Journal of Biosciences and Medicines, 2023, 11, 196-209
https://www.scirp.org/journal/jbm
ISSN Online: 2327-509X
ISSN Print: 2327-5081

The Main and Interactive Effects of Fat and Salt Contents of the Diet on Characteristics of Metabolic Syndrome in Male Wistar Rats

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How to cite this paper: Jahan-Mihan, A., Schwarz, K., Ninya, L. and Kimble, T. (2023) The Main and Interactive Effects of Fat and Salt Contents of the Diet on Characteristics of Metabolic Syndrome in Male Wistar Rats. *Journal of Biosciences and Medicines*, **11**, 196-209.
https://doi.org/10.4236/jbm.2023.116016

Received: April 26, 2023
Accepted: June 23, 2023
Published: June 26, 2023

Abstract

The current Western diet contains high amounts of salt and fat. High salt and fat diets are known to have negative impacts on food intake (FI), body weight (BW), body composition (BC), glucose metabolism, and blood pressure. These factors have been studied as separate entities, but the main and interactive effects of dietary salt and fat received little study. The objective of this study was to examine the effect of sodium and fat content of the diet on FI, BW, and BC in male Wistar rats. Male Wistar Rats (n = 48) were allocated into 4 groups (n = 12) and received the following diets: 1. Normal sodium normal fat, 2. Normal sodium high fat, 3. High sodium normal fat, and 4. High sodium high-fat diet for 12 weeks. BW and FI were measured weekly. BC and organs' weight were recorded post-termination. Regardless of sodium content, a greater FI was observed in normal-fat diet groups compared with high-fat diet groups. However, higher BW and fat (%) were observed in high-fat diet groups. Fasting blood glucose was higher in rats fed normal fat diets compared with those fed high-fat diets. Systolic and diastolic blood pressure was lower in rats fed either high fat, high salt, or normal fat, normal salt diet. In conclusion, fat but not salt content in the diet is a determining factor in the regulation of FI and body weight. Moreover, glucose metabolism can be influenced by both the fat and salt content of the diet.

Keywords

Fat, Sodium, Food Intake, Body Weight, Metabolic Syndrome, Blood Pressure

1. Introduction

Obesity is an exponentially growing health burden worldwide. Currently, two-thirds

of the population in the United States are either overweight or obese [1]. Obesity comorbidities include dyslipidemia, high blood pressure, high cholesterol, glucose intolerance, and excess abdominal fat which are known as characteristics of metabolic syndrome [2]. The Western diet is characterized by its high fat and high sodium content. This diet is a major contributing factor in developing obesity and metabolic syndrome. The mean daily sodium (Na^+) intake of Americans is about 3500 mg/d which is far beyond the recommended level: ≤2300 mg/d [3]. The mean consumption of fat is 34.6% and 35.1% of the total calorie intake in American males and females respectively [4]. Excessive intake of sodium and fat is strongly correlated with a higher risk of stroke, hypertension, and heart disease. Moreover, high salt and fat diets have negative impacts on body weight and composition [5] [6], glucose homeostasis [7], inflammatory markers [8], circadian locomotor activity [9], and satiety hormones [5]. These factors have been studied as separate entities, but little research has been done on the impact of the interaction of these nutrients. The obesogenic effect of a high-fat diet has been proven [10] [11]. A high-fat diet resulted in obesity in rats and in humans [12] [13] [14]. However, the effect of fat on food intake is more compound and is influenced by several factors including the food matrix. Although both fat and salt are appetitive, the effect of either on food intake and appetite is ambiguous. A high-fat, normal sodium diet resulted in the least amount of food eaten, but with the greatest overall energy intake. Similarly, rats fed a high-sodium and high-fat diet also ate less compared to those eating a normal diet [15]. It can be explained by the fact that fat taste sensitivity has a suppressing effect on dietary fat intake [16]. In one study, salt increased food and energy intake while fat had no effect on food intake in healthy adults [16]. Authors suggested that salt increases passive energy intake in adults and it may override fat-mediated satiation in subjects who are sensitive to the taste of fat. In contrast, Blundell *et al.* suggested that foods with high dietary fat have a weak satiation effect in the short term, which leads to overconsumption (high-fat hyperphagia) [17]. In obese women, a high-CHO lunch and mid-afternoon snack led to less energy intake compared with a high-fat lunch. However, post-meal satiety was similar [18]. In another study, high-fat foods led to passive overconsumption in young male subjects which generate relatively weak satiety [19]. This effect can be influenced by the high energy density of the meal and the high palatability of high-fat foods. This discrepancy among studies can be due to different mechanisms regulating short- vs. long-term food intake. While the short-term food intake regulatory system is regulating meal size and meal frequency, food intake in long term is influenced by more systematic signals including the level of fat storage in the body [19] [20].

We used Wistar rats as a model for this study. Wistar rats have been used in numerous studies examining metabolic syndrome and food intake regulation due to similarity in metabolic and physiologic mechanisms to human. Moreover, a well-controlled environment helps to minimize the effect of outliers.

Therefore, the objective of this study was to examine the main and interactive effects of dietary fat and salt on food intake, body weight, and characteristics of metabolic syndrome in male Wistar rats. We hypothesized that dietary salt and fat have synergistic effects promoting food intake, body weight, and characteristics of metabolic syndrome in male Wistar rats.

2. Materials and Methods

Ethical statement

The experiment with Wistar rats followed the National Institutes of Health guide for the care and use of laboratory animals (NIH Publications No. 8023, revised 1978). All experimental procedures involving animals were approved by the University of North Florida Institutional Animal Care and Use Committee (IACUC) (Protocol No: IACUC#18-002).

Experimental design

A power analysis was performed based on data from a previous study [21] and based on statistical power (80%) and the two-sided significance level (0.05). Male Wistar rats (n = 48, BW: 115.33 ± 1.87 g) were allocated to four groups (n = 12/group) and received one of the following treatments for 12 weeks: 1. High salt, high-fat diet (HSHF), 2. High salt, normal fat diet (HSNF), 3. Normal salt, high-fat diet (NSHF) and Normal salt, normal fat diet (NSNF). Body weight (BW) and food intake (FI) were measured on a weekly basis. Moreover, short-term FI (1, 2, and 12 hours) was measured at week 12. Systolic and diastolic blood pressure was measured at weeks 1, 4, 8, and 12. An oral glucose tolerance test was also conducted at weeks 1, 4, 8, and 12. At the end of the study, following 12 hours of fasting, rats were euthanized. Body composition was calculated based on fat and lean mass at the end of the study.

Animals and diets

Male Wistar rats were housed individually in plastic cages with ventilation at 22˚C ± 1˚C and a 12-hour dark-light cycle (lights on at 2100 hours and off at 0900 hours) with free access to water and food. Wellbeing of rats were assessed through physical assessments that were carried out prior to, during, or after procedures. No substantial adverse events were notified in any group throughout the study.

Standard diets (AIN-93G) were purchased from Dyets (Dyets Inc. Bethlehem, Pa, USA). The compositions of the diets (per kg) are illustrated in **Table 1**. Salt was manually added to high-salt diets. Morton non-iodized table salt (590 mg Na/1.5 g salt; purity: 99%) was purchased from a Walmart store. The final salt content of normal and high salt diets was 0.5% and 4% respectively.

2.1. Procedures

Long- and short-term food intake

Long-term FI was measured by weighing the food containers at the beginning and at the end of each week throughout the study. Diets were provided ad

Table 1. Composition of the Normal and High fat Diets.

	Normal Diet	High Fat Diet
Cal	3597	3963
	g/kg	
Casein	200	200
Sucrose	92.7	68.8
Soybean oil	40	25
Lard	0	245
t-Butyhydroquinone	0.008	0.005
Cornstarch	426.79	0
Dyetrose	140	125
Cellulose	50	50
Mineral Mix	35	10
Calcium Carbonate	0	5.5
Potassium Citrate	0	16.5
Vitamin Mix	10	10
Choline Bitartrate	2.5	2
L-Cystine	3	3

*Salt was manually added to high-salt diets (40 g/kg diet).

libitum in jars. Spillage was collected, measured, and subtracted to obtain the actual intake.

Short-term FI was also measured at week 12. After an overnight fast (12 hours), food intake at 1, 2, and 12 hours was measured.

Blood glucose measurement

As previously described [22], tail vein glucose concentration was assayed using a handheld commercial glucometer (Contour® Next Blood Glucose Meter, Bayer Healthcare LLC, Mishawaka, IN, USA) using test strips. The accuracy and variance of the glucometer and test strips were examined by applying control solutions (levels 1 and 2) provided by the manufacturer (Bayer, Bayer Healthcare LLC, Mishawaka, IN, USA).

Blood pressure measurement

As previously described [23], blood pressure and pulse were measured by BP 2000 Visitech system™ through a non-invasive optical plethysmography tail-cuff method. This equipment has been utilized in many other studies including our previous studies.

Rats were held individually in a restrainer on a warmed platform (30˚C) which is a relaxing temperature for rats. The tail was stabilized gently and blood pressure was measured through a non-invasive tail-cuff method. At each time point, BP was measured 10 times (the first 5 times were mock measurements as adaptation and were not recorded and the next 5 times were recorded as actual

measurements). A computer derived the inflation and deflation of the tail cuff, as a sensor positioned at the base of the tail detected when blood flow started and stopped.

Oral glucose tolerance test protocol

Rats fasted overnight for 12 hours. A blood sample was withdrawn from the tail vein before glucose administration and at 15-, 30-, and 60-minutes following glucose administration (0·375 g glucose/ml, 5 g glucose/kg BW).

Body composition

Fat mass (FM) and lean mass were measured following euthanasia at week 20. FM was measured by dissection of extracted abdominal, epididymal, and perirenal fat.

2.2. Statistical Analysis

Data are expressed as means with standard errors. The effect of the fat and salt and their interactions on BW, FI, SBP, and DBP, oral glucose tolerance test (OGTT) was analyzed by two-way ANOVA. When repeated measures were made over time on BW, and food intake, the PROC MIXED MODEL procedure was used with fat, salt, and time as the main factors. When interactions were statistically significant, a one-way ANOVA followed by a post hoc Tukey's test was conducted to evaluate treatment effects. All analyses were conducted using SAS (version 9.4, SAS Institute, Cary, NC). Statistical significance was defined at $p < 0.05$.

3. Results

Regardless of salt content, a greater FI was observed in normal-fat diet groups (NSNF and HSNF) compared with high-fat diet groups (HSHF and NSHF) ($p < 0.0001$) (Figure 1). However, higher BW was observed in high-fat diet groups (NSHF and HSHF) ($p < 0.004$) (Figure 2). Body composition was also affected by the fat content of the diet, while salt content had no impact. Higher abdominal fat and lower fat-free mass (as % BW) (as an absolute amount and as a % BW) were observed in groups on high-fat diets (NSHF and HSHF) compared with those fed normal fat diets ($p < 0.05$) (Table 2). Short-term food intake (1, 2, and 12 hours after fasting) was lower in rats fed high-fat diets compared with those fed normal fat diets at 1 hour ($p < 0.04$) and 2 hours ($p < 0.01$) after fasting, regardless of the salt content of the diet. However, the cumulative food intake in 12 hours was influenced by both fat and salt ($p < 0.0001$): While higher food intake was observed in rats fed high-salt diets, high-fat diets reduced food intake (Table 3).

Fasting blood glucose (FBG) was affected by fat but not the salt content of the diet. FBG was higher in rats fed normal fat diets compared with those fed high-fat diets cumulatively throughout the study ($p < 0.03$) (Table 4). However, this effect disappeared over time and there was no effect of either fat or salt on FBG at week 12. The glucose response to glucose preload was influenced by salt

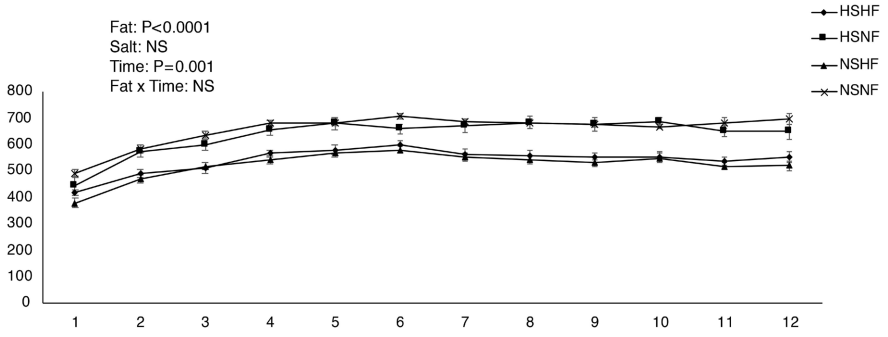

Figure 1. Effect of fat and salt on food intake (n = 12/group). Data are means ± SEM; Food intake was analyzed by MIXED procedure followed by Tukey's post hoc test with fat, salt, and time as main factors. Different letters at each time point are significantly different ($p < 0.05$); **HSHF:** High salt, high-fat diet; **HSNF:** High salt, normal fat diet; **NSHF:** Normal salt, high-fat diet; **NSNF:** Normal salt, normal fat diet. **NS:** not significant.

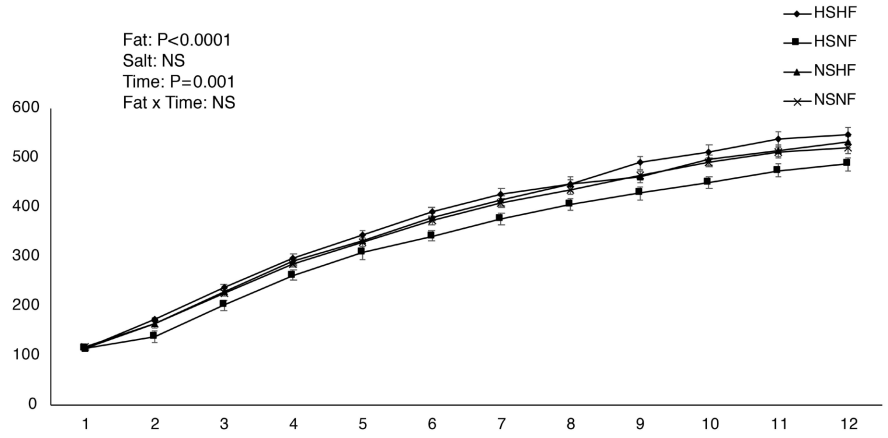

Figure 2. Effect of fat and salt on body weight (n = 12/group). Data are means ± SEM; Food intake was analyzed by MIXED procedure followed by Tukey's post hoc test with fat, salt, and time as main factors. Different letters at each time point are significantly different ($p < 0.05$); **HSHF:** High salt, high-fat diet; **HSNF:** High salt, normal fat diet; **NSHF:** Normal salt, high-fat diet; **NSNF:** Normal salt, normal fat diet. **NS:** not significant.

Table 2. Effect of fat and salt on fat and fat-free mass at week 12.

	HSHF	HSNF	NSHF	NSNF	p
Body weight	530 ± 10.12[a]	500.43 ± 15.54[b]	537.45 ± 11.53[a]	556.80 ± 10.12[a]	Fat: p = 0.05
Fat mass	38.12 ± 3.44[a]	25.12 ± 2.35[b]	39.33 ± 3.89[a]	33.17 ± 2.98[ab]	Salt: NS
Fat free mass	491.88 ± 8.31	475.31 ± 11.59	498.11 ± 9.13	523.63 ± 8.68	Time: p = 0.0001
Fat mass%	7.04 ± 0.95[a]	4.88 ± 0.92[b]	7.19 ± 0.88[a]	5.94 ± 0.48[ab]	
Fat free mass%	92.95 ± 6.74	95.08 ± 5.83	92.56 ± 7.78	93.76 ± 4.32	

Data are Means ± SE; n = 12/group. Different letters in each row are significantly different ($p < 0.05$); fat and fat-free mass were analyzed by MIXED Model followed by Tukey's post hoc test with fat and salt as the main factors. **HSHF:** High salt, high-fat diet; **HSNF:** High salt, normal fat diet; **NSHF:** Normal salt, high-fat diet; **NSNF:** Normal salt, normal fat diet. **NS:** not significant.

Table 3. Effect of fat and salt on short-term Food intake at week 12.

Diet	HSHF	HSNF	NSHF	NSNF	p
0 - 1 h	6.35 ± 0.77	7.85 ± 0.70	5.42 ± 0.45	6.52 ± 0.51	Fat: p = 0.04; Salt: NS
0 - 2 h	7.73 ± 0.68^{ab}	9.68 ± 1.00^{a}	6.46 ± 0.41^{b}	8.18 ± 0.64^{ab}	Fat: p = 0.01; Salt: NS
0 - 12 h	17.4 ± 0.88^{ab}	22.50 ± 1.01^{a}	13.99 ± 0.48^{b}	19.03 ± 0.43^{ab}	Fat: p = 0.0001; Salt: p = 0.0001

Data are means ± SEM; n = 12/group. Short-term FI was analyzed by MIXED procedure followed by Tukey's post hoc test with fat and salt as the main factors. Different letters in each row are significantly different ($p < 0.05$). **HSHF:** High salt, high-fat diet; **HSNF:** High salt, normal fat diet; **NSHF:** Normal salt, high-fat diet; **NSNF:** Normal salt, normal fat diet. **NS:** not significant.

Table 4. Effect of dietary fat and salt on FBG and BG in response to glucose at weeks 1 (baseline), 4, 8 and 12 (n = 12/group).

Diet		HSHF	HSNF	NSHF	NSNF	p
FBG	Wk 1		6.11 ± 0.36			Fat: p = 0.03
	Wk 4	5.08 ± 0.31^{b}	5.92 ± 0.47^{b}	5.54 ± 0.24^{b}	7.43 ± 0.59^{a}	Salt: NS
	Wk 8	5.97 ± 0.22b	6.38 ± 0.73^{b}	6.74 ± 0.58^{ab}	7.10 ± 0.57^{a}	Time: p = 0.001
	Wk 12	5.81 ± 0.19	6.70 ± 0.56	6.23 ± 0.10	6.24 ± 0.22	
OGTT	Wk 1		656.18 ± 29.36			Fat: NS
	Wk 4	539.79 ± 39.54	556.81 ± 23.64	637.22 ± 39.71	575.07 ± 27.96	Salt: p = 0.04
	Wk 8	517.36 ± 26.05	479.03 ± 39.71	629.24 ± 57.82	552.02 ± 29.94	Time: p = 0.001
	Wk 12	663.26 ± 34.58^{ab}	568.54 ± 27.96^{b}	723.54 ± 29.94^{a}	634.38 ± 69.23^{ab}	Fat x Time: p = 0.01

Data are Means ± SE; n = 12/group. **Wk 1:** Baseline. FBG and OGTT were analyzed by MIXED Model followed by Tukey's post hoc test with fat and salt as the main factors. FBG: Fasting Blood Glucose. OGTT: Oral Glucose Tolerance Test. Different letters in each row are significantly different ($p < 0.05$). **HSHF:** High salt, high-fat diet; **HSNF:** High salt, normal fat diet; **NSHF:** Normal salt, high-fat diet; **NSNF:** Normal salt, normal fat diet. **NS:** not significant.

but not the fat content of the diet cumulatively throughout the study. This response was found to be higher in rats fed normal salt diets compared with those fed high salt diets ($p < 0.04$) (Table 4). The effect of fat on glucose response to glucose preload was increased over time, it was higher in rats fed high-fat diets compared with those fed normal fat diets at week 12 ($p < 0.05$) (Table 4).

Systolic and diastolic blood pressure were not influenced by either the fat or salt content of the diet but were influenced by their interactions. Systolic and diastolic blood pressure were lower in rats fed either HSHF or NSNF diet compared with those fed HSNF, or NSHF diet ($p < 0.005$ and $p < 0.03$ respectively) (Table 5). However, pulse was influenced by the salt content of the diet only. It was higher in rats fed high salt diets compared with those fed normal salt diets ($p < 0.001$) (Table 5).

4. Discussion

Consistent with previous studies, the results of this study support the notion that fat and salt contents of the diet are factors determining food intake, body weight, and body composition [5] [6] [7] [12]-[18]. The results of this study showed that fat but not sodium influences food intake in the long term. High-fat diets

Table 5. Effect of dietary fat and salt on SBP, DBP, and pulse at weeks 1 (baseline), 4, 8 and 12.

Diet		HSHF	HSNF	NSHF	NSNF	P
SBP	Wk 1	107.41 ± 5.39			Fat: NS	
	Wk 4	111.23 ± 14.5^{b}	124.75 ± 12.18^{ab}	139.85 ± 9.96^{a}	118.88 ± 1349^{ab}	Salt: NS
	Wk 8	125.2 ± 5.75^{ab}	154.13 ± 12.64^{a}	137.85 ± 11.45^{ab}	112.35 ± 4.97^{b}	Time: p = 0.001
	Wk 12	129.2 ± 3.87^{b}	145.48 ± 5.13^{a}	143.07 ± 2.84^{a}	139.08 ± 2.03^{ab}	Fat x Salt: p < 0.005
DBP	Wk 1	60.48 ± 7.93			Fat: NS	
	Wk 4	69.53 ± 2.80	64.33 ± 6.63	74.23 ± 8.20	60.67 ± 12.28	Salt: NS
	Wk 8	57.07 ± 8.84	71.25 ± 14.06	77.11 ± 14.92	63.28 ± 5.15	Time: p < 0.02
	Wk 12	73.73 ± 3.11	96.17 ± 15.57	77.50 ± 6.88	79.06 ± 9.72	Fat x Salt: p < 0.03
Pulse	Wk 1	492.76 ± 22.00			Fat: NS	
	Wk 4	480.64 ± 18.07	424.29 ± 16.63	451.84 ± 18.52	426.08 ± 23.09	Salt: p < 0.05
	Wk 8	425.83 ± 13.31	433.70 ± 29.08	359.02 ± 45.20	380.15 ± 20.39	Time: p = 0.001
	Wk 12	434.39 ± 11.62	436.34 ± 13.59	425.27 ± 14.66	422.38 ± 18.06	Fat x Time: NS

Data are Means ± SE; n = 12/group. **Wk 1:** Baseline. FBG and OGTT were analyzed by MIXED Model followed by Tukey's post hoc test with fat and salt as the main factors. FBG: Fasting Blood Glucose. OGTT: Oral Glucose Tolerance Test. Different letters in each row are significantly different (p < 0.05). **HSHF:** High salt, high-fat diet; **HSNF:** High salt, normal fat diet; **NSHF:** Normal salt, high-fat diet; **NSNF:** Normal salt, normal fat diet. **NS:** not significant.

resulted in lower food intake. The results from previous studies examining the effect of dietary fat and sodium on food intake are mixed. A high-fat, normal sodium diet resulted in the least amount of food eaten but with the greatest overall energy intake in humans [16]. In another study, rats fed a high-sodium and high-fat diet also ate less compared to those eating a normal-fat diet. It can be explained by the fact that fat taste sensitivity has a suppressing effect on dietary fat intake [16]. In contrast, in another study, high-fat foods led to passive overconsumption in young male subjects [14]. It is suggested that foods with high dietary fat have a weak satiation effect in the short term, which leads to overconsumption (high-fat hyperphagia) [17]. In a clinical study, high-fat foods resulted in passive overconsumption [14]. The high energy density of the meal and the high palatability of high-fat foods are influencing factors as well. In another study, salt increased food and energy intake while fat had no effect on food intake in healthy adults [16]. It has been suggested that salt increases passive energy intake in adults and it may override fat-mediated satiation in subjects who are sensitive to the taste of fat. It has also been suggested that the satiating effects of a diet with high fat: carbohydrate ratio is weaker compared with a diet with a lower ratio [5]. These inconsistencies can be explained by differences in their experimental designs, particularly the period of the food intake that has been studied: Mechanisms regulating short- and long-term food intakes are fundamentally different. While the short-term food intake regulatory system is controlling meal size and meal frequency, food intake in long term is influenced by more systemic signals including body weight and the level of fat storage in the

body [19] [20]. For example, in the short term, taste may play a more significant role while total energy intake and energy density are major players in long-term food intake regulation [19] [20] which is consistent with our results: While short-term food intake in 12 hours was influenced by both fat and salt, long-term food intake was influenced by the fat content of the diet only. Although a high-fat diet may increase the meal size, it may reduce the meal frequency: during the inter-meal interval, a high-fat diet resulted in more sustained satiety compared with a high CHO preload, leading to a lower meal frequency [15].

The results of this study support the role of fat and salt in the regulation of glucose metabolism. While FBG was not affected by either fat or salt content of the diet at the end of the study, the glucose response to glucose preload was higher in rats fed a high-fat, normal salt diet compared with other groups at week 12. The effect of dietary fat and salt on glucose metabolism has been studied separately in previous studies [24] [25] [26]. In rats, a high-fat diet increased body weight but had no significant effect on oral glucose tolerance [27]. Although the negative effects of a high-fat diet on glucose metabolism are reported, they are mostly secondary to the obesity induced by a high-fat diet [28]. However, some studies suggested this effect might be independent of obesity [7]. Mice on high fat, high salt diet for ten weeks, showed elevated fasting blood glucose and impaired glucose control during glucose tolerance tests [7]. In another study, high salt, high-fat diet deteriorated glucose intolerance, with impairment in insulin secretion. The authors suggested that it can be due to the attenuation of expansion of β-cell mass in the pancreas [29]. Similarly, a high salt diet enhanced insulin signaling and induces insulin resistance in Dahl salt-sensitive rats [30] [31]. In contrast, in hypertensive, overweight subjects, fasting plasma glucose, insulin, and homeostasis model assessment were higher and blood pressure was lower in subjects on a low salt diet as compared with those who were on a high salt diet. The authors concluded that the increase in insulin resistance on a low-salt diet is not affected by salt sensitivity to blood pressure [32]. These results are consistent with our findings in this study. The interactive effects of sodium and glucose can be partially through Na-glucose cotransporter (SGLT2), their common mechanism of transportation. SGLT2 inhibitors have been utilized as drugs to prevent heart failure in diabetics [33]. In one study, Tofogliflozin, an SGLT2 inhibitor, improved cardiac hypertrophy, and fibrosis and reduced ketone usage in myocardial tissue in Dahl Salt-Sensitive and Salt-Resistant Rats Fed a High-Fat Diet [33]. In another study, SGLT2 blockade slowed the progression of diabetic kidney disease by reducing physical strain on the glomerulus in rats [34].

Blood pressure can be influenced by various factors including but not limited to age, family history, high sodium or low potassium diet, obesity, and alcohol [35]. The underlying mechanisms by which sodium affects the blood pressure can be through water retention, vascular remodeling, and endothelial dysfunction [36]. We found that the interactions of dietary fat and salt proved significant impact on

systolic and diastolic blood pressure; rats fed either HSHF or NSNF diet had lower systolic and diastolic blood pressure compared with rats fed HSNF or NSHF diet. The results from previous studies showed the negative effects of salt [37] and long-term high fat intake [38] on the risk of cardiovascular diseases, perhaps due to the obesogenic effect of a high-fat diet on blood pressure. However, this effect might be conditional on the individuals' salt sensitivity [3]. Moreover, the duration of this study might be too short to induce hypertension by the diet's higher fat and salt contents as evidenced by the significant interaction between the effect of time and fat and salt on blood pressure. However, pulse was higher in rats fed high salt diets compared with those fed normal salt diets. Further studies are needed to investigate the role of sodium in the regulation of the pulse. Although there is no direct implementation of these findings in human, they may be considered as a basis for future clinical trials since rodents and human are sharing similar metabolic and physiologic mechanisms.

The relatively short duration is the major limitation of this study. Moreover, a dose- and time-dependent study will be helpful to determine whether the effect of fat and salt on measured parameters is dose-dependent. However, the current design is suitable for the proposed hypothesis, which examined the main and interactive effects of dietary fat and salt on food intake, body weight, body composition, and characteristics of metabolic syndrome in male Wistar rats.

5. Conclusion

In conclusion, the results of this study support the notion that dietary fat and salt and their interaction are determining factors in the regulation of food intake, body weight, body composition, and blood pressure in male Wistar rats. Dietary fat showed a detrimental effect on body composition and body weight and glucose metabolism regardless of the salt content of the diet while higher salt content deteriorated the pulse rate.

Financial Support

This study is supported by the US foundation, University of North Florida.

Authorship

Kea Schwarz formulated the research questions, designed the study, carried out the study, analyzed the data, interpreted the findings and wrote the article; Leila Ninya formulated the research questions, designed the study, carried out the study, analyzed the data, interpreted the findings and wrote the article; Tatyana Kimble formulated the research questions, designed the study, carried out the study, analyzed the data, interpreted the findings and wrote the article; Alireza Jahan-Mihan supervised students throughout the study and revised and finalized the manuscript.

Conflicts of Interest

The authors declared no conflicts of interest.

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List of Abbreviations

BW: Body weight
BC: Body composition
Cal: Calorie
DBP: Diastolic blood pressure
FBG: Fasting blood glucose
FI: Food intake
g: Gram
HSHF: High salt, high-fat diet
HSNF: High salt, normal fat diet
IACUC: Institutional Animal Care and Use Committee
NS: Not significant
NSHF: Normal salt, high-fat diet
NSNF: Normal salt, normal fat diet
OGTT: Oral glucose tolerance test
SBP: Systolic blood pressure
T: Time