturated fats (PUFAs) or monounsaturated fats with saturated fats reduces the risk of coronary heart disease and cardiovascular disease due to their ability to lower blood lipid and lipoprotein levels.[1] Soybean oil is used world-wide as an oil rich in PUFA.[2] This oil contains 51.00% linoleic acid, an omega-6 (n-6) PUFA, 22.60% oleic acid, an omega-9 monounsaturated fat, and 6.80% alpha-linolenic acid, an omega-3 (n-3) PUFA.[3] However, there is a controversy regarding the adverse effects of n-6 PUFA consumption, especially for oils containing this fatty acid.[1,4-6]

Childhood obesity is a widespread public problem, and an estimated 38.20 million children under 5 years of age are overweight or obese.[7] These children can be affected by obesity and its complications, such as cardiovascular disease, coronary heart disease, and type 2 diabetes, in adulthood.[8] There have been many discussions regarding the role of breastfeeding in reducing the risk of obesity. The n-6 PUFAs promote lipogenesis during the gestation/ suckling period. However, according to the recently published review article, the evidence of the relationship between breast milk composition and subsequent obesity is inconsistent, and further investigations are needed.[9]

Animal models are essential tools for addressing researchers' questions by exhibiting specific defects or characteristics relevant to their goals. Much of the research on metabolic disorders has been conducted in mammalian species, such as rats and mice. However, due to biological differences between rodents and humans, alternative animal models can enhance our understanding

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of these disorders. Birds, as the closest taxonomic outgroup to mammals, offer a unique perspective on evolutionary processes related to feeding. Unlike mammalian neonates, bird hatchlings can independently select and consume food shortly after hatching,[10] allowing researchers to study metabolic disorders through dietary manipulation. Additionally, chickens share key metabolic similarities with humans, such as predominant hepatic lipogenesis (rather than adipose tissue lipogenesis).[11] Genomic studies also indicate that birds, particularly chickens, have a genome structure more similar to humans than rodents.[12] Broiler chickens, in particular, serve as a compelling model for metabolic research due to their hyperphagia, rapid anabolic growth, and predisposition to abdominal fat accumulation from birth.[11] They also exhibit hyperglycemia and insulin resistance,[13] making them a valuable model for studying childhood obesity, especially given the ethical and practical challenges of conducting dietary interventions in human children.

This study aimed to investigate the effect of mid-term feeding of broilers with a diet rich in soybean oil as an animal model of childhood obesity on several factors of metabolic disorders. For this purpose, we analyzed the histopathological status of the thoracic aorta, liver, and abdominal fat tissues, expression levels of several genes involved in the regulation of food intake in the diencephalon, plasma levels of cholesterol and triglycerides, hematological parameters, daily food intake, and body weight from birth to the age of 36 days.

Materials and Methods

Animal procedures were performed in Physiology Laboratory at Shahid Bahonar University of Kerman, Kerman, Iran. Animal husbandry and care were provided by the Poultry Diseases Division in Faculty of Veterinary Medicine, Shahid Bahonar University of Kerman. Animals were treated in compliance with the Guide for Care and Use of Laboratory Animals, National Research Council of the National Academies, 8th edition (revised 2011). The experimental protocol was approved by Kerman University of Medical Sciences Ethics Committee, Kerman, Iran (Ethical Code: IR.KMU.AEC.1403.022).

Animals. One-day-old broiler chicks (Ross 308) were purchased from a local hatchery (Mahan Chicken Meat Production Complex Public Joint Stock Co., Kerman, Iran). The animals were kept under standard breeding conditions in individual cages. Chicks had access to light, food, and water 24 hr a day. A starter ration was purchased (Dan va Oloofeh Shargh Co., Birjand, Iran). Then, soybean oil (Golnaz Vegetable Oil Co., Kerman, Iran) was added to this ration to obtain two diets containing 4.00 and 11.00% fat. (Tables 1 and 2). In poultry research studies, the fat content of a high-fat diet is usually adjusted to 10.00 - 20.00%.[14-16]

Table 1. Composition of diets used by broilers during the study.

Ingredients	Amount (g kg^{-1})
Corn	562
Wheat soft	107
Fish meal 60.00%	40.00
Soya meal 44.00%	255.40
Oyster shells	10.80
Dicalcium phosphate	12.80
Methionine	1.50
Lysine	1.00
Salt	2.50
Complements	7.00

Basal diet components purchased from a local factory. The base ingredients were identical for all diets, and only soybean oil was added to this base diet to produce two diets containing 4.00% and 11.00% fat.

Experiments. This study was conducted in two groups. In the control group (n = 10), the birds received a 4.00% fat diet. In the treatment group (n = 10), chicks were fed a 4.00% fat diet for the first 3 days, a 7.00% fat diet from days three to six, or an 11.00% fat diet from days six to 36 to adapt their digestive system to the high-fat diet.[17] Daily food intake and body weight were measured using a digital scale (0.10 g). On the 36th day, the birds were anesthetized using the isoflurane (Baxter, Deerfield, USA) inhalation method, then slaughtered, and blood samples were collected to measure biochemical and hematological parameters using ethylenediaminetetraacetic acid as an anti-coagulant. The serum samples used for biochemical tests were separated and stored at – 80.00 ˚C, and the hematological parameters were measured on the day of slaughter. Abdominal fat, liver, and thoracic aorta (before bifurcation point) samples were separated, weighed, fixed in 10.00% formalin, and kept until histological slides were prepared. The brains of the birds were removed, and the diencephalon containing the hypothalamus was separated according to the method of Chowdhury *et al.*[18] and kept at – 80.00˚C until gene expression was evaluated. The fixed tissue samples were dehydrated, clarified, and embedded in paraffin. Then, they were molded in paraffin blocks, and 5.00 µm sections were prepared using MR2258 microtome (Histoline, Pantigliate, Italy). The sections were mounted on slides and stained with Hematoxylin and Eosin For aortic tissue data, 10 cross-sections and five regions for each section were studied for each bird. The thickness of the tunica intima and tunica media, and the diameter of the lumen were measured using an optical microscope and T-capture Software (version 3.9; Tuscen Photonics Co., Fuzhou, China). For abdominal fat and liver tissue data, 10 sections were prepared for each chicken. Fat cells were counted, and their area was measured using T-capture and Anix Software (version 1.5; Anix Co., Tehran, Iran). All slides were examined for the presence of any of any pathological lesions. According to the standard protocols, white and red blood cells and blood platelets were counted using a Neubauer slide, a Melangor pipette, and a light microscope.

Table 2. Analysis of three diets used in the experiments.

Analysis	Basic diet	4.00% fat diet	11.00% fat diet
Metabolizable energy (Kcal kg^{-1})	2,900	3,025	3,460
Crude protein (%)	20.00	19.58	18.12
Fat (%)	2.70	4.00	11.00
Calcium (%)	0.94	0.92	0.85
Available phosphorus (%)	0.47	0.46	0.43
Sodium (%)	0.16	0.16	0.15
Methionine (%)	0.50	0.49	0.45
Methionine + cysteine (%)	0.80	0.78	0.73
Lysine (%)	1.20	1.18	1.10

According to the relevant protocols, the hematocrit was measured using micro-hematocrit tubes and a micro-centrifuge. For the differential count of white blood cells (WBCs), Giemsa-stained blood slides were prepared, and the percentages of heterophils (equivalent to neutrophils in mammals), lymphocytes, eosinophils, and monocytes were determined using an optical micro-scope.[19] Based on the manufacturer's protocols, cholesterol and triglyceride plasma levels were measured using relevant kits (Pars Azmoon Co., Tehran, Iran) and a clinical biochemistry analyzer (Liasys; Ames, Rome, Italy). In this study, the mRNA expression levels of Agoti-related peptide (AgRP), neuropeptide Y (NPY), cocaine- and amphetamine-regulated transcript (CART), and pro-opiomelanocortin (POMC) were determined by real-time polymerase chain reaction. The total RNA extraction, complementary DNA synthesis, gene expression, and the primers used were based on a previous study.[20]

Statistical analysis. All the data are presented as the mean ± standard error of the mean. Relative (percent body weight) abdominal fat was obtained by dividing the fat weight by the body weight and multiplying by 100. The number of each type of WBC was obtained by multiplying the percentage of each by the total WBC. The data from two experimental groups were analyzed using an independent Student's *t*-test with SPSS software (version 23.0; IBM Corp., Armonk, USA). A Pearson correlation coefficient was used to determine whether the relative abdominal fat content was significantly related to the number and area of fat cells and the intima layer thickness. Repeated measures ANOVA was used for daily food intake and body weight data. The $p < 0.05$ was considered to indicate statistical significance.

Results

Effect of feeding with a soybean oil-enriched diet on the aorta, liver, and abdominal fat tissues. Significant increases in wall thickness and the thickness of the tunica intima were observed in the treatment group. The lumen diameter did not change (Fig. 1). No pathological lesions were observed in the aorta tissue in the control group (Fig. 2A). Fat infiltration and increased distance between elastic fibers were observed in the tunica intima of the treatment group (Fig. 2B). No abnormalities were observed in the hepatocytes of the liver tissue in the control group (Fig. 2C). Fatty changes (steatosis) were evident in the treatment group, and the hepatocytes were mostly swollen (Fig. 2D). The relative abdominal fat mass (Fig. 3A) and fat cell area (Fig. 3B) were increased, and the fat cell numbers decreased significantly in the treatment group (Fig. 3C). There was also a significant correlation between fat cell area and both aortic thickness and relative fat mass (Table 3).

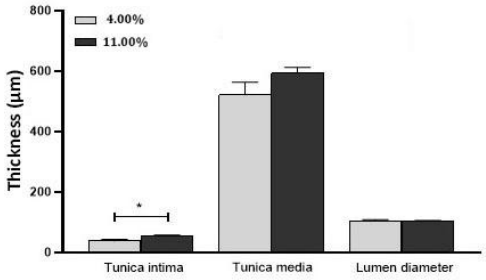

Fig. 1. Effect of feeding soybean oil-enriched diets on broiler chick thoracic aorta thickness and lumen diameter. *: $p < 0.05$.

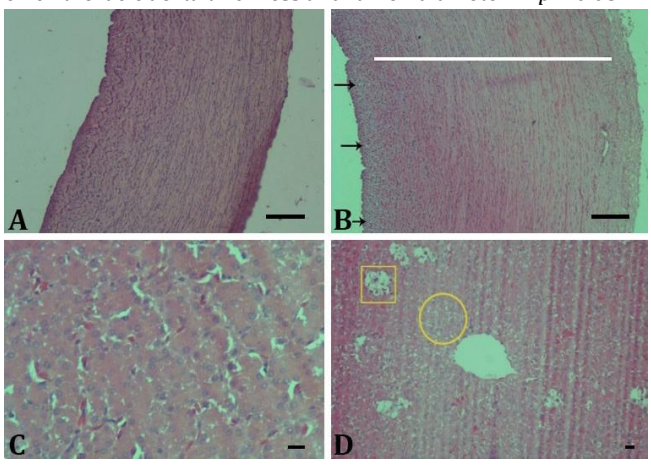

Fig. 2. A) Histological structure of the aortic wall in the 4.00% fat diet group, **B)** Histological structure of the aortic wall in the group receiving 11.00% fat diet, **C)** Liver histological structure in the 4.00% fat diet group showing normal hepatocytes, and **D)** In the group receiving 11.00% fat diet, fatty changes are seen as micro-vesicular (inside the circle) and macro-vesicular (inside the square). Arrows: Hollow vacuoles; white line: Increased thickness of the tunica media. (Hematoxylin and Eosin staining, bars in A and B = 100 µm, in C and D = 10.00 µm).

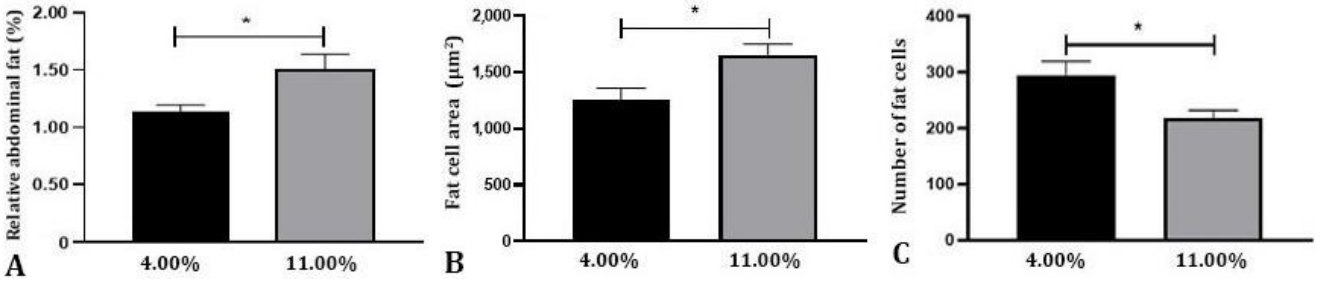

Fig. 3. Effect of feeding soybean oil-enriched diets on broiler chicks' **A)** Relative abdominal fat, **B)** Fat cell area, and **C)** Number of fat cells. * $p < 0.05$.

Table 3. Relationships between the relative amount of abdominal fat and the number and area of fat cells and intima layer thickness.

Parameters	Abdominal fat	Intima thickness	Aorta thickness	Aorta diameter	Adipocyte area	Adipocyte number	Fat mass	Mass/ number	Mass/ area
Abdominal fat (%)	1.000	**0.841***	0.672	– 0.208	0.807	– 0.738	0.919	0.933	0.379
Intima thickness (µm)		1.000	0.745	0.350	0.950	-0.957	0.696	0.810	0.052
Aorta thickness (µm)			1.000	0.118	**– 0.901***	**– 0.898***	0.624	0.719*	– 0.055
Aorta diameter (µm)				1.000	0.315	– 0.279	– 0.230	-0.074	– 0.036
Adipocyte area (µm²)					1.000	– 0.967	**0.706****	0.846	– 0.125
Adipocyte number						1.000	**– 0.613****	-0.756	0.243
Fat mass							1.000	0.962	**0.589***
Mass/number								1.000	0.367
Mass/area									1.000

* $p < 0.05$; ** $p < 0.01$.

Effect of feeding with a soybean oil-enriched diet on diencephalon NPY, AgRP, POMC, and CART mRNA levels. The results of this study showed that the mRNA levels of NPY, AgRP, POMC, and CART in the diencephalon of chicks fed a soybean oil-enriched diet did not change significantly compared to the control group. However, the AgRP level tended to decrease in the treatment group (p = 0.08; Fig. 4).

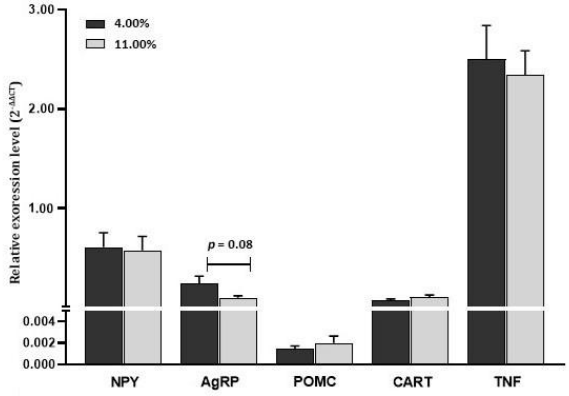

Fig. 4. Effect of feeding soybean oil-enriched diets on mRNA levels of neuropeptide Y (NPY), agoti-related peptide (AgRP), pro-opiomelanocortin (POMC), cocaine- and amphetamine-regulated transcript (CART), and tumor necrosis factor (TNF) in the diencephalon of broiler chicks

Effect of feeding with a soybean oil-enriched diet on cholesterol, triglyceride, and hematological parameters. Cholesterol (control: 121.90 ± 16.52 *versus* treatment: 137.80 ± 15.61; p = 0.46) but not triglycerides (control: 82.22 ± 25.39 *versus* treatment: 92.30 ± 25.00; p = 0.40) increased significantly in the treatment group compared to the control group. The two groups had similar (non-significant) hematological parameters, including WBC, RBC, platelet, heterophil, lymphocyte, basophil, eosinophil, and monocyte count, and hematocrit (data not shown).

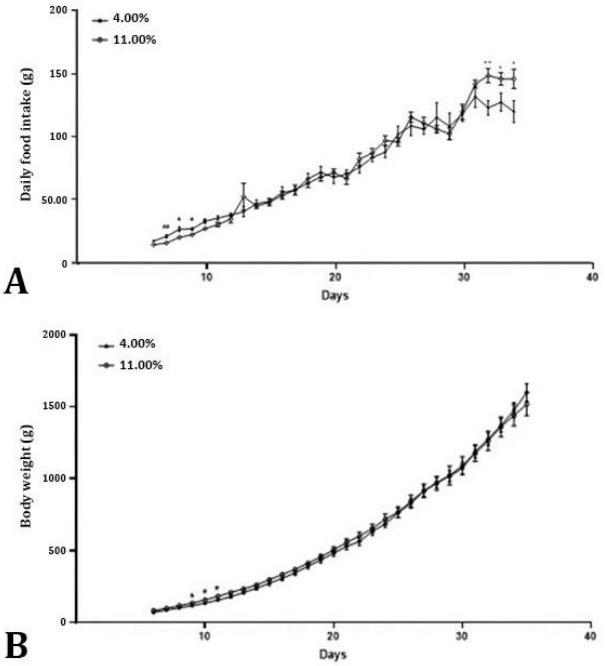

Fig. 5. Effect of feeding soybean oil-enriched diets on **A)** Daily food intake, and **B)** Body weight. * $p < 0.05$.

Effect of feeding with a soybean oil-enriched diet on daily food intake and body weight. The food intake of chicks fed a soybean oil-enriched diet increased significantly on days two to four. In contrast, it decreased in the last 3 days. However, ANOVA with repeated measures showed no significant difference in food consumption during the 36 days of keeping of these animals (Fig. 5A). Additionally, the body weight increased significantly in the treatment group on days nine - 11, although there was no significant change in the entire period of chick maintenance (Fig. 5B).

Discussion

There is a debate about whether replacing saturated fats with PUFAs leads to a reduction in coronary heart disease. Several lines of evidence favor this theory, and many findings have been reported against it. However, the results of this study contradict this theory.

According to a recently published narrative review article, based on clinical and epidemiological findings in humans, n-6-rich soybean oil reduces cholesterol levels and does not affect inflammation or oxidation markers.[21] However, in contrast to these findings, in this study, neonatal broilers fed a diet enriched with soybean oil for 36 days exhibited: 1st, a significant increase in the percentage of abdominal fat mass and area of the cells, indicating adipose cell hypertrophy, 2nd, a significant increase in the thickness of the intima layer, penetration of fat cells into it, and movement of the elastin strands away from each other, being evidence of the early stages of atherosclerosis, 3rd, penetration and fat accumulation in liver tissue, indicating steatosis, and 4th, increased plasma cholesterol levels.

In agreement with our results, there is evidence regarding the role of the n-6 PUFA arachidonic acid and its precursor linoleic acid in inflammation.[4,6,22,23] It has also been found that feeding pregnant and lactating mice a diet rich in linoleic acid causes increases in body weight, fat mass, and adipocyte size in their offspring at the age of 8 weeks. The mechanisms involved in this effect include an increase in the expression of peroxisome proliferator-activated receptor, a known agent of lipogenesis, through prostacyclin signaling.[24] Several lines of evidence on the role of a high-fat diet in the pathogenesis of non-alcoholic fatty liver disease (NAFLD), adipose tissue dysfunction, and atherosclerosis have been reported, and an increase in inflammatory processes is considered the primary and common cause of these disorders.[25] However, briefly, the pathological mechanisms are as follows: Consuming a diet rich in saturated fats, n-6, and carbohydrates but poor in n-3 and fiber is associated with NAFLD and insulin resistance.[26] Insulin causes an increase in lipogenesis in both hepatocytes and adipocytes.[27,28] Subsequently, pro-inflammatory cytokines, such as tumor necrosis factor α and interleukin 1 and 6, are released in this tissue, indicating the stimulation of immune responses.[29] Adipokines and cytokines secreted from adipose tissue play essential roles in the development of NAFLD. Increased abdominal fat is closely related to insulin resistance caused by an imbalance between the release of leptin and adiponectin from adipose tissue. Increased leptin levels can lead to liver inflammation and fibrosis, while adiponectin reduces body fat by weakening gluconeogenesis and lipogenesis. Increased and decreased leptin and adiponectin levels were observed in NAFLD patients.[30] The NAFLD can play a role in the development and exacerbation of atherosclerosis. Although the exact underlying mechanisms are unknown, the assumed mechanisms include chronic inflammation, fat accumulation, and oxidative stress. In all of these cases, dyslipidemia, lipotoxicity, and dysfunction of fat, endothelial, and liver tissues occur, leading to the release of a wide range of pro-inflammatory cytokines from the above-mentioned tissues.[25,31]

Regarding the pathology of atherosclerosis, infiltration of monocytes from pro-inflammatory activated endothelial cells into the intima layer occurs, where they become inflammation-promoting macrophages. These macrophages actively absorb modified low-density lipoproteins (oxidized LDLs) and release many inflammatory cytokines and chemokines necessary to promote inflammation. The elevated entry of LDL and accumulation of cholesterol esters in the macrophages of intima layer lead to production of floating cells, which play a crucial role in all stages of atherosclerosis, from initial vascular injuries to formation of atherosclerotic plaques.[32] Similarly, it has been reported that fixed hepatic macrophages (Kupffer cells) in NAFLD absorb oxidized LDL and participate more in forming atherosclerotic lesions.[25] In this regard, evidence has shown that consumption of n-6-enriched vegetable oils and the subsequent enrichment of LDL with n-6 elevate the sensitivity of LDL particles to oxidation due to the multiple double bounds,[33] and oxidized LDL also enhance the production of floating cells in the vascular endothelium.

As mentioned in this research, hypertrophy of abdominal fat cells was observed after the animals were fed a high-fat diet. The same results have been reported in four-day-old chickens fed a high-fat diet.[34] It has been reported that a combination of long-chain fatty acids induces the differentiation of chicken adipocytes by increasing peroxisome proliferator-activated receptor gamma.[35] Studies have shown that fat cell hypertrophy occurs mainly in abdominal adipose tissue due to a relative lack of progenitor cells, while subcutaneous fat increases mainly due to cell hyperplasia. It has been shown that accumulation of triglycerides in hypertrophied fat cells leads to dysfunction of these cells and increases the risk of metabolic disorders mentioned earlier.[36]

In birds, unlike mammals, fats are absorbed into the portal vein and directly transferred to the liver. On the other hand, in birds, similar to humans, lipogenesis occurs mainly in the liver, while in many mammals, it occurs in fat cells.[11] Therefore, it can be concluded that in the treatment group, more fat digestion products, such as fatty acids, were transferred to the liver and then converted into triglycerides attached to very LDLs , which were subsequently transferred to the fat tissue. Notably, very LDL is more prone to triglycerides than cholesterol; more interestingly, high amounts of very LDL are present in the plasma of broiler chickens.[11]

As shown in the results, in the initial and final days, food intake increased and decreased, respectively, in chickens fed a high-fat diet. Additionally, in the first few days, body weight increased in the treatment group. The deliciousness of the high-fat diet might cause an increase in food intake at initial periods. In this regard, taste, smell, and even the appearance of food can increase appetite by sending signals from gustatory, olfactory, auditory, and optic pathways to decision-making centers based on reward, such as the nucleus accumbens and ventral tegmental area (mesolimbic system).[37] The mesolimbic system has a bilateral relationship with the hypothalamus, the center of receiving, integrating energy signals, and ultimately regulating food consumption.[38] Increased food consumption during this period can lead to a positive energy balance, and increased body weight. However, decreased food consumption in the last period could be due to the delivery of satiety signals from fat tissue to the hypothalamus. Among the most important signals is the hormone leptin, which directly affects the hypothalamic arcuate nucleus to reduce food intake. In this regard, our results showed that AgRP mRNA expression tended to decrease. Several lines of evidence indicate that this hormone inhibits AgRP/NPY neurons (food intake stimulants) and stimulates POMC/CART (food intake inhibitor neurons) in the arcuate nucleus of the hypothalamus.[39] In general, ANOVA with repeated measurements showed that feeding broilers high-fat food for 36 days did not change their feed consumption or body weight gain, which can be attributed to the homeostasis of energy generated by the hypothalamus.

In the present study, plasma cholesterol increased after consumption of food rich in soybean oil. This finding contradicts the evidence that this oil lowers human cholesterol.[21] However, the mechanism of this effect has yet to be discovered and needs more research. In this study, hematological parameters were similar between the two treatment groups. In agreement with this finding, there was no change in blood RBC or WBC count in rats with obesity induced by a short-term high-fat diet (6 weeks).[40] However, hematological parameters are increased in individuals with prolonged diet-induced obesity (6 months).[41] There is an observed relative and sometimes absolute leukocytosis driven by obesity. Obesity is associated with leukocytosis and increased platelet count. However, the association between obesity and RBCred blood cell count may be present but remains uncertain.[42]

In this study, neonatal broiler chicks exhibiting an obesity phenotype and genotype, considered a suitable model for investigating childhood obesity, were fed a medium-term diet rich in PUFAs derived from soybean oil. This dietary intervention did not lead to significant changes in food intake, body weight, plasma triglyceride levels, blood cell counts, or the expression of key hypothalamic neuropeptides involved in appetite regulation. However, it induced hypercholesterolemia, early-stage atherosclerosis, an increased percentage of abdominal fat through adipocyte hypertrophy, and liver steatosis. Based on these findings, it can be concluded that consumption of n-6 PUFA-rich foods in children may elevate the risk of obesity-related metabolic diseases. Nevertheless, for a more comprehensive comparison, it is recommended to include other fat or oil sources, particularly those rich in saturated fats, alongside soybean oil.

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Conflict of interest

The authors have no conflict of interest to declare being relevant to the content of this article.

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