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Research Article

Alterations in Atherogenic Risk Predictor Indexes from Administration of Monosodium Glutamate (MSG) in Lactating Wistar Rats

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Abstract

The concomitant increased risk of cardiovascular diseases has been associated with hyperlipidemia among other things, making it a perpetual area of research interests. MSG has been reported to disrupt hypothalamic functions thus precipitating metabolic imbalances in forms of obesity etc. As a result, this study was aimed at evaluating changes in lipid profiles and atherogenic risk predictor indices in lactating Wistar rats. Twenty-four (24) lactating rats were grouped into 4 consisting of six animals each (n = 6) thus; Group I served as normal control and was given distilled water (1 ml/kg BW), while group II was given metoclopramide (5 mg/kg BW). Groups III and IV were administered MSG at 1850 and 3700 (mg/kg BW) respectively for 14 days. Animals were sacrificed and blood samples obtained. Lipid profiles of animals were assayed from the sera and the ratios estimated. Total cholesterol and triglycerides were significantly ($P < 0.05$) increased in MSG treated groups compared to the control. LDL-c was increased in all MSG treated groups compared to control, however, this change was only significant in the MSG (3700 mg/kg BW) treated group. HDL-c decreased in all MSG treated groups compared with control, although this change was not statistically significant. Castelli's risk ratios I and II, atherogenic coefficient and atherogenic indexes increased with MSG treatment. In conclusion, the result of this study shows increased predisposition of lactating mothers to cardiovascular complications with increased consumption of MSG.

Keywords: *Atherogenic Risk Predictors; Lipid Profiles; Lactation; Monosodium Glutamate*

Introduction

Cardiovascular disease is regarded as one of the common health complications in a given community and the leading cause of death globally. According to World Health Organization (WHO). 23.3 million people will die from cardiovascular diseases by the year 2030 [1]. Damages to endothelial cells can result from abnormal lipid profiles causing endothelial dysfunction, reduced vasodilatory ability thus allowing lipids to pass through endothelial layers [2]. Hyperlipidemia; a lipoprotein metabolic disorder is characterized by high serum low density lipoprotein (LDL) and low serum high density lipoprotein (HDL) [3]. It is also referred to as hyperlipoproteinemia owing to fatty acids mobilization in the blood attached to proteins [4]. Conditions like hypercholesterolemia is associated with more hypertensive

patients in African populace [5]. Excess LDL cholesterol have been implicated in heart attacks through blockage of arteries. Heart disease often present with high triglyceride levels combined with low HDL cholesterol or high LDL cholesterol seems to speed up atherosclerosis, which is the buildup of fatty deposits in artery walls that increase the risk for heart attack and stroke [6]. It may develop from unbalanced diet, genetic predispositions and obesity among others [7,8]. Approximately 100 million people in the united states suffered from hypercholesterolemia (> 5.2 mmol/L) in 2008. Hypercholesterolemia especially elevated low-density lipoprotein (LDL) cholesterol is a major risk factor for the development of atherosclerosis and subsequent ischemic disease which is a leading cause of death worldwide [9]. MSG induces appetite positively and stimulates weight gain due to its irritation of the sensory receptors and enhancing the palatability of food [10]. Disruption of the brain hypothalamus areas controlling body mass and energy metabolism is strongly involved in inducing several metabolic diseases in the MSG-induced animal model [11-13]. Lactating mothers usually present with increased consumption of delicacies which are prepared using MSG in our locality thus predisposing them to high consumption rate. Metoclopramide is used as a galactagogue [14]. Although quite a few of literatures have reported the use of metoclopramide as galactagogue from its action od D_2 dopamine receptors, there is paucity of information of its effect on lipid profiles in lactating rats.

Aim of the Study

Therefore, this study aimed at evaluating changes in atherogenic indexes in lactating rats.

Materials and Methods

Four (4) transparent white plastic cages, water bottles and feeding troughs, syringes, cotton wool, oral cannula, antiseptic, hand gloves, plain bottles, pipettes, electronic weighing machine, centrifuge (bench top), dissecting kit, ketamine and diazepam, monosodium glutamate, metoclopramide hydrochloride (10 mg) (NAFDAC REG NO: 04-6476), digital weighing balance (0.01 sensitivity) distilled water and monosodium glutamate.

Experimental animals

A total of 24 nulliparous adult female Wistar rats and twelve (12) adult male Wistar rats were used for the study. The male rats were used for the purpose of mating with the females. Animals with body weight (130 - 200g) were sourced from the Department of Human Physiology, Ahmadu Bello University animal house. These animals were housed in plastic cages with adequate air vents. Soft sawdust material was utilized for bedding with free access to food and water throughout the period of study.

Ethical approval

Handling of laboratory animals was carried in accordance with the guidelines of the National Institute of Health on care and use of laboratory animals. Local Institutional ethical approval for the use of laboratory animals for research was obtained from the Ahmadu Bello University ethical committee on animal use and care with approval number: ABUCAUC/2018/092.

Experimental design and animal groupings

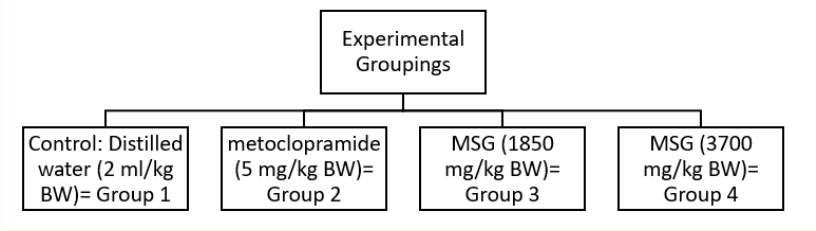

Figure 1: *Experimental groupings. Animals were treated orally for 14 days using an oral cannula. Doses of MSG used were adopted from [15].*

Preparation of drug

A fresh stock concentration was prepared daily in distilled water thus 100 mg/mL stock concentration was formed from 1000 mg of MSG dissolved daily in 10 ml of distilled water. Monosodium glutamate (Ajinomotto) was sourced from Samaru local market of Sabon Gari LGA, Kaduna state Nigeria.

Animal sacrifice and sample collection

Ketamine and diazepam at 75 and 5 (mg/kg) were administered intraperitoneally at the end of the experiment as the anaesthetic agents. Blood samples were collected via cardiac puncture using 5 ml syringes and emptied into plain tubes and the sera separated afterwards by centrifugation at 3,000g for 10 minutes.

Determination of lipid profiles

The serum cholesterol was determined according to the method of Meiattini *et al*. [16] and Allain *et al*. [17]. Serum high density lipoprotein was determined by the methods of Burstein., *et al*. and Grove [18,19]. Triglyceride was determined according to the method described by Friedman and Young [20]. Serum low density lipoprotein was determined according to the method described by Salah., *et al* [21].

Estimations of atherogenic index and lipid ratios

The atherogenic index and lipid ratios in this study were obtained using the following established formulas as described by Akpinar., *et al*. [22] and Bhardwaj., *et al* [23]. Castelli's risk ratios I and II were estimated as (TC/HDL-c and LDL-c/HDL-c) respectively. Atherogenic coefficient was estimated as (TC - HDL-c / HDL-c) while Atherogenic index was estimated as Log [TG/HDL-c].

Statistical analyses

Data obtained from the study were expressed as mean ± SEM and the statistical analysis was carried out using version 20 of SPSS with the aid of one-way analysis of variance (ANOVA) followed by Tukey post hoc test. Values with $P < 0.05$ were considered statistically significant. Scatter plots were drawn using Excel and the linearity between variables observed. Pearson correlation was carried out for all variables with scatter points close to a straight line.

Results

Effect of MSG on total cholesterol of lactating wistar rats

Cholesterol (mg/dl) was statistically significantly higher [$F = (3, 12) = 23.012$; $P = 0.001$] in MSG (3700 mg/kg) treated groups compared to control and metoclopramide treated groups; 149.10 ± 1.56 (mg/dl) vs 109.48 ± 2.87 (mg/dl) vs 114.03 ± 6.25 (mg/dl) respectively. More so, cholesterol (mg/dl) was also statistically significantly increased [$F = (3, 12) = 23.012$; $P = 0.001$] in the MSG (1850 mg/kg) compared to metoclopramide treated group; 124.83 ± 2.19 (mg/dl) vs 114.03 ± 6.25 (mg/dl) respectively.

Effect of MSG on triglyceride of lactating wistar rats

Triglyceride (mg/dl) was statistically significantly higher [$F = (3, 12) = 8.888$; $P = 0.002$] in MSG (3700 mg/kg) treated groups compared to control and metoclopramide treated groups; 122.85 ± 4.01 (mg/dl) vs 104.78 ± 3.27 (mg/dl) vs 101.27 ± 2.20 (mg/dl) respectively. There was also statistically significant increase [$F = (3, 12) = 8.888$; $P = 0.002$] in the MSG (1850 mg/kg) compared to metoclopramide treated group; 118.80 ± 4.25 (mg/dl) vs 101.27 ± 2.20 (mg/dl) respectively.

Effect of MSG on low density lipoprotein cholesterol (LDL-c) of lactating wistar rats

There was statistically significant increase [F = (3, 12) = 5.207; *P* = 0.016] of LDL-c (mg/dl) in the MSG treated groups compared to both control and metoclopramide treated groups viz; MSG (1850 mg/kg) vs control vs metoclopramide (52.28 ± 1.10 mg/dl vs 43.15 ± 1.85 mg/dl vs 44.75 ± 2.30 mg/dl). MSG (3700 mg/kg) vs control vs metoclopramide (51.93 ± 2.73 mg/dl vs 43.15 ± 1.85 mg/dl vs 44.75 ± 2.30 mg/dl).

Effect of MSG on high density lipoprotein cholesterol (HDL-c) of lactating wistar rats

There was a non-significant decrease [F = (3, 12) = 1.371; P = 0.299] in HDL-c (mg/dl) in metoclopramide and the MSG treated groups compared to control; metoclopramide vs control (37.43 ± 2.41 mg/dl vs 40.18 ± 1.72 mg/dl), MSG (1850 mg/kg) vs control (36.83 ± 3.92 mg/dl vs 40.18 ± 1.72 mg/dl) and MSG (3700 mg/kg) vs control (31.93 ± 3.18 mg/dl vs 40.18 ± 1.72 mg/dl).

Effect of MSG on cardiac risk ratios of lactating dams

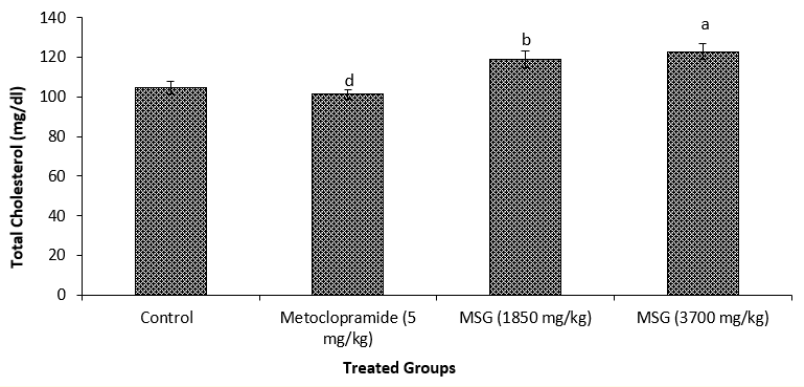

Figure 2: *Total cholesterol level of lactating dams. MSG = monosodium glutamate. One-way analysis of variance (ANOVA) followed by Tukey post hoc test for multiple comparisons were carried out. Superscripts (a) (b) and (d) indicate statistically significant difference (P < 0.05) compared to control, metoclopramide and MSG (3700 mg/kg) groups respectively.*

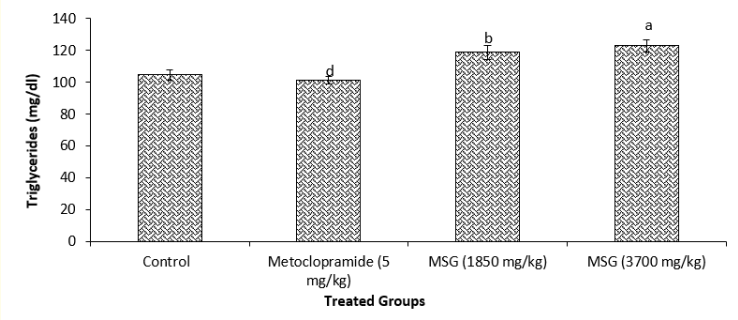

Figure 3: *Triglyceride level of lactating dams. MSG = monosodium glutamate. One-way analysis of variance (ANOVA) followed by Tukey post hoc test for multiple comparisons were carried out. Superscripts (a) (b) and (d) indicate statistically significant difference (P < 0.05) compared to control, metoclopramide and MSG (3700 mg/kg) groups respectively*

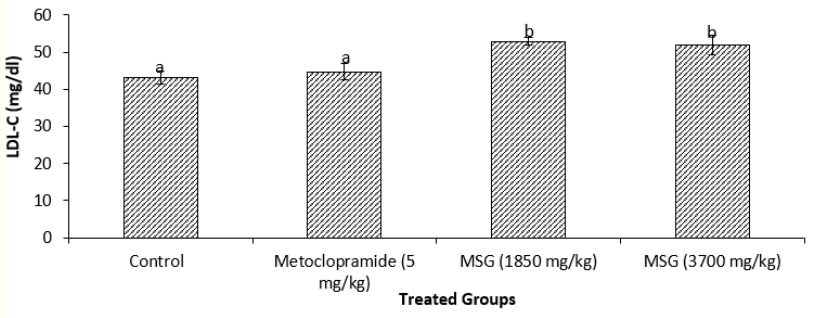

Figure 4: *LDL-c of lactating dams. MSG = monosodium glutamate, LDL-C = low density lipoprotein cholesterol. One-way analysis of variance (ANOVA) followed by Tukey post hoc test for multiple comparisons were carried out. Superscripts (a) and (b) indicate statistically significant difference (P < 0.05) compared to control and metoclopramide groups respectively.*

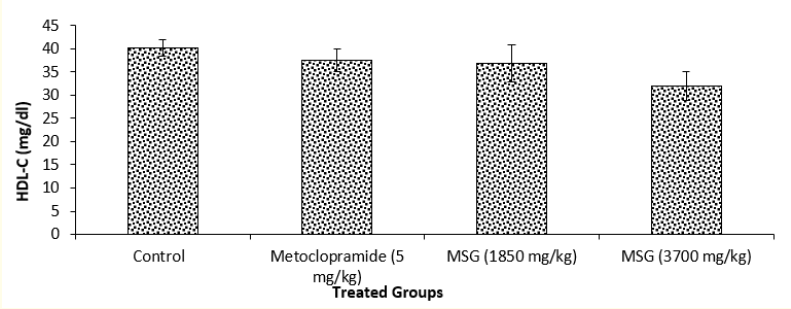

Figure 5: *HDL-c of lactating dams treated with metoclopramide and MSG for 14 days. MSG = Monosodium glutamate, HDL-c = High density lipoprotein cholesterol. One-way analysis of variance (ANOVA) was carried out.*

There was statistically significant increase in the Castelli's risk index-I [F = (3, 12) = 3.389; *P* = 0.044] in MSG (3700 and 1850 mg/kg) compared to control. There was also statistically significant increase in Castell's risk index-II [F = (3, 12) = 3.500; P = 0.05] in MSG (3700 mg/kg) compared to control. However, there was no statistically significant difference in atherogenic coefficient [F = (3, 12) = 3.391; P = 0.054]. There was statistically significant difference [F = (3, 12) = 12.630 P = 0.0001] in atherogenic index of all the groups compared to MSG (3700 mg/kg). There was statistically significant difference [F = (3, 12) = 8.775; P = 0.001] in body weight (g) as well as in the heart weight of treated groups compared to control and metoclopramide [F = (3, 12) = 15.956; P = 0.0001].

Effect of MSG on correlation between HDL-c (mg/dl) and atherogenic index of lactating dams

There was a statistically significant strong negative correlation between atherogenic index of plasma of lactating dams and the HDL-c (mg/dl) [r = - 0.895; P = 0.0001].

Animal Groups	Castelli's Risk Index-I	Castelli's Risk Index-II	Atherogenic Coefficient	Atherogenic Index
Control	2.71 ± 0.11	1.08 ± 0.09	1.74 ± 0.11	0.32 ± 0.02
METO (5 mg/kg)	3.10 ± 0.33	1.21 ± 0.10	2.11 ± 0.33	0.41 ± 0.02[d]
MSG (1850 mg/kg)	4.16 ± 0.35	1.47 ± 0.16	3.17 ± 0.36	0.42 ± 0.03[d]
MSG (3700 mg/kg)	4.08 ± 0.59	1.68 ± 0.19	3.09 ± 0.59	0.59 ± 0.05[a]

Table 1: *Atherogenic indexes.*

METO: Metoclopramide; MSG: Monosodium Glutamate; SEM: Standard Error of Mean. Analysis was carried out using SPSS version 20. Data were analyzed using one--way analysis of variance (ANOVA) followed by Tukey post hoc test for multiple comparison. Data are represented as MEAN ± SEM. Superscripts (a) (b) and (d) indicate statistically significant difference (P < 0.05) compared to control, metoclopramide and MSG (3700 mg/kg) groups respectively.

Effect of MSG on correlation between LDL-c (mg/dl) and atherogenic index of lactating dams

There was a statistically significant positive correlation between atherogenic index of plasma of lactating dams and the LDL-c (mg/dl) [r = 0.523; P = 0.019].

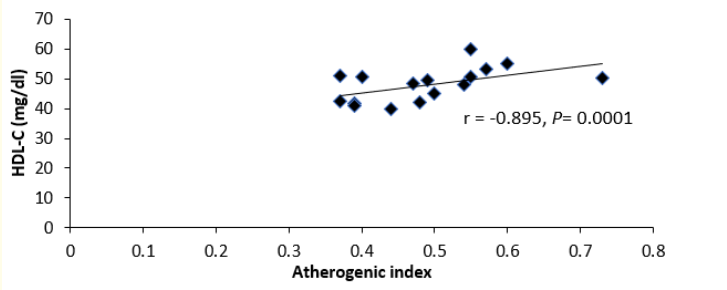

Figure 6: *Correlation between atherogenic index and HDL-c (mg/dl) of lactating dams treated with metoclopramide and MSG for 14 days. Pearson correlation was carried out.*

Effect of MSG on correlation between total cholesterol (mg/dl) and atherogenic index of lactating dams

There was a statistically significant positive correlation between atherogenic index of plasma of lactating dams and total cholesterol (mg/dl) [r = 0.510; P = 0.02].

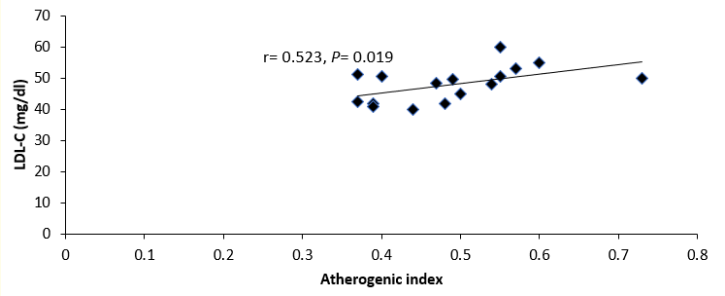

Figure 7: *Correlation between atherogenic index of plasma and LDL-c (mg/dl) of lactating dams treated with metoclopramide and MSG for 14 days. Pearson correlation was carried out.*

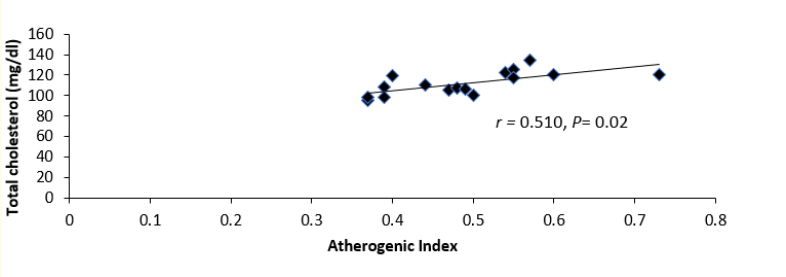

Figure 8: *Correlation between atherogenic index of plasma and total cholesterol (mg/dl) of lactating dams treated with metoclopramide and MSG for 14 days. Pearson correlation was carried out.*

Effect of MSG on correlation between triglycerides (mg/dl) and atherogenic index of lactating dams

There was a statistically significant strong positive correlation between atherogenic index of plasma and triglycerides of lactating dams [r = 0.659; P = 0.003].

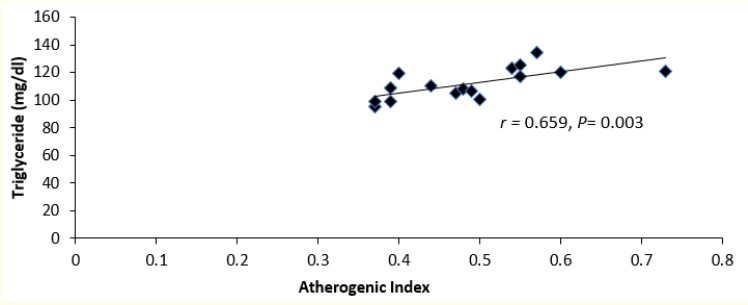

Figure 9: *Correlation between atherogenic index of plasma and triglycerides (mg/dl) of lactating dams treated with metoclopramide and MSG for 14 days. Pearson correlation was carried out.*

Discussion

There have been reports of positive benefits of lactation exercises on maternal lipid profiles and fat distribution as lactating mothers exhibit a less atherogenic lipid profile and increased fat mass mobilization especially during the first year postpartum [24,25]. In this study, serum total cholesterol increased with MSG treatment. This result agrees with Singh., *et al*. [26] who reported increased total cholesterol with oral MSG consumption. However, it is imperative to note that their study was not conducted with lactating animals. Oxysterols which are oxygenated derivatives of cholesterol have been reported to inhibit biosynthesis of cholesterol oxidized in the liver [27,28]. Ortitz., *et al*. [29] have reported damages to the liver from monosodium glutamate consumption. Thus, the result of total cholesterol in this study could have been indirectly caused by the negative impact of MSG oral consumption on optimal hepatic functions in these lactating rats. Triglycerides which are the main constituents of body fats, present in the blood play the role of bidirectional transference of adipose fats as well as blood glucose from the liver [30]. The increase observed in this study from treatments of MSG could suggest a possible

interference of these substances with the pancreas. There have been reports of MSG dietary consumption decreasing pancreatic β-cell in rats [31]. Pancreatic lipase is also referred to as pancreatic triacylglycerol lipase which hydrolyzes dietary fats thus, aiding the conversion of triglycerides substrate. Hassah et al. [33] have also reported physiochemical changes in pancreatic histology with MSG treatment in rats with increased plasma activities of lipase and amylase owing to gross and microscopic lesions in the pancreas. LDL cholesterol often called bad cholesterol plays the role of conveying cholesterol from the liver to the cells. High concentration of LDL-c is a predisposition to arterial diseases. The results of LDL-c and HDL-c in this study is in concert with Singh., *et al*. [26] who reported hyperlipidemia and hyperlipoproteinemia with oral MSG consumption as possible initiators of atherosclerosis. In this study, oral administration of MSG and metoclopramide presents with characteristics consistent with onset of cardiac complications. Atherogenic index derived from triglycerides and HDL-c has been employed to measure blood lipids levels. It is an indicator of dyslipidemia and associated cardiovascular complications [34]. Atherogenic Index has also been reported to be correlated with the other indexes like LDL-c [35]. The result of Atherogenic index in this current study suggests oral consumption of MSG at high dosage increases predisposition to cardiac complications stemming from dyslipidemia associated with lactation in rats. Although during pregnancy, maternal modifications in hormones among other things could precipitate hyperlipidemia considered as physiological [36], this condition is usually reversed during lactation period except with exogenous interference as in the case of this study. Castelli's risk ratio-I also referred to as cardiac risk ratio-1 (CRR) and Catelli's risk ratio II are both fractions used in assessment of coronary artery diseases (CAD). The increased CRR-I and CRR-II from treatment with MSG in this study suggests a possible increased risk of CAD with higher consumption of MSG during lactation. The strong negative correlation between atherogenic index and HDL-c in this study is in concert with literatures. This result implies a decrease in atherogenic index with increasing HDL-c (good cholesterol). However, there is a weak positive correlation between atherogenic index and LDL-c in lactating rats following oral MSG administration. This implies increasing atherogenic index with increase in LDL-c (bad cholesterol). Thus, oral consumption of MSG at high doses during lactation should be with caution if not avoided at all. Further studies should be carried out on the possible molecular mechanisms of MSG actions on lipid profiles during lactation and its neurotoxic effects and detrimental effects on the reproductive organs. The use of metoclopramide as a galactagogue in this study did not present with any significant changes in lipid profiles and atherogenic indexes in lactating rats.

Conclusion

The findings of this study demonstrate the risk of cardiac complications associated with oral consumption of MSG in high quantity during lactation as indicated by:

1. Increasing serum total cholesterol, triglyceride, LDL-c, atherogenic index, cardiac risk ratios I and II.
2. Decreasing serum HDL-c level in lactating Wistar rats.

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