High Sodium Containing Diet Attenuates Body Weight and Improves Hyperlipidemia in High-Fat Diet-Induced Obese Mice

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(Received: February 18, 2025; Accepted: December 10, 2025; Published (web): June 25, 2026)

ABSTRACT: In addition to the high sugar and high fat, processed food containing high salt is considered detrimental to the worldwide obesity epidemic. The present study aimed to investigate the effect of a modified diet with high-salt (4% NaCl) on fat deposition in high-fat diet-induced obese mice. Forty healthy Swiss Albino mice (Sex: Female) were taken and divided equally into two groups named the non-obese group and the obese group. To induce obesity, mice were fed a high-fat diet. The non-obese and obese mice were further subdivided into four groups depending on their feed, i.e., normal diet, a normal diet with high salts, a high-fat diet and a high-fat diet with high salts. Central obesity was determined by monitoring body weight and Lee index. Furthermore, different organ weights, serum triglycerides, total cholesterol, high-density lipoprotein and low-density lipoprotein levels were assessed. Liver function was monitored by analyzing SGOT and SGPT. The high-fat diet was able to induce adiposity and increase the obesity-related parameters significantly. Treatment with high salts along with high fat showed a significant ($p < 0.05$) reduction in obesity and in the parameters tested, except for the significant ($p < 0.05$) elevation of high-density lipoprotein compared with their controls. The results of this study predicted that a high salt (4% NaCl) diet has an inhibitory action on fat absorption, which might be effective to attenuate the central obesity and obesity-related parameters.

Key words: Obesity; high-salt; triglycerides; abdominal fat; cholesterol.

INTRODUCTION

Dietary sodium is an essential nutrient required for the maintenance of various physiological events including nutrient absorption, acid-base balance, membrane potential, cell plasma volume and nerve impulse transmission.[1] In contrast, restricted sodium intake has been recommended for more than a century to prevent hypertension and cardiovascular and renal disorders.[2] Studies found that lowering the salt intake reduces blood pressure and eventually decreases the risk of cardiovascular disease.[3,4]

Among other risk factors, obesity is one of the most prevalent risk factors for cardiovascular complications.[5] Obesity has been characterized by the excess accumulation of fat. Multiple factors, including excess calorie intake, sedentary lifestyle and genetics are responsible for the development of obesity.[6-8] Adipose tissue has a systemic effect on the regulation of blood pressure and lipids.[9] During obesity, sodium retention is associated with an increase in renal tubular re-absorption and eventually causes hypertension.[10] The high fat and sugar in processed foods are implicated in adiposity and it has been postulated that high salt intake has a critical role in the adipogenic process.[11,12] Based on the previous studies, we also hypothesized that high salt, along with high fat, will increase obesity.

In recent years, Graudal *et al*,[13] reviewed that low sodium intake reduces blood pressure but increases renin, aldosterone, nor-adrenaline, blood cholesterol and triglycerides. Moreover, an inverse association of blood cholesterol and salt intake in hypertensive women with excess weight has been

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Dhaka Univ. J. Pharm. Sci. **25**(1): 1-10, 2026 (June)
DOI: https://doi.org/10.3329/dujps.v25i1.91118

reported.[14]. A preventive effect of high salt against the obesogenic action of high fat has been documented in high-fat diet-induced obese rats.[15]. Furthermore, the role of salt on lipid metabolism and fat deposition is equivocal. A low salt diet causes atherosclerosis by inducing fat deposition in arterial walls, whereas a high salt diet inhibits fat deposition and reduces the efficiency of fat synthesis.[16,17]

Because of ambiguity, the role of high salt in public health is still an area of research interest. Despite the detrimental effect of high salt in hypertension, it is not clear yet how high salt plays its role in different physiological events at molecular and cellular levels, especially in the regulation of body weight gain, lipid profile and associated liver function in an obese individual. Taking these into mind, we aimed to explore the effect of high salt on body weight gain, serum lipid and liver function in high-fat diet-induced obese mice.

MATERIALS AND METHODS

Experimental animals. Forty healthy female Swiss Albino mice were brought from Jahangir Nagar University, Savar and Dhaka, Bangladesh and were placed under controlled conditions at 25±2°C temperature and 55±10% humidity in polypropylene cages with 12 hours light-dark cycle and allowed to acclimatize for 7 days to the laboratory conditions before the conduct of the experiment. All the experiments were performed in the animal research laboratory at the Department of Pharmacy, Noakhali Science and Technology University.

xperimental design. The Swiss Albino mice (Sex: Female) were separated equally into two groups, considering as non-obese group and the obese group. Obesity was induced by feeding the mice a high-fat diet for two weeks according to our previous study protocol.[18] Both the obese and non-obese mice were further subdivided into four groups depending on their diet. Group-1: Normal diet (ND), Group-2: Normal diet with 4% NaCl (ND+HS), Group-3: High-fat diet (Normal diet with high fat) (HFD), Group-4: High-fat diet with 4% NaCl (HFD+HS)

Water was freely accessible to all of the mice in each group. Body weight and food intake behavior were monitored throughout the study period of eight weeks. At the end of the experimental period, mice were fasted for 18 hours and anesthetized by inhaling chloroform (Ref: K36582331, Merck KGaA, Germany), killed by decapitation and internal organs were obtained by surgical dissection, before collecting blood samples from the mice's heart and various body organs such as liver, heart, spleen and abdominal fat.

Ethical clearance. The study protocol was approved by the institutional ethical committee of Noakhali Science and Technology University (approval number 10/2019) and all the experiments were carried out in accordance with the relevant guidelines.

Body weight monitoring. Bodyweight gain was monitored by weighing the mice on a weight balance (Adventure™, AR2140) daily.

Biochemical assays. Blood was allowed to clot at room temperature (25^0C) for 30 minutes and serum was separated by centrifugation at 3500 rpm for 10 minutes and stored in -80ºC freezer before analysis. The serum triglyceride (TG), total cholesterol (TC), HDL-cholesterol, SGOT, and SGPT were analyzed by respective analytical kits as per the manufacturer's instructions available commercially. Triglycerides (Ref-1155017, Linear Chemicals SL, Barcelona, Spain), Cholesterol (Ref-1118017, Linear Chemicals SL, Barcelona, Spain), HDL-Cholesterol (Ref-1133010, Linear Chemicals SL, Barcelona, Spain), SGOT (Ref-1130009, Linear Chemicals SL, Barcelona, Spain) and SGPT (Ref-1132005, Linear Chemicals SL, Barcelona, Spain). Procedures were validated in our previous study.[19]

Statistical analysis. All data are expressed as Mean ± SEM. The statistical significance of the findings was examined using the Student's t-test and GraphPad Prism software, version 8.00 for Windows (GraphPad Software, La Jolla, CA, USA). Statistical differences were considered to be significant at $p \leq 0.05$.

RESULTS AND DISCUSSION

Effect of a high-salt diet on body weight. The effect of a high salt diet on body weight and food intake behavior was monitored during the experimental period. No significant changes in body weight were noticed during the early phase, but after two weeks, mice fed on a high-fat diet showed a significant ($p < 0.05$) elevation in body weight, and that was continued to the end of the experiment. Mice fed with a high-fat diet showed a 38.89% increase in body weight, but when mice were treated with high salts along with high fat, significant ($p < 0.05$) suppression in body weight gain (21.81%) was recorded. In contrast, high salt failed to reduce the body weight of mice fed on normal chow significantly (Figure 1A) When the obese mice were fed with high salt and normal diet, a significant ($p < 0.05$) reduction in body weight was observed (Figure 1B).

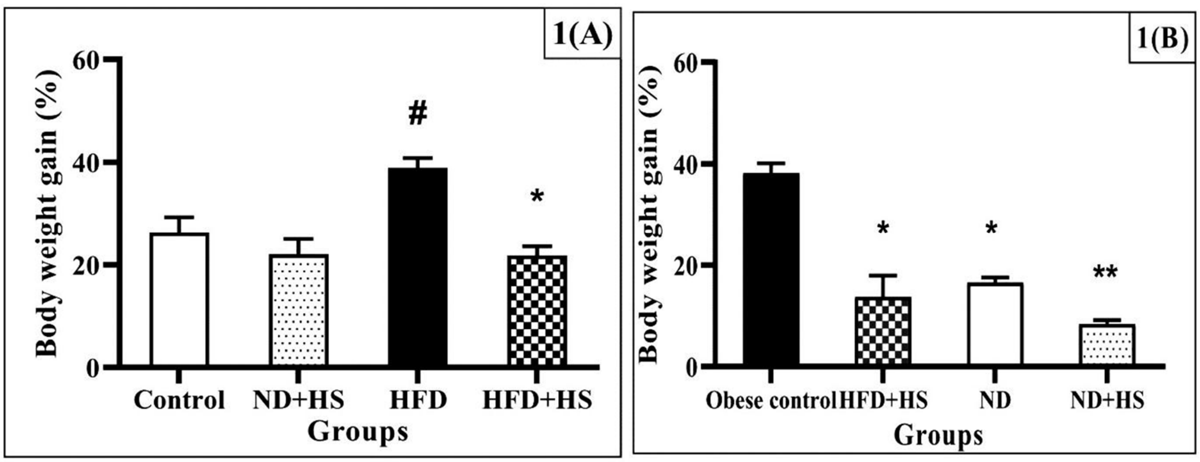

Figure 1. Changes in body weight among different groups. 1(A) non-obese group (#$p < 0.05$ vs Control and *$p < 0.05$ vs HFD) and 1(B) obese group (*$p \leq 0.05$ and **$p < 0.01$ vs Obese control).

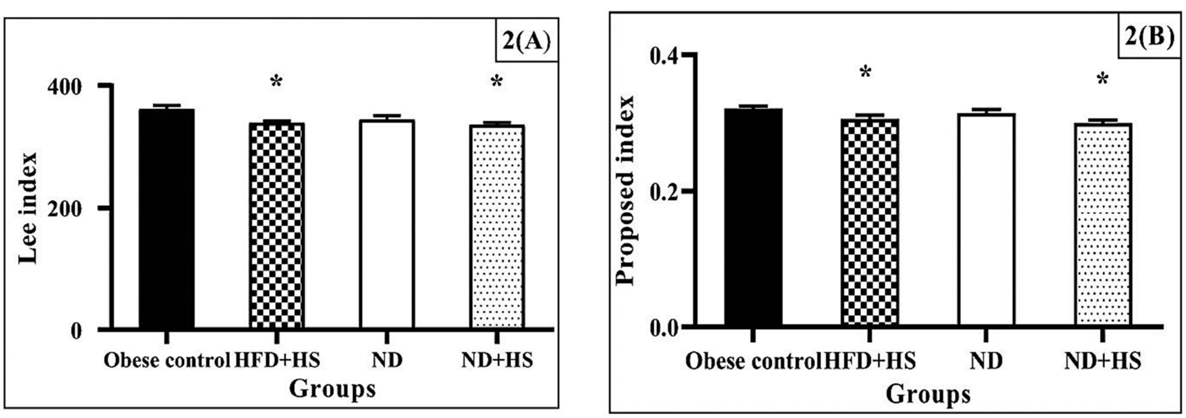

Figure 2. Effect of high-salt diet on anthropometrical parameters of obese group. 2(A) Lee index and 2(B) Proposed index (*$p < 0.05$ vs Obese control).

Lee index was measured to determine the adiposity of the obese mice. Mice fed with a high-fat diet showed a notable increase in Lee index in the obese control group compared with the non-obese group (data not shown), but a significant ($p < 0.05$) reduction was observed in the mice grouped for a high-salt diet compared with the obese control (Figure 2A). In addition to this, when we calculated central obesity considering waist circumference in lieu of weight in the Lee Index, similar results were observed (Figure 2B). But interestingly, in both the indexes, obese mice fed on normal diet did not show any significant differences compared with obese control.

Effect of a high-salt diet on organ weight. The weight of different organs was calculated and the results are presented in figure. 3. When mice were treated with a high-fat diet, it showed a significant elevation ($p < 0.05$) in weights of liver, heart and abdominal fat than those of normal control. But mice treatment with (HFD+HS) showed significant ($p < 0.05$, $p < 0.01$) reductions in organ weights of liver, heart, and abdominal fat than that of HFD group, but no significant difference was noticed in (ND+HS) treated group compared with normal control (Figure 3A). In obese mice, significant ($p < 0.05$, $p < 0.01$) decrease in organ weights of liver, heart and abdominal fat were found both in (HFD+HS) and (ND+HS) treated groups than that of obese control (Figure 3B). There was no significant difference found in spleen weights in any of the groups.

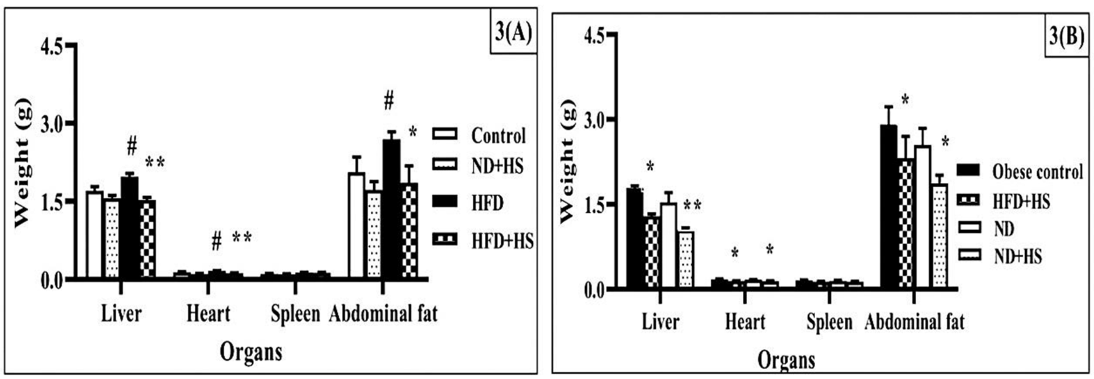

Figure 3. Effect of high-salt diet on organs weight. 3(A) non-obese group (#p <0.05 vs Control and *p < 0.05, **p < 0.01 vs HFD) and 3(B) Obese group (*p <0.05, **p <0.01 vs Obese control).

Effect of a high-salt diet on hyperlipidemia. The serum lipid profile was monitored in the different groups of mice. A significant level of elevation of serum triglyceride (TG), total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C), and reduction in high-density lipoprotein-cholesterol (HDL-C) was exhibited in HFD group than that of the control group. But treatment with high-salt reverts the phenomena significantly ($p < 0.05$) (Figure 4A, 5A, 6A, 7A). Furthermore, high salts were able to attenuate the serum lipids even in the obese group. Treatment with (HFD+HS) and (ND+HS) caused a significant ($p < 0.01$) level of declination in serum lipids except for HDL-C where the value increased (Figure 4B, 5B, 6B, 7B).

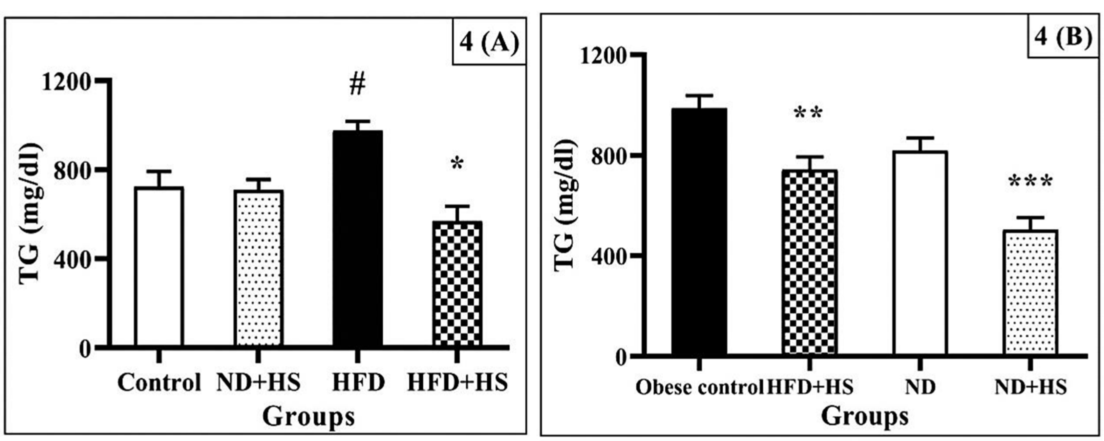

Figure 4. Effect of high-salt diet on TG level. 4(A) non-obese group (#p < 0.05 vs Control and *p <0.05 vs HFD)and 4(B) Obese group (**p < 0.01, ***p < 0.001 vs Obese control).

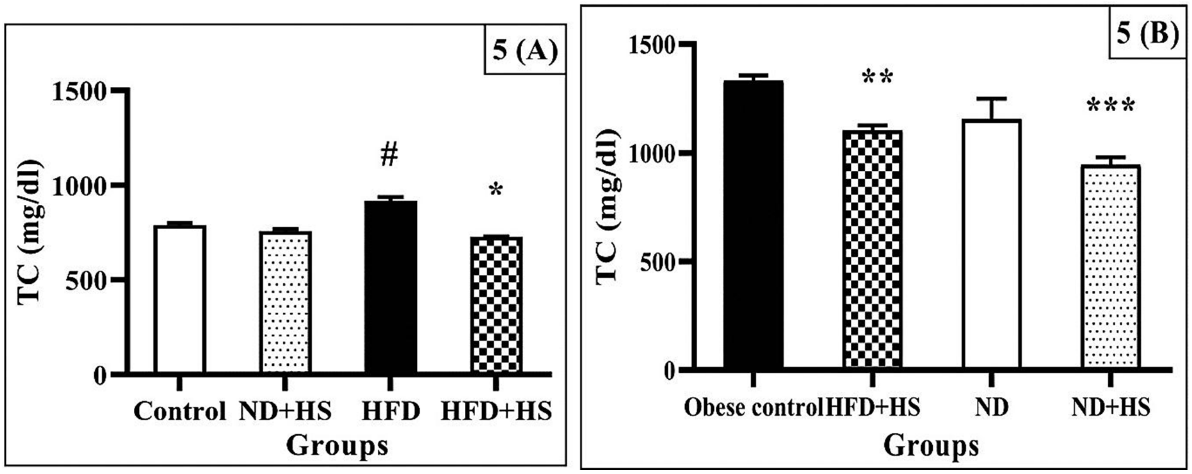

Figure 5. Effect of high-salt diet on TC level. 5(A) non-obese group (#p <0.05 vs Control and *p <0.05 vs HFD) and 5(B) Obese group (**p <0.01 and ***p <0.001 vs Obese control).

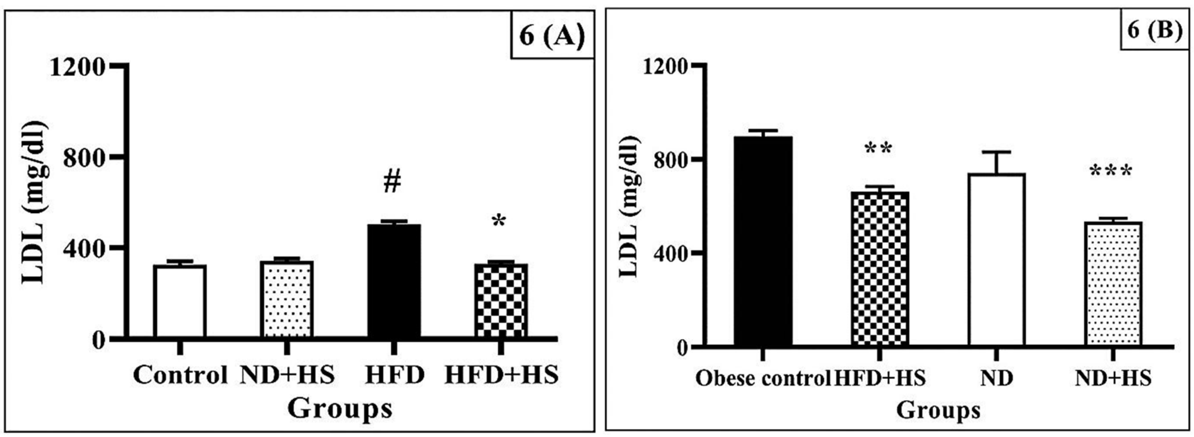

Figure 6. Effect of high-salt diet onLDL-C level. 6(A) non-obese group (#p <0.05 vs Control and *p <0.05 vs HFD) and 6(B) Obese group (**p <0.01, ***p <0.001 vs Obese controL.

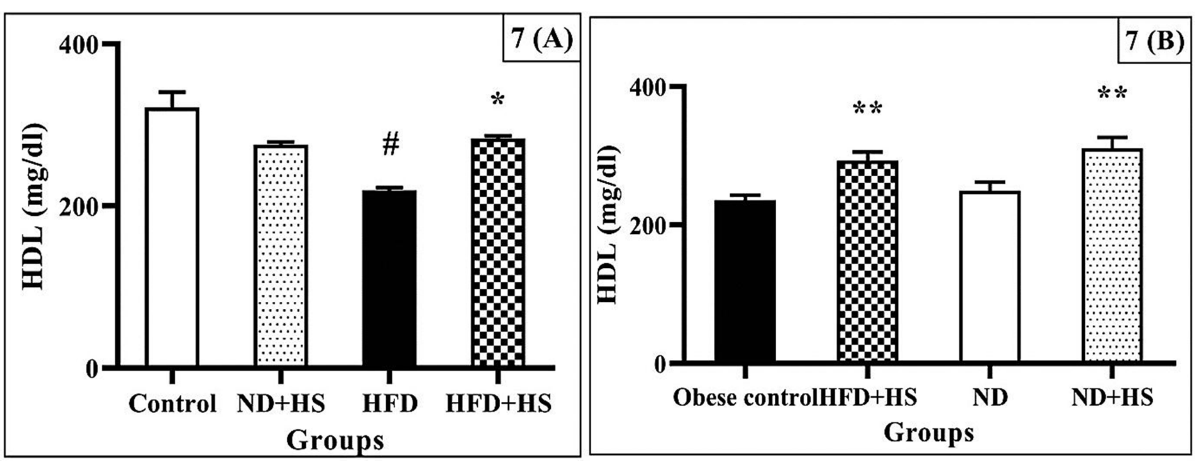

Figure 7. Effect of high-salt diet onHDL-C level. 7(A) non-obese group (#p <0.05 vs Control and *p <0.05 vs HFD) and 7(B) Obese group (**p <0.01 vs Obese control).

Effect of a high-salt diet on SGOT and SGPT. Serum SGOT and SGPT levels were significantly ($p < 0.05$) increased in the HFD group than that of the control group, but a significant ($p < 0.05$) reduction was noticed in (HFD+HS) group when compared with the HFD group. But high salt failed to reduce the levels in mice fed on a normal diet. As predicted, in the obese group, mice treated with high salts along with HFD or normal chow, showed a significant ($p < 0.05$) decrease in SGOT and SGPT levels as compared with the obese control. Data are presented in Figures 8A, 8B and 9A, 9B.

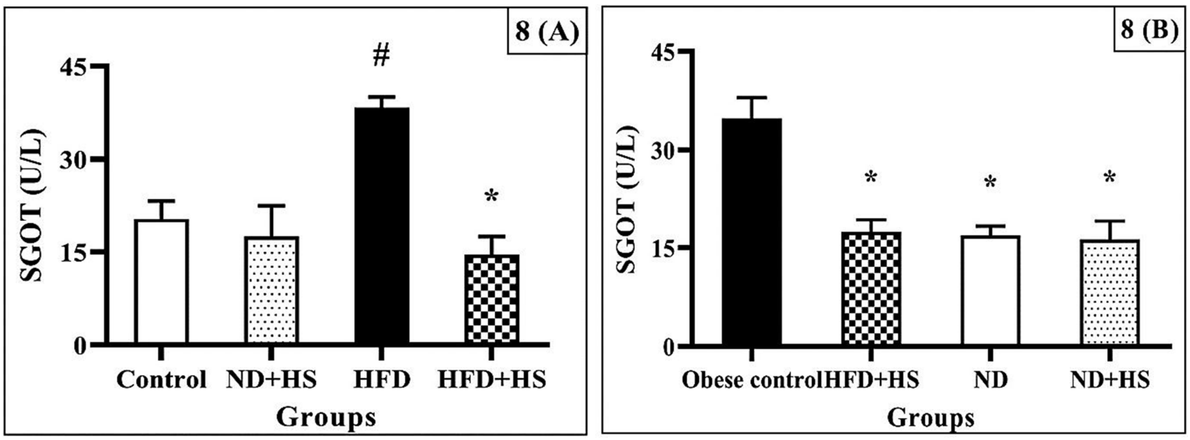

Figure 8. Effect of high-salt diet on SGOT level. 8(A) non-obese group (#p <0.05 vs Control and *p <0.05 vs HFD) and 8(B) Obese group (*p <0.05 vs Obese control).

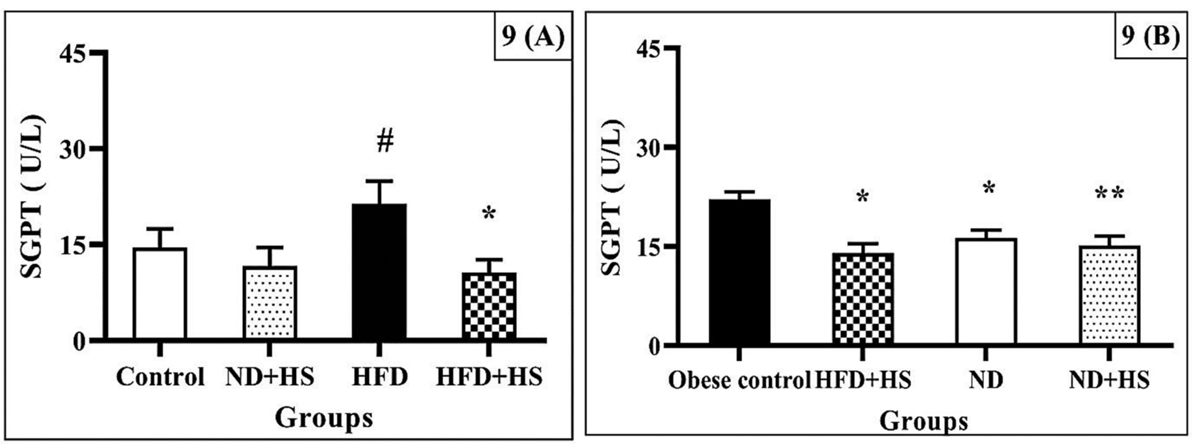

Figure 9. Effect of high-salt diet on SGPT level. 9(A) non-obese group (#p <0.05 vs Control and *p <0.05 vs HFD) and 9(B) Obese group (*p <0.05, **p <0.01 vs obese control).

Rodents are commonly used to study obesity because, like humans,[20,21] they readily gain weight on a high-calorie diet. Long-term high-fat diet (HFD) feeding induces obesity[22] in rodents, with body weight gain serving as a primary indicator. In this study, mice fed an HFD developed obesity within two weeks, which persisted throughout the experiment. High salt intake did not significantly affect the body weight of non-obese mice on a normal diet, consistent with mixed findings in previous reports.[23-24]

In this study, mice fed with HFD showed a significant increase in body weight than the mice on normal diet. Contrary to our initial hypothesis, when the high salt (4.0% NaCl) was added to HFD, a significant loss in body weight was noticed in the

HFD induced obese mice. Development of obesity was monitored by body weight gain and by measuring Lee index. After the development of obesity, obese mice were fed on normal chow or HFD with or absence of high salts. Interestingly we observed the body weight reducing tendency in obese mice on high salts along with HFD or normal chow compared with their counter parts. The reduced body weight was followed by the lowering of Lee index. Moreover, considering, waist circumference in lieu of body weight, to monitor the central obesity in rodents, we proposed a new index reported in our earlier study.[19] In this case, index was high in HFD group compared with normal diet but treatment with high salts reduced in index in similar fashion as observed in Lee index. These episodes in mice suggested that the high salt has remarkable capacity to prevent the body weight gain. Lowering of body weight may be attributed by the suppression of food intake by high salts diet but surprisingly we did not notice any remarkable differences in food intake behavior of the mice among the various groups studied. Excess fats in serum may be deposited in the liver, heart and abdomen. In this context, we measured the weight of these organs of the mice fed on different diets applied in this study. High fat diet increased the weight of liver, heart abdominal fat but the diet supplemented with high salts was able to reverse the effects of high fat diet significantly. This change in the body weight was supported by the change of abdominal fat pad weight in a similar fashion. In agreement with our study, Jin *et al.*[25] suggested that male rats fed on high fat and high salt (4.0%) has less epididymal fat than the rat on high fat only. A similar reduction of body weight in mice fed on high salt and high fat was investigated in several studies,[15,26,27] but contradicts others.[28,29] However, we did not exclude the possibility of lower salt content (2.0%) than we used could increase the adiposity as described by Dobrian *et al.*[30] It is noteworthy that in Japan, where the average consumption of salt is high having a lowest rate of obesity in the world.[31,32] Although we did not monitored the mechanism of body weight reduction in our study but reduction of fat pad weight along with other organ may be underlying cause of the reduction of body weight in our study. Moreover, rennin-angiotensin mediated reduction in digestive efficiency described by Weidemann *et al.*[26] as an evidence for reduction of bodyweight and adiposity in male mice fed on high fat and high salt cannot be overruled. Thus the weight loss in obese mice fed high salt may be associated with inherent capacity of high salts.

Furthermore, hyperlipidemia, characterized by hypertriglycemia and hypercholesterolemia, is another evidence for the development of adiposity. Long chain fatty acids especially eicosanoids are known as the natural ligand for the PPARγ, the master player of adipogenesis.[33] Long-term consumption of a high fat diet leads to an imbalance in serum lipid profiles which is characterized by elevated serum TG, TC and LDL-c levels and reduced HDL-c levels and this imbalance is a major risk factor for cardiovascular disease and atherosclerosis.[34,35] To assess the effect of high salts in lipid profile we measured the serum TG, TC, HDL-c in our experimental model. As expected, in our study, mice on HFD diet showed a notable elevation in TGs, TC, LDL levels than that of control group. Whereas significant reduction in the lipid profiles were observed in mice fed on HFD supplemented with high salt consistent with the reduction of body weight. Role of salt in regulation of lipid metabolism is not well understood. He *et al.*[4] reviewed as no significant changes in lipid profile in association with salt intake but Grdual *et al.*[13] reported that low salt in diet increase the level of cholesterol and triglyceride. Additionally, inverse association with serum cholesterol and salt intake in hypertensive obese women has been reported by Padilha *et al.*[14] The mechanism of lipid lowering effects of high salt may partly be attributed by epinephrine[13,36] and insulin resistance via rennin-angiotensin-aldosterone system which eventually reduces the Glucose transporter type-4 (GLUT4) translocation to cell membrane.[37] High salt intake increases the epinephrine concentration leading to inhibit the insulin action and causing insulin resistance. Insulin resistance stimulates lipolysis and release free fatty acid from adipose tissue decreasing the concentration of triglyceride and cholesterol in blood.[38] Moreover high salt intake increases the plasma leptin concentration,[39] which is also responsible for insulin resistance in obesity.

High fat diet allows animals to develop liver diseases similar to the phenotype observed in human with nonalcoholic fatty live disease (NAFLD).[40] Fatty liver is developed by the deposition of fats in liver, changes the liver function and the serum level of hepatic enzymes is used as indictor of liver activity.[41,42] Considering this, we examined the serum level of SGOT and SGPT. In our study, HFD was found to elevate the serum SGOT and SGPT significantly but high salt diet was able to attenuate the elevated serum SGOT and SGPT levels significantly. Although, the cause behind the ameliorating effects of high salt in NAFLD is not clear and our results contradicts others[43] but we speculate that its action may be partly attributed by the reductions in triglycerides and cholesterol. Moreover, increased energy expenditure due to increased plasma osmolality by high-salt might also be accounted for decreasing serum triglyceride and cholesterol in obese mice. Figure 10 shows the proposed mechansim.

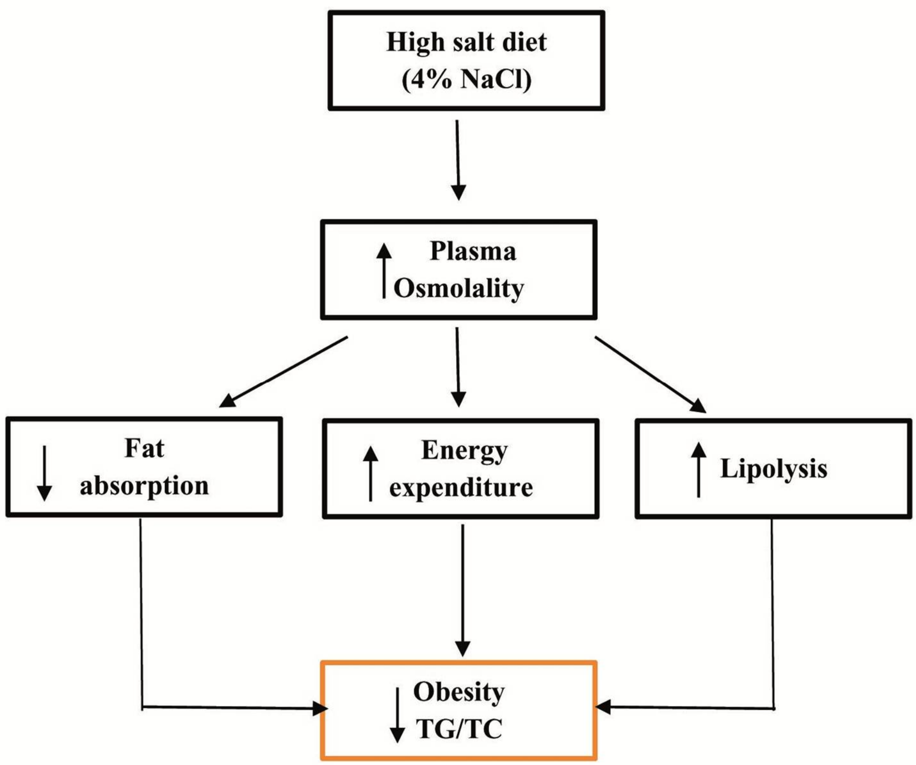

Figure 10. Proposed mechanism.

CONCLUSION

In conclusion, our study revealed that high salt has an inhibitory action on adiposity and effective to attenuate the elevated serum lipids that are most evident in high fat dietary conditions. The underlying mechanism of these effects of high salt on adiposity and related consequences in this study is unknown but warrants further investigation. If the clear mechanism responsible for the absence of adiposity in the presence of a high-salt diet is understood, a new intervention may be developed to combat obesity and related complications.

Conflict of Interest

None.

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