ORIGINAL ARTICLE

Effect of nutrition and atherogenic index on the occurrence and intensity of insulin resistance

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KEY WORDS

atherogenic index, diet, insulin resistance

ABSTRACT

INTRODUCTION Diet is one of the factors that can stimulate genetic predisposition and, in consequence, lead to insulin resistance. An adequate supply of nutrients and energy-rich diet as well as increased physical activity are the most effective methods to prevent metabolic disorders.

OBJECTIVES The objective of this study was to determine whether there are any associations between nutrition and the occurrence of insulin resistance.

PATIENTS AND METHODS The study included 143 individuals. Fasting glucose and insulin levels were measured and the HOMA-IR index was calculated for each patient. Nondiabetic patients were divided into the study and control groups. We conducted anthropometric measurements (body mass, height, and waist circumference), biochemical analysis (fasting glucose and insulin), and dietary interview.

RESULTS We observed a negative correlation between the percentage of sucrose in the diet and the HOMA-IR value, and a positive correlation between the percentage of protein intake and the HOMA-IR value. Moreover, there was a significantly higher intake of lactose in men without insulin resistance compared with those with insulin resistance.

CONCLUSIONS The results encourage to conduct further, more detailed research involving a larger group of patients to better understand associations between dietary content and insulin resistance.

INTRODUCTION Insulin resistance is a disturbance of glucose homeostasis. It is characterized by reduced sensitivity of target tissues to insulin action despite normal or elevated levels of insulin in blood serum.[1] An adequate glucose transport into the cells is dependent on insulin. This hormone acts on the receptors in the cell membranes of the insulin-dependent cells, which include hepatocytes, adipocytes, myocytes and others.[2] It should be noted that many epidemiological studies have clearly underlined the importance of genetic factors in the pathogenesis of insulin resistance. Attention has also been drawn to an important relationship between environmental factors and the onset of insulin resistance in genetically predisposed individuals.[3]

Among the factors affecting the response of tissues to insulin are age, sex, physical activity, iatrogenic factors, body weight, and, inseparably connected with the body mass index (BMI), body fat, which secretes adipocytokines, as well as diet and drugs.[4]

Diet is one of the factors that can influence genetic conditions and, in consequence, lead to insulin resistance. An adequate supply of nutrients, energy-rich diet, and increased physical activity are the most effective methods to prevent metabolic disorders. In the primary and secondary prevention, the Mediterranean diet is widely used. The composition of this diet helps prevent obesity and its complications.[5] Excessive energy consumption results in energy storage in the adipose tissue, especially in the visceral area. A systematic reduction of calorie intake leads to weight loss, fat loss, and increased insulin sensitivity. The most important thing is the quality of food, that is, the content and proportions of various nutrients.[6]

The objective of this study was to determine whether there is a relationship between nutrition and the development of, or increase in, insulin resistance.

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TABLE 1 Anthropometric characteristics of the study and control groups

Parameter		Study group (n = 76)		Control group (n = 67)	
		women (n = 43)	men (n = 33)	women (n = 43)	men (n = 24)
age, y	mean ±SD	45.7 ±13.6	40.9 ±12.7	43.2 ±15.7	45.8 ±16.7
	range	18–70	19–64	21–72	20–79
BMI, kg/m^2	mean ±SD	31.8[a] ±8.1	30.8[b] ±5.2	25.6[a] ±5.8	27.3[b] ±2.7
	range	19.9–52.7	23.1–46.4	15.6 –44.6	22.5–34.8
waist circumference, cm	mean ±SD	99.9[a] ±17.7	104.0 ±15.0	89.0[b] ±16.0	98.5 ±8.2
	range	71–156	83–152	67 – 136	84–120
WHR	mean ±SD	0.91 ±0.1	0.98 ±0.08	0.89 ±0.07	0.98 ±0.04
	range	0.72–1.18	0.85–1.16	0.74–1.09	0.9–1.05

a $P < 0.001$ study vs. control group; **b** $P < 0.05$ study vs. control group

Abbreviations: BMI – body mass index, SD – standard deviation, WHR – waist-to-hip ratio

PATIENTS AND METHODS The study involved 143 individuals (86 women and 57 men; age, 19–79 years; mean age, 43 ±14.6 years). The characteristics of the study group are presented in TABLE 1. We measured fasting glucose levels using the hexokinase method and fasting insulin levels using the Insulin IRMA Kit (Immunotech, Czech Republic). We then calculated the Homeostasis Model Assessment–Insulin Resistance (HOMA-IR) for each individual to determine the severity of insulin resistance. The following formula was used[7]: HOMA-IR = (glucose × insulin) / 405, where glucose is fasting glucose (mg/dl) and insulin is fasting insulin (mU/ml).

Patients were divided into the study and control groups. The study group (n = 76) included 43 women and 33 men, while the control group (n = 67) included 43 women and 24 men.

The criterion for inclusion in the study group was a HOMA-IR value of 2 or higher (insulin resistance), and, in the control group, a value of less than 2 (no insulin resistance).[6,8] Patients diagnosed with type 1 or 2 diabetes and hormonal disorders were excluded from the study. We conducted anthropometric measurements (body mass, height, and waist circumference), biochemical analyzes (fasting glucose and insulin levels), and a 24-hour dietary recall (2 working days and 1 day holiday). All patients were characterized with a moderate level of physical activity as assessed by the International Physical Activity Questionnaire.

All patients were measured and weighed using a body composition analyzer, InBody 220 (BioSpace, Korea). The BMI was calculated using the following formula[9]: BMI = weight (kg) / height^2 (m^2). Waist and hip circumferences were measured with a centimeter tape. The waist circumference was measured accurately midway between the lower edge of the rib cage and the upper edge of the hips. The hip circumference was measured at the height of the greater trochanters.[10] The waist-to-hip ratio (WHR) was also calculated. After obtaining blood samples from the patients' veins, fasting glucose and insulin levels were measured. A dietary interview was conducted to determine mean daily energy intake and the average amount of macronutrients such as protein, fats broken down into fatty acids, (polyunsaturated [PUFA], monounsaturated [MUFA], and saturated [SAFA] fatty acids), total carbohydrates, sucrose, lactose, cholesterol, and dietary fiber. The percentages of proteins, fats (PUFA, MUFA, and SAFA), total carbohydrates, sucrose, and lactose were also calculated. To accurately determine meal portions reported by patients, the atlas of food products was used.[11] A quantitative analysis of daily nutritional rations was conducted using a computer program, Diet 4 (National Food and Nutrition Institute, Poland).[12] The average body mass was measured in patients and controls. Based on the results, daily food intake was compared with the safety standards for people with moderate physical activity. The following shares of energy were included: protein, 12%; fat, 30% (SAFA, 10%; MUFA, 14%; PUFA, 6%); carbohydrates, 58% (max. sucrose, 10%; max. lactose, 10%), and the content of up to 300 mg of cholesterol along with about 25 g of fiber.[13]

Two atherogenic indices of diet were calculated: the Keys factor and P/S ratio. The Keys factor was calculated using the following formula: Keys factor = $1.35 \times (2 \times \%SAFA - \%PUFA) + 1.5 \times \sqrt{(Chol/1000\ kcal)}$, where %SAFA is the percentage of energy in daily food intake of SAFA; %PUFA is the percentage of energy in daily food intake of PUFA; and Chol – mg of cholesterol in a daily nutritional ration.[14] The normal value of this factor is between 32 and 38 for women and between 28 and 34 for men.[15] The P/S ratio represents the portion of PUFA to SAFA, namely, P/S = PUFA/SAFA. PUFA should cover 7% and SAFA 8% of food energy intake, so the optimum ratio should be 0.87.[14] The results that considerably deviate from these values are considered invalid.[14]

The calculations were done using StatSoft Statistica 7.0. Normality of distribution of continuous variables was verified using the Kolmogorov–Smirnov test with the Lilliefors correction and the Shapiro–Wilk test. To compare the quantitative variables with normal distribution, the *t* test was used, and, in cases with nonnormal distribution, the Mann–Whitney nonparametric test was

TABLE 2 Assessment of energy content in diet and protein consumption in the study and control groups

Parameter		Study group (n = 76)		Control group (n = 67)	
		women (n = 43)	men (n = 33)	women (n = 43)	men (n = 24)
energy value, kcal	mean ±SD	1626.6 ±493.3	2058.4 ±661.2	1564.2 ±487.2	2153.1 ±739.6
	range	676.9–2848.6	1048.4–3733.4	565.6–2425.7	1117.9–3713
	implementation of nutritional recommendation, %	77.4	77.6	80.2	87.9
protein, g	mean ±SD	72.2 ±20.9	91.3 ±32.7	66.0 ±18.9	92.0 ±23
	range	32.1–126.7	44.5–185.4	24.8–101.0	39.1–148.7
	implementation of nutritional recommendation, %	114.6	114.8	112.8	125.2
protein, %	mean ±SD	18.2 ±3.6	17.8 ±3.0	17.3 ±3.1	17.4 ±3.5
	range	12.6–27.1	10.9–24.9	12.9–30.3	10.0–24.9

Differences between the groups are not significant.

Abbreviations: see TABLE 1

applied. In addition, the Spearman's rank correlation test was used to determine the association between HOMA-IR and anthropometric measurements as well as dietary factors. A *P*-value of less than 0.05 was considered statistically significant. To characterize the groups, the following descriptive statistics were used: sample size, standard deviation, and the arithmetic mean and range. A forward stepwise regression model was also performed.

RESULTS BMI values were significantly higher in men and women with insulin resistance compared with controls. However, the mean waist circumference was significantly higher only in women with insulin resistance. Age and WHR values were similar between the groups regardless of sex. The characteristics of the study group are presented in TABLE 1.

Cardiovascular risk factors were present both in the study group and in controls. In the control group, high blood pressure was reported in 10 individuals (15%), hypercholesterolemia in 11 (16.5%), and heart attack/stroke in 3 (4.5%). In the study group, high blood pressure was observed in 27 patients (35.5%) and hypercholesterolemia was reported in 10 individuals (13.5%), half of which received cholesterol-lowering drugs. Only 1 person (1.3%) experienced a heart attack and stroke. The differences between the 2 groups were nonsignificant.

A quantitative assessment of the diet was made in both groups (TABLES 2, 3, and 4). Mean energy consumption was slightly higher in women in the study group and men in the control group, but the differences were nonsignificant. Patients' diet was characterized by insufficient daily energy and protein intake. However, the mean percentage of daily energy intake from protein was higher in women and men with insulin resistance (TABLE 2). We calculated the extent to which nutritional recommendations were implemented by patients, and observed that patients reported excessive levels of protein intake.

There were no significant differences in the consumption of carbohydrates and fiber (TABLE 3). The only significant differences were observed in the consumption of lactose in grams and the percentage of mean daily food intake between men in the study group and those in the control group, with higher intake reported in the latter. Of note, although the mean fiber content in the diet was higher in the study group, the maximum amount of fiber in the diet (nonsignificant differences) was higher in the control group. The study group consumed less carbohydrates than the control group, and none of the groups consumed enough carbohydrates and fiber.

No significant differences were observed in the consumption of fat or dietary cholesterol and the atherogenic index of the diet (TABLE 4). We observed a similar contribution of fatty acids in dietary energy content in both groups (TABLE 4). The daily food intake of all study subjects did not cover the requirements for MUFA and PUFA; however, it was near the upper limit of normal or exceeded the normal levels (as in men in the control group) for SAFA. The atherogenic index of the diet did not differ between the 2 groups. The Keys factor in women in both groups was too low. It was normal for men in the control group; it was lower in the study group than in the control group, but still within the normal range. Considering sex and the optimum P/S ratio (0.87), the values were too low for both groups.

This paper presents only the nutritional analysis of the conducted study. Based on the results, the correlation between HOMA-IR and anthropometric measurements, fractions of the lipid profile, and the percentages of macronutrients in dietary energy intake, the following model of forward stepwise regression was obtained: HOMA-IR = 0.126 × BMI + 0.009 × TG − 0.059 × sucrose − 1.133. BMI was the most strongly correlating factor in the model.

Considering the correlations between macronutrient intake and insulin resistance described by HOMA-IR, a positive correlation was observed between the HOMA-IR value and the percentage

TABLE 3 Carbohydrate intake in the study and control groups

Parameter		Study group (n = 76)		Control group (n = 67)	
		women (n = 43)	men (n = 33)	women (n = 43)	men (n = 33)
carbohydrates, g	mean ±SD	208.0 ±67.0	259.8 ±82.8	208.7 ±60.2	277.0 ±126.0
	range	93.2–441.6	140.2–484.3	93.4–319.8	104.2–640.3
	implementation of nutritional recommendation, %	68.0	67.6	73.0	78.0
carbohydrates, %	mean ±SD	51.9 ±8.8	51.1 ±7.0	54.4 ±7.2	50.6 ±9.6
	range	36.3–76.6	26.8–58.8	42.3–75.3	30.6–69.0
fiber, g	mean ±SD	16.6 ±5.9	20.9 ±7.9	15.8 ±5.5	21.3 ±10.3
	range	6.8–31.4	8.3–49.1	7.2–59.7	6.3–59.8
	implementation of nutritional recommendation, %	66.4	83.6	63.2	85.2
sucrose, g	mean ±SD	43.1 ±25.1	43.4 ±30.0	47.3 ±30.5	55.7 ±35.5
	range	11.8–101.8	8.6–134.6	0–140.3	13.0–139.7
	implementation of nutritional recommendation, %	82.0	65.5	97.0	91.0
sucrose, %	mean ±SD	10.5 ±4.6	8.4 ±4.5	11.8 ±5.6	9.9 ±4.2
	range	2.8–21.6	2–21.6	0–31.7	2.6–18.3
lactose, g	mean ±SD	10.0 ±6.9	8.6[a] ±10.0	10.3 ±6.4	13.7[a] ±12.2
	range	0.1–39.0	0.1–43.1	0–22.3	1.8–57.2
	implementation of nutritional recommendation, %	19.0	12.9	21.1	22.4
lactose, g	mean ±SD	2.6 ±1.7	1.7[a] ±2.1	2.8 ±1.9	2.5[a] ±1.7
	range	0–8	0–9.9	0–8.8	0.3–6.9

a significant differences between the groups ($P < 0.05$)

Abbreviations: see TABLE 1

of protein in the mean daily food intake (FIGURE 1). Moreover, there was a negative correlation between the HOMA-IR value and the percentage of sucrose in the diet (FIGURE 2).

DISCUSSION It is well known that insulin resistance participates in the pathogenesis of type 2 diabetes and cardiovascular diseases.[16,17]

The main cause of metabolic disorders and so called civilization diseases is unhealthy lifestyle. People, especially those professionally active, spend most of their time in the office or in the car. Too many duties usually do not leave enough time for sport or any type of physical activity. Lack of physical exercise, excessive food intake, excessive animal fat and sugar in the diet lead to an increase in weight and distort the proportion between subcutaneous and visceral fat tissues as well as between the amount of fat and muscle tissues.[18]

In the present study, mean daily carbohydrate intake among women was 208 g and did not differ between the study and control groups. In men, mean daily carbohydrate intake was not significantly higher in patients with insulin resistance compared with those without it (277.0 g/d and 259.8 g/d, respectively). The mean percentage of carbohydrates in the diet of the study subjects was about 51%, and it was higher only among women in the control group (mean 54.4% of the total food energy content). Fiber intake was similar in both groups although slightly higher in men. Volek et al.[19] studied the relationship between carbohydrate intake and insulin resistance and showed that reduction in carbohydrate intake had a positive effect on improving insulin sensitivity. Carbohydrate intake decreased from 47% to 12% in those subjects, which was also associated with a decrease in the mean intake of dietary fiber from 13 ±4 g to 9 ±5 g. Such a significant reduction in carbohydrate intake was followed by a decrease of the HOMA-IR value by 50%. Moreover, the serum glucose concentration decreased by a mean of 10 mg/dl, and insulin by a mean of half the baseline level.[19]

In our study, there were no significant differences in food energy between the study and control groups. Volek et al.[19] investigated the effect of lowering carbohydrates in the diet on metabolic syndrome and whether reducing carbohydrates was more beneficial than reducing fat. In both studies, food energy was significantly reduced. It should be noted that dietary restrictions used in both studies led to the reduction

TABLE 4 Assessment of dietary intake of fatty acids and atherogenic index of diet in the study and control groups

Parameter		Study group (n = 76)		Control group (n = 67)	
		women (n = 43)	men (n = 33)	women (n = 43)	men (n = 33)
fat, g	mean ±SD	58.4 ±26.5	75.9 ±30.5	55.9 ±24.4	78.3 ±30.0
	range	16.8–134.3	36.7–173.5	8.7–120.8	36.8–151.1
	implementation of nutritional recommendation, %	83.4	86.2	86.0	95.5
fat, %	mean ±SD	31.5 ±7.6	33.2 ±7.1	31.2 ±7.2	32.8 ±6.3
	range	13.7–43.9	22.8–54.1	13.5–44.8	22.6–45.4
cholesterol, mg	mean ±SD	281.2 ±148.2	369.3 ±157.8	273.3 ±111.5	374.9 ±155.7
	range	74.4–802.3	101.0–851.3	51.1–508.8	139.6–676.4
	implementation of nutritional recommendation, %	93.7	123.1	91.1	124.9
saturated fatty acids, g	mean ±SD	21.8 ±9.7	28.8 ±13.7	22.3 ±10.8	32.2 ±13.2
	range	5.6–46.82	7.7–65.4	3.8–47.8	14.5–67.3
	implementation of nutritional recommendation, %	92.7	98.3	97.0	118.0
saturated fatty acids, %	mean ±SD	11.9 ±3.4	12.6 ±4.4	12.3 ±3.4	13.5 ±3.1
	range	5.0–19.6	6.6–24.5	5.9–19.9	8.1–18.9
monounsaturated fatty acid, g	mean ±SD	24.3 ±12.3	31.0 ±14.4	22.0 ±10.3	30.5 ±12.8
	range	6.5–63.0	11.4–81.9	2.6–52.6	11.2–60.4
	implementation of nutritional recommendation, %	74.0	75.4	72.6	79.6
monounsaturated fatty acid, %	mean ±SD	13.0 ±4.0	13.5 ±3.5	12.3 ±3.6	12.8 ±3.3
	range	5.3–22.0	7.7–25.6	4.1–21.1	7–19.9
polyunsaturated fatty acid, g	mean ±SD	7.3 ±5.3	9.5 ±4.4	6.9 ±3.3	8.8 ±3.6
	range	1.7–35.3	4.0–20.9	1.1–18.7	2.2–17.8
	implementation of nutritional recommendation, %	52.0	54.0	48.5	53.6
polyunsaturated fatty acid, %	mean ±SD	3.9 ±1.8	4.2 ±1.7	3.9 ±1.5	3.7 ±1.0
	range	1.6–12.5	2.2–9.0	1.8–9.2	1.8–6.5
Keys factor	mean ±SD	27.6 ±9.5	29.1 ±13.1	28.8 ±9.3	32.5 ±9.0
	range	7.9–49.0	7.4–63.5	11.1–50.0	13.7–49.1
P/S ratio	mean ±SD	0.36 ±0.22	0.4 ±0.28	0.34 ±0.15	0.29 ±0.14
	range	0.15–1.42	0.1–1.29	0.16–0.86	0.09–0.81

Differences between the groups are not significant.

Abbreviations: see **TABLE 1**

of the HOMA–IR value. It can be assumed that this was due to decreased energy intake resulting from reduction of body weight.[19]

We observed that sucrose content in the diet was slightly higher in the control than in the study group. Moreover, contrary to previously published studies, we observed an inverse correlation between the HOMA-IR value and sucrose intake. Among others, Czerwonogrodzka et al.[20] showed that obese children consumed larger amounts of sucrose, which may be one of the dietary factors leading to overweight and obesity.[20]

Our results revealed a higher percentage of lactose intake in the diet of men with insulin resistance compared with insulin-sensitive men (2.5% vs. 1.7%, respectively). We believed that if the study had included a larger population sample and investigated not only the consumption of sucrose and lactose but also of fructose, it would have been easier to determine the role of carbohydrates in the pathogenesis and treatment of insulin resistance.

In our study, we noted that fat intake did not differ significantly between subjects with and without insulin resistance. We observed slightly

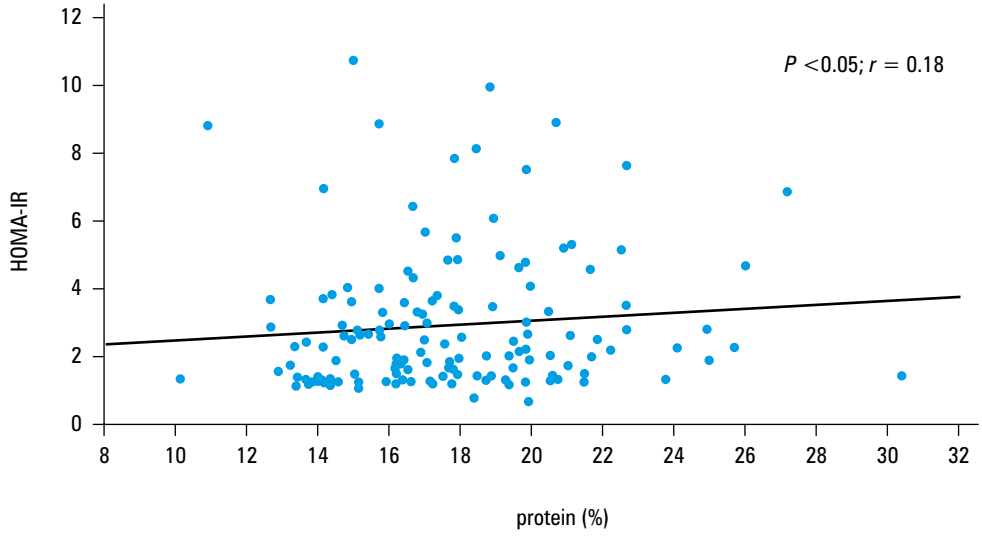

FIGURE 1 Correlation between HOMA-IR and the percentage of energy intake from protein

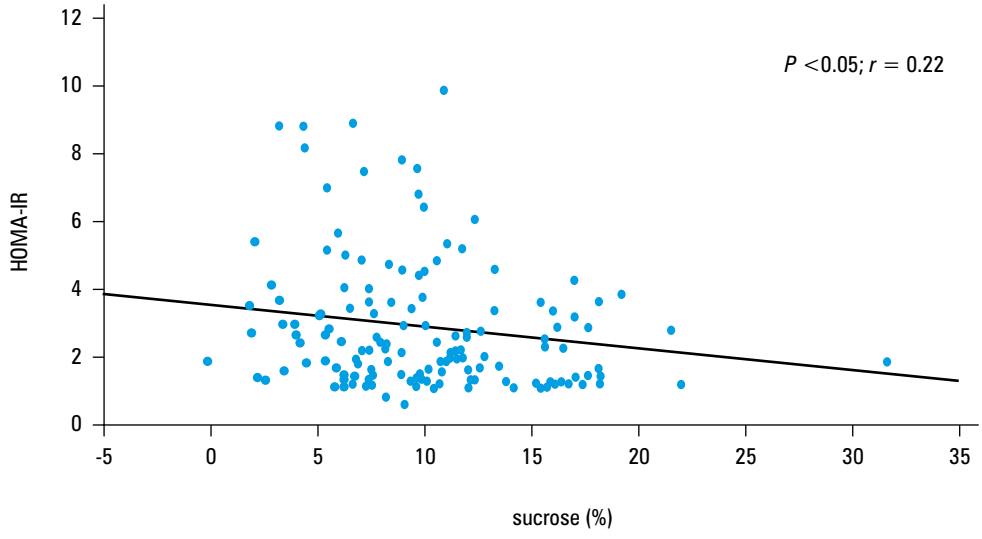

FIGURE 2 Correlation between HOMA-IR and the percentage of energy intake from sucrose

higher SAFA consumption by subjects without insulin resistance, while the intake of MUFA and PUFA was slightly higher among subjects with insulin resistance. Of note, the percentage of SAFA in the diet of our subjects was similar to that reported by WOBASZ, a Polish national survey performed in 2005.[15]

In our study, the mean Keys factor in the control group was 28.8, which is within the reference range,[14,15] while among women in the study group and among men both in the study and control groups, it was lower than the recommended values (27.6, 32.5, and 29.1, respectively). Additionally, the P/S ratio was lower than the recommended value (0.87) among women and men both in the study and control groups (0.36 and 0.34; 0.4 and 0.29, respectively). Similar results were obtained by Szczuko et al.[14] in a study that assessed the atherogenic index of the diet in young men.

Our results demonstrated a slightly higher percentage of protein in the diet. Volek et al.[19] showed that limiting carbohydrate intake resulted in higher protein consumption (in grams), while limiting fat intake caused lower consumption of protein (in grams). This is a result of significant protein and fat content in similar products. Moreover, it should be noted that in the study by Volek et al.,[19] regardless of macronutrient limitation, the total energy consumption decreased significantly in both groups. However, only in the group with increased protein from 16% to 28% energy intake, insulin sensitivity increased significantly. Further studies are needed to assess how different types of animal and vegetable proteins with their specific amino acid composition affect insulin resistance – its development and severity. This knowledge is necessary to revise dietary recommendations and treatment options in subjects with risk factors for insulin resistance.

In summary, there is no significant evidence for a direct link between nutrition and insulin resistance. Diet may affect insulin resistance in the long term but this was not confirmed in our study. However, a negative correlation was observed between the percentage of sucrose in the diet and HOMA-IR value and a positive correlation between protein intake and the HOMA-IR value.

Conclusions Our study showed a significantly higher intake of lactose in men without insulin resistance compared with those with insulin resistance. The results of our study encourage to conduct further, more detailed research involving a larger group of patients to better understand associations between dietary content and insulin resistance.

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ARTYKUŁ ORYGINALNY

Ocena wpływu żywienia i aterogenności diety na występowanie i nasilenie insulinooporności

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SŁOWA KLUCZOWE

aterogenność diety, dieta, insulinooporność

STRESZCZENIE

WPROWADZENIE Sposób żywienia jest jednym z czynników, które mogą stymulować uwarunkowania genetyczne i w konsekwencji prowadzić do wystąpienia insulinooporności. Odpowiednio zbilansowana podaż składników pokarmowych i prawidłowa energetyczność diety oraz zwiększona aktywność fizyczna są najskuteczniejszą profilaktyką zaburzeń metabolicznych.

CELE Celem badań było sprawdzenie czy istnieją zależności między sposobem żywienia, a wystąpieniem insulinooporności.

PACJENCI I METODY Badanie przeprowadzono w grupie 143 osób. Dokonano pomiaru stężenia glukozy i insuliny na czczo, a następnie wyliczono HOMA-IR dla każdego pacjenta. Pacjenci bez cukrzycy zostali podzieleni na grupę badaną i kontrolną. Badania obejmowały pomiary antropometryczne (masa ciała, wzrost i obwód talii), oznaczenia laboratoryjne (glukoza i insulina na czczo) oraz wywiad żywieniowy.

WYNIKI Stwierdzono ujemną korelację między procentową zawartością sacharozy w diecie, a wartościami HOMA-IR oraz dodatnią między spożyciem białka, a wartościami HOMA-IR. Ponadto obserwowano istotnie wyższe spożycie laktozy w grupie mężczyzn bez insulinooporności w porównaniu do mężczyzn z insulinoopornością.

WNIOSKI Wyniki przeprowadzonych badań zachęcają do podjęcia dalszych, bardziej szczegółowych analiz na większej grupie pacjentów w celu lepszego zrozumienia zależności między sposobem żywienia a insulinoopornością.

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