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A SIX-WEEK DIET HIGH IN FAT, FRUCTOSE AND SALT AND ITS INFLUENCE ON LIPID AND MINERAL STATUS, IN RATS

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ABSTRACT

Introduction. Fat, fructose, and salt consumption has increased in industrialized countries, but there are few studies that have investigated the effect of this eating pattern on metabolic and physiological states. The purpose of this study was thus to assess lipid and carbohydrate metabolism and to estimate iron, zinc, copper, calcium, and magnesium status in rats fed a diet high in fat, fructose, and salt, compared to the control diet.
Material and methods. Wistar rats were assigned to groups fed either a standard diet or a diet high in fat, fructose, and salt (M). After 6 weeks, the animals were weighed and killed. Liver, kidney, heart, pancreas, hair, and blood samples were collected. The total cholesterol, triglyceride, fasting glucose, and insulin levels in serum were measured. The iron, zinc, copper, calcium, and magnesium concentrations in tissues and serum were determined.
Results. It was found that the M diet led to a significant increase in cholesterol and triglyceride levels in the serum of rats. Among rats fed the M diet, a markedly higher serum level of iron and a significantly lower serum level of zinc were observed. A significantly lower iron level in the pancreas, zinc level in the kidneys and pancreas, and copper level in the kidneys it was found in rats with the M diet. The modified diet resulted in markedly lower concentrations of magnesium in the hearts. In the hair of rats on the M diet, higher levels of iron and zinc were observed. The relative masses of the kidneys were markedly higher in rats with the M diet, as compared with the C diet.
Conclusions. Diets high in fat, fructose, and salt disturb lipid status and kidney mass. This modified diet impairs mineral balance in the body.

Key words: fat, fructose, salt, lipids, zinc, copper, iron, magnesium, calcium

INTRODUCTION

The consumption of fructose, fat, and salt has increased in recent years throughout the world [Madero et al. 2011]. Experimental and clinical research have shown that the increased levels of these components in processed food is related to high body mass, blood pressure, and insulin resistance. The intake of beverages containing fructose has resulted in increases in the risk of obesity, diabetes, hypertension, gout, and non-alcoholic fatty liver disease [Bray 2012, Johnson et al. 2007]. Experimental data suggest that continuous fructose consumption results in endothelial dysfunction, salt retention, insulin resistance, and dyslipidemia [Bremer et al. 2011, Madero et al. 2011].

However, it has also been shown that high-fat diets have more adverse responses than high-fructose diets, in that they induce obesity and low insulin sensitivity in studies with rats [Zaman et al. 2011]. It has also been observed that high-fat diets lead to increased

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glucose, insulin, and triglyceride levels in rats and promote insulin resistance and inflammation [Hao et al. 2012, Jacob et al. 2012]. Moreover, it has been observed that high-fat and high-fructose diets impair macro and trace mineral status [Tsuchiya et al. 2013, Song et al. 2012, Hwang et al. 2011, Holbrook et al. 1989].

Recent studies suggest that diets high in salt and sodium are associated with enhanced inflammation, lipid disorders, target organ damage, and also with declines in renal function in hypertension [Ohta et al. 2012, Yilmaz et al. 2012]. In animal models and humans, it has been observed that excess salt intake impairs endothelial function, increases serum uric acid, and decreases nitric oxide production – this last being a homeostatic mechanism that regulates blood pressure in the body [Forman et al. 2012, Reuter et al. 2009, Dishy et al. 2003].

The results of several studies indicate that each of these three dietary factors (fructose, fat, and salt) adversely affects the metabolic state of the body. However, there are a few studies that investigate the effect on physiological and metabolic state of diets containing all three components. The aim of the present study was thus to assess the effects of diet with high contents of fat, fructose, and salt on selected blood biochemical parameters, tissue weights, and selected parameters of mineral status in rats.

MATERIAL AND METHODS

Animals

All animal procedures were performed according to approved protocols and in accordance with the recommendations for the proper care and use of laboratory animals. The experiment was performed with the agreement of the local bioethics committee (approval no. 44/2007). Six-week-old male Wistar rats, with mean initial body weight of 187 ±13.5 g, were obtained from the Department of Toxicology, University of Medical Sciences, Poznań. The animals were housed individually in stainless steel cages coated with metal-free enamel, and kept under cycles of 12 hours light to 12 hours dark. Room temperature was maintained at 21 ±1°C with 55-65% humidity.

Experimental design

The experiment was performed using 20 male Wistar rats. The animals were randomly assigned to two groups equal in size: control (C) and modified (M). The control group was fed a semisynthetic AIN-93M diet (the C diet), while the modified group was fed a semisynthetic diet high in fat, fructose, and sodium (the M diet). The full composition of the diets is presented in Tables 1 and 2.

Table 1. Ingredient and nutrient composition of the diets, $g \cdot kg^{-1}$ diet

Ingredient	Diet	
	control (C)	modified (M)
Casein	140	140
Wheat starch	625	–
Sucrose	100	–
Potato starch	50	–
Vitamin mixture	10	10
Mineral mixture	35	35
Sunflower oil	40	40
Lard	–	150
Sodium chloride	–	35
Fructose	–	590
Total energy, kcal/100 g diet	424	510
Total protein, % of energy	13	13
Total fat, % of energy	4	18

Table 2. Content of minerals in the diets, $mg \cdot kg^{-1}$

Mineral	Diet	
	control (C)	modified (M)
Iron	35.1 ±0.5	37.1 ±0.6
Zinc	21.0 ±0.4	21.7 ±0.4
Copper	5.99 ±0.2	6.07 ±0.2
Calcium	11 880 ±32	11 699 ±43
Magnesium	453.5 ±4.6	461.4 ±5.2

Values are presented as mean ±SD.

All rats were provided *ad libitum* diet and distilled water for 42 days. Dietary intake was recorded daily (Table 3). Body weight was recorded every week before food distribution.

Table 3. Daily diet intake in rats

Diet, g·day^{-1}	
control (C)	modified (M)
21.2 ±1.2	18.9 ±1.6

Values are presented as mean ±SD.

Tissue and serum collection

At the end of the experimental period, the animals were weighed and anesthetized with a sodium thiopental injection (40 mg/kg body weight), and killed by cardiac puncture. The liver, kidneys, heart, and pancreas were dissected, weighed, and stored frozen (–70°C) until the analysis for mineral contents. The hair was taken from between the shoulder blades. Blood samples were collected by cardiac puncture in serum-separated tubes (without using an anticoagulant). The coagulated blood was left to clot at room temperature for 30 min, and then centrifuged for 15 min at 2,000 r.p.m. at 4°C. The supernatant fluid was then separated. Serum samples were stored at –70°C until analyzed.

Biochemical measurements

Total cholesterol (TC), triglyceride (TG), and fasting glucose levels in serum were measured using commercial kits (Randox Laboratory Ltd., UK) in the laboratory in Poznań, Poland. The concentration of cholesterol and triglycerides in serum was assayed using the enzymatic method. The concentration of glucose in the blood serum was estimated using the glucose oxidase method. Serum insulin was determined by radioimmunoassay method: a rat insulin RIA kit was used (Insulin RIA Kit; Linco Research, Inc., USA).

Determination of minerals

The mineral content in the serum (iron, zinc, and copper) and tissues (iron, zinc, copper, calcium, and magnesium) was determined after digestion in 65% (w/w) spectra pure HNO_3 (Merck) in the Microwave Digestion System (MARS 5, CEM Corp., USA). Thereafter, the concentrations of minerals in the mineral solutions were measured using the flame atomic absorption spectrometry method (AAS-3, Carl Zeiss, Jena, Germany). The accuracy of the method was verified using certified reference materials (pig kidney BCR no. 186, Brussels) and was 92% for iron, 95% for zinc, 102% for copper, 98% for magnesium, and 92% for calcium.

Statistical analysis

Detailed statistical analysis was performed using Statistica for Windows 10.0 (StatSoft, Inc., Poland). The normality of the variables' distribution was verified using the Shapiro-Wilk test of normality. The comparisons between groups were carried out using the Mann-Whitney U test. Significance was set at the $P < 0.05$ level. The results were expressed as arithmetic means and standard errors.

RESULTS

The average intake of the diet in the control group (C) and the modified group (M) was comparable (Table 3). The modified diet (M) resulted in a significant increase in cholesterol and triglyceride serum levels in rats, as compared to the control group (C) (Table 4). A markedly higher level of iron and a significantly lower level of zinc in the serum of rats fed the modified

Table 4. Biochemical parameters in serum of rats

	Diet		P
	control (C) (n = 10)	modified (M) (n = 10)	
Chol-T, mmol·l^{-1}	1.78 ±0.2^a	2.08 ±0.1^b	<0.05
TG, mmol·l^{-1}	0.32 ±0.05^a	0.41 ±0.06^b	<0.05
Glucose, mmol·l^{-1}	9.2 ±1.0	9.8 ±0.7	NS
Insulin, pmol·l^{-1}	33.0 ±5.9	35.2 ±5.1	NS
Serum Fe, µmol·l^{-1}	26.5 ±1.1^a	31.0 ±1.1^b	<0.05
Serum Zn, µmol·l^{-1}	11.8 ±0.5b	5.7 ±0.3^a	<0.01
Serum Cu, µmol·l^{-1}	10.0 ±0.6	11.1 ±0.9	NS

Values are presented as mean ±SD, P – significance level.

Table 5. Content of minerals in tissues, ng-g'[1] d.w.

Diet	control (C) (n= 10)	modified (M) (n = 10)	P
Liver			
Fe	303.6 ±35.4	308.3 ±19.8	NS
Zn	100.2 ±10.0	92.3 ±6.6	NS
Cu	14.9 ±1.7	14.6 ±1.9	NS
Ca	112.8 ±20.9	120.9 ±19.5	NS
Mg	616.0 ±33.6	588.4 ±15.6	NS
Kidneys			
Fe	169.6 ±10.7	156.3 ±10.8	NS
Zn	107.7 ±13.8[b]	92.1 ±6.4[a]	<0.05
Cu	33.6 ±2.6[b]	24.3 ±4.6[a]	<0.05
Ca	163.9 ±25.2	166.8 ±25.8	NS
Mg	581.2 ±36.1	609.1 ±32.4	NS
Heart			
Fe	240.7 ±7.4	239.8 ±14.3	NS
Zn	67.4 ±2.3	75.2 ±2.6	NS
Cu	25.1 ±1.3	20.1 ±1.1	NS
Ca	155.1 ±14.6	156.4 ±15.5	NS
Mg	749.3 ±23.5[b]	528.9 ±32.4“	<0.05
Pancreas			
Fe	121.4 ±10.0[b]	86.4 ±10.6°	<0.05
Zn	130.2 ±7.2[b]	86.0 ±11.7[a]	<0.01
Cu	6.1 ±0.5	5.1 ±0.4	NS
Ca	297.4 ±10.7	259.3 ±16.4	NS
Mg	1 298.3 ±72.6	1 174.3 ±43.0	NS
Hair			
Fe	13.4 ±3.0[a]	18.7 ±4.3[b]	<0.05
Zn	103.3 ±12.7	108.8 ±6.5	NS
Cu	12.8 ±0.5	13.1 ±1.8	NS
Ca	238.2 ±31.7	197.0 ±14.3	NS
Mg	66.6 ±5.8	66.0 ±4.6	NS

Vałues are presented as mean ±SD, d.w. - dry weight, P - significance level.

Table 6. Percentage of relative mass of tissues

Diet	control (C) (n = 10)	modified (M) (n = 10)	P
Liver, % b.m.	3.13 ±0.23	3.57 ±0.43	NS
Kidneys, % b.m.	0.66 ±0.02[a]	0.82 ±0.04[b]	<0.05
Heart, % b.m.	0.30 ±0.03	0.32 ±0.03	NS
Pancreas, % b.m.	0.31 ±0.02	0.32 ±0.03	NS

Values are presented as mean ±SD, b.m. - body mass, P - significance level.

diet were also observed (Table 5). The M diet led to decreases in the iron and zinc levels in the pancreas and increased the content of iron in the hair. A significantly lower level of zinc and copper in the kidneys, and of magnesium in the hearts, of rats on the M diet was also observed (Table 5). The diet high in fat, fructose, and salt significantly increased the relative kidneys mass in the rats (Table 6). Other parameters were compara- ble between control and modified groups (Tables 4-6).

DISCUSSION

In several studies, an association has been found between diet composition and lipid and carbohydrate status. Many experimental and clinical studies have investigated the effects of diets containing high lev- els of fat or fructose, or a combination of these two components. In this study, a model diet high in fat, fructose, and sodium chloride was used.

It was found in the present study that the modified diets had significant influences on lipid disorders and on iron, zinc, copper, and magnesium status. Slightly increased insulin and glucose concentrations in serum were also observed.

In a recent study, Briand et al. [2012] observed that diets high in fat and fructose induced liver steatosis, with high cholesterol and triglyceride serum levels, and enhanced intestinal cholesterol absorption. The high-fat diet may result not only in an increase in the absorption of cholesterol, but also in an increase in cholesterol synthesis in the liver. It is known that liver fat content is associated with cholesterol synthesis.

High liver-fat content increases the activity of sterol regulatory element-binding protein 2 (SREBP2), which is the most important regulator of cellular cholesterol synthesis. Silbernagel et al. [2012] have observed that this transcription factor was highly expressed in the liver. Dietary fructose may also increase hepatic lipogenesis [Samuel 2011]. However, fructose probably affects cholesterol synthesis less than fat does, because fructose does not provoke the endogenous secretion of insulin, which is considered to be an important regulator of cholesterol synthesis [Silbernagel et al. 2012, Cozma et al. 2012]. In clinical studies, it has been found that a high-fructose diet elevates plasma TG level in healthy people [Silbernagel et al. 2011]. It has also been observed that long-term high intake of salt leads to increases in serum cholesterol concentration in humans and in animal models, but the mechanism responsible for this effect has not yet been elucidated [Pamidimukkala and Jandhyala 2004, Lima et al. 2009].

On the basis of the above data, it appears that all three components modified in the diets (fat, fructose, and sodium chloride) used in this experiment had an impact on the disorder of the lipid metabolism in the rats on the M diet.

In this study, high levels of fructose, fat, and sodium in the diet lead to a slight increase in glucose and insulin serum levels. Experimental studies have shown that the sustained consumption of fructose promotes the development of insulin resistance and glucose intolerance [Stanhope and Havel 2009]. Coate et al. [2011] found that diets high in fat and fructose diminished hepatic glucose uptake and hepatic glycogen synthesis in dogs with partial pancreatectomies. Fonseka-Alaniz et al. [2008] found that long-term (9 week) consumption of a high-sodium diet influenced glucose and insulin serum concentration in rats. These authors concluded that this high sodium intake may interfere with glucose and insulin metabolism, and that chronic salt overload enhances adipocyte insulin sensitivity to glucose uptake and increases adipose visceral masses [Fonseka-Alaniz et al. 2008]. The lack of impact of the modified diets on insulin and glucose levels in serum observed in the present study may be caused by an insufficiently long experimental period of six weeks.

In this study, the modified diet led to increased iron levels in serum, but to decreased levels of copper in the kidneys and decreased concentrations of zinc in serum, kidneys, and the pancreas of the rats. The mineral disorders observed in the rats fed the modified diet may be caused by high levels of fat and fructose in the diet, and also by interactions between minerals. It has been found that a high-fructose diet impairs copper status and leads to iron overload [Minamiyama et al. 2010]. It has been shown that increased fat in the diet increases iron absorption [Chausmer 1998], but there are also contradictory reports [Afkhami-Ardekani et al. 2008]. It has been found that high-fructose diets induce copper deficiency, probably through impaired duodenum Ctr-1 expression, which leads to lower copper absorption [Minamiyama et al. 2010, Sondergaard et al. 2006]. The lower zinc concentrations in the tissues of rats on the modified diet may be caused by interactions between the iron and the zinc [Bonomo et al. 2012]. The results of this study confirm the data obtained by Wapnir and Devas [1995], who also found that high-fructose, high-fat diets decrease copper and zinc levels in the kidneys of rats.

The low status of zinc in the rats on the M diet, and especially the lower concentrations of zinc in the pancreas, may result in the decreased synthesis of insulin in this tissue, because zinc is essential for the synthesis and action of insulin [Chimienti et al. 2005, Chausmer 1998]. In this study, such an effect was not observed, presumably due to the insufficiently long duration of the study.

In this study, it was found that the modified diet lead to a decrease in magnesium in the heart of the rats. Low levels of magnesium in heart tissue are associated with heart arrhythmia and high cardiovascular risk [Del Gobbo et al. 2012]. In other studies, it has been shown that fructose in the diets of magnesium-deficient rats was associated with higher plasma triglyceride concentrations and higher tissue susceptibility to peroxidation [Busserolles et al. 2003]. It may therefore be assumed that diets high in fat, fructose, and sodium may increase the risk of cardiovascular disease and disturb lipid metabolism by affecting the magnesium balance in the body.

In the present study, it was observed that the modified diet increased the relative kidney mass in rats. It is possible that this renal enlargement could be the body's mechanism for adapting to the intake of large amounts of salt. Previous studies have shown that a high-salt

diet increases renal weight, and it has been suggested that this may be due to increases in the number of cells, enlargement of existing cells, and/or changes in circulating renal growth factors [Manitius et al. 1995, McCormick et al. 1989]. The other components modified in the diets – fat and fructose – may also have had an impact on kidney size [Manitius et al. 1995].

This study has its limitations. First of all, ferritin and other inflammation markers were not assessed. Insulin concentration was measured, but the concentration of C peptide, a more stable analyte, was not. The lack of measurement of these parameters permits only speculation on the effect of the modified diet on carbohydrate metabolism. Magnesium and calcium were assessed only in tissues, as insufficient serum was obtained from the rats.

CONCLUSION

A finding of this study is that diets high in fat, fructose, and salt decrease zinc status and disturb iron, copper, and magnesium status in rats. It was additionally confirmed that a diet high in fat, fructose, and salt disturbs lipid metabolism and increases kidney mass in rats. The results obtained in this study suggest that the impact of the Western diet – high in fat, sugar, and sodium – on the development of diseases of civilization may be associated with disorders of mineral metabolism, especially zinc, in the body.

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WPŁYW SZEŚCIOTYGODNIOWEJ DIETY Z DUŻĄ ZAWARTOŚCIĄ TŁUSZCZU, FRUKTOZY I SOLI NA GOSPODARKĘ LIPIDOWĄ ORAZ MINERALNĄ U SZCZURÓW

STRESZCZENIE

Wstęp. Spożycie tłuszczu, fruktozy i soli wzrasta w krajach uprzemysłowionych. Jak dotąd jednak, w niewielu badaniach ocenia się wpływ tych nawyków żywieniowych na stan metaboliczny i fizjologiczny organizmu. Dlatego celem pracy była ocena metabolizmu lipidów i węglowodanów oraz określenie gospodarki żelaza, cynku, miedzi, wapnia i magnezu u szczurów karmionych dietą z dużą zawartością fruktozy, tłuszczu i soli w porównaniu ze szczurami na diecie standardowej.

Materiał i metody. Szczury rasy Wistar podzielono na dwie grupy: pierwszą żywiono dietą standardową (C), drugą – dietą z dużą zawartością tłuszczu, fruktozy i soli (M). Po sześciu tygodniach doświadczenia zwierzęta były ważone i usypiane. Z ciała zwierząt pobrano wątrobę, nerki, serce, trzustkę, sierść oraz próbki krwi. W surowicy krwi oznaczono stężenie cholesterolu, triglicerydów, glukozy i insuliny. Zawartość żelaza, cynku, miedzi, wapnia i magnezu oznaczono w surowicy krwi oraz w tkankach.

Wyniki. Stwierdzono, że dieta M spowodowała znaczący wzrost stężenia cholesterolu i triglicerydów w surowicy krwi szczurów. Ponadto u szczurów na diecie M obserwowano istotny wzrost stężenia żelaza i znaczące zmniejszenie stężenia cynku w surowicy. Wykazano znacząco mniejsze stężenie żelaza w trzustce, cynku – w nerkach i trzustce oraz miedzi – w nerkach szczurów na diecie M. Dieta modyfikowana wpłynęła na znaczące obniżenie magnezu w sercu szczurów. W sierści szczurów z dietą M zaobserwowano znacząco większe stężenie żelaza. Względna masa nerek szczurów na diecie M była znacząco większa aniżeli masa nerek szczurów na diecie C.

Wnioski. Dieta z dużą zawartością tłuszczu, fruktozy i soli zaburza gospodarkę lipidową oraz względną masę nerek. Dieta modyfikowana prowadzi do zakłócenia równowagi składników mineralnych w organizmie.

Słowa kluczowe: tłuszcz, fruktoza, sól, lipidy, cynk, miedź, żelazo, magnez, wapń, nerki

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