ANNALES
UNIVERSITATIS MARIAE CURIE-SKŁODOWSKA
LUBLIN – POLONIA
VOL. XXIV, N 4, 15 SECTIO DDD 2011

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Evaluation of serum adiponectin concentration in patients with type 2 diabetes

Ocena stężenia adiponektyny w surowicy krwi pacjentów z cukrzycą typu 2

INTRODUCTION

Adipose tissue is now known to express and secrete a variety of hormones and cytokines, which play a crucial role in the regulation of energy homeostasis, insulin sensitivity, lipid/carbohydrate metabolism and may contribute to the development of type 2 diabetes and cardiovascular disease, and these are collectively known as adipocytokines [11,18,27,34].

Among them adiponectin, a recently discovered protein produced by adipocytes, is thought to be a possible mediator between obesity, insulin resistance and type 2 diabetes. Accumulating experimental and clinical evidence has demonstrated that circulating adiponectin is bioactive protein with multivalent functions such as insulin sensitizing, anti-inflammatory, anti-atherogenic, lipid-oxidation enhancing, and vasodilatory activities [20,24,27].

This protein was identified in 1995 concurrently by the other four research groups, using different approaches, and has been reported as Acrp30 (adipocyte complement related- protein of 30kDA), apM1 protein (adipose most abundant gene transcript-1), AdipoQ, and GBP28 (gelatin-binding protein of 28kDa). Secreted exclusively and abundantly from white adipose tissue, adiponectin is a 30 kDa protein composed of 244 amino acids with high structural homology to collagen VIII, X, TNF-α and complement C1q [9,11,18,24]. It has been reported that adiponectin is present in the adipocytes and circulation as three major oligomeric forms: trimer (LMW, low molecular weight), hexamer (MMW, middle molecular weight) or multimer (HMW, high molecular weight). Among the isoforms of adiponectin, HMW-adiponectin is reported to be the most bioactive [22,27,28,30].

Adiponectin comprises approximately 0.01% of the total plasma protein in healthy humans, with plasma concentrations ranging from 3 to 30 μg/ml. Slightly increasing with age, adiponectin levels have a diurnal variation with nadir at night and peak in the morning [18,24,26] .

Although its structure and source are known, the role and clinical implications of adiponectin have not been definitively established [18]. Evidence is mounting that it is involved in insulin resistance, diabetes, inflammation, and atherosclerosis [20,24,27]. Recent research confirms that adiponectin concentration decreases with increasing body fat and its low levels may lead to the development of pathological states associated with obesity such as type 2 diabetes and cardiovascular disease [1,12,14,31,32].

The aim of this study was to evaluate serum adiponectin concentration in overweight and obese patients with type 2 diabetes, in comparison to healthy participants with normal body mass, and also assess the possible relationship of this cytokine with selected demographic, anthropometric and biochemical parameters.

MATERIAL AND METHODS

P a r t i c i p a n t s. The study was conducted in 40 patients with diabetes type 2 treated at the Endocrinology Clinic of the Independent Public Clinical Hospital No. 4 (SPSK 4) in Lublin, with the average duration *of the disease from diagnosis 9.4*±8.1 years. Among the enrolled subjects there were 17 women and 23 men with mean age 58.9±11.1 years. All patients underwent clinical examination. In the examined group of patients in medical history the following were found: arterial hypertension (75%), coronary artery disease (32%) and myocardial infarction (20%). According to the *criteria of World Health Organization,* based on body mass index (BMI), the percentage of diabetic patients who had overweight (BMI 25-29.9 kg/m^2), obesity (BMI 30-39.9 kg/m^2) and morbid obesity (BMI ≥40 kg/m^2), was as follows: 28%, 60% and 12%, respectively. Patients were excluded if they were known to have hepatic, renal and immunological disorders or cancer.

The control group was composed of 25 healthy subjects (11 women and 14 men) with the mean age of 55.3±7.1 years, with no disturbances in carbohydrate metabolism and with normal body mass, attending the periodic health checks at the Department of Laboratory Diagnostics of the Independent Public Clinical Hospital No. 1 in Lublin.

All participants gave written informed consent before study commencement. The study was performed in agreement with considerations, recommended by Ethics Committee of Medical University in Lublin.

B l o o d s a m p l i n g a n d m e a s u r e m e n t s. In every subject enrolled to the study concentrations of adiponectin, glucose, HbA_1C, total cholesterol, HDL-cholesterol, triglicerydes, urea, creatinine and *activities* of alanine *aminotransferase* (ALT) and aspartate *aminotransferase* (AST) were determined in fasting blood samples. LDL-cholesterol was calculated according to the Friedewald formula.

The material for the study was the peripheral blood obtained from the ulnar vein. Blood samples were drawn after an 8-12 h overnight fast between 8:00 and 10:00 to the tubes containing K_3EDTA in order to determine HbA_1C level and into the clot tubes in order to assess adiponectin and other biochemical parameters. Serum was separated from the collected blood samples by centrifugation for 10 min at 2000 rpm, aliquoted and stored frozen at -20˚C until assayed.

Serum adiponectin concentration was determined using a solid phase enzyme-linked immunosorbent assay, based on the principle of competitive binding (Human Adiponectin ELISA Kit, BioVendor Laboratory Medicine, Brno, Czech Republic) according to the manufacturer's protocol. The antibodies were specific for the human adiponectin protein, with an assay sensitivity of 210 ng/ml, using the volume of 50 µl diluted 30-times sample. The intra- and inter-assay precisions expressed as CV (%) were in the ranges 6.4-7.0% and 7.3-8.2%, respectively.

The concentrations of biochemical parameters were measured with the use of standard laboratory methods applied on Cobas 6000 analyzer (Roche, Basel, Switzerland) with dedicated reagents from the same company according to the manufacturer's specification.

Body mass index (BMI) was calculated as a quotient of body mass (kg) and squared height (m^2).

Statistical analysis. Statistical analysis was performed using the Statistica version 7.0 programme (StatSoft). The clinical data and values of selected biochemical parameters in all subjects were expressed by using elements of descriptive statistics (mean X, standard deviation SD, median Me). The distribution of the examined parameters was tested with the use of Shapiro-Wilk test. Variables were compared by Student's t-test when normally distributed and in case of nonparametric variables; comparisons were made by Mann-Whitney U-test. Correlations between analyzed variables were assessed with the use of the Pearson's and Spearman's tests.

A p value ≤ 0.05 was considered as statistically significant in all analyses.

RESULTS

Table 1 shows the results of determinations of the selected biochemical parameters and adiponectin concentrations in the patients with type 2 diabetes and healthy participants. The mean adiponectin concentration in the study group was 5.0±2.2 µg/ml and was significantly lower ($p<0.0001$) compared to the control group (8.9±3.3) (Fig.1). However, in the examined group females (6.0±2.2 µg/ml) had a significantly higher ($p<0.01$) adiponectin level than males (4.2±1.7 µg/ml) (Fig. 2).

Moreover, in diabetic patients we observed significantly higher concentrations of glucose ($p<0.0001$), triglicerydes ($p<0.0001$), creatinine ($p<0.001$) and activities of ALT ($p<0.001$) and AST ($p<0.01$) in comparison with control subjects, whereas the level of HDL-cholesterol in the study group was significantly lower than in the control group ($p<0.0001$). Among other biochemical parameters, total cholesterol, LDL-cholesterol and urea concentrations were not significantly different in the examined group of patients with type 2 diabetes in comparison to the control group. The mean value of HbA_1C in diabetic patients was 8.7±2.1%.

Table 1. Serum concentrations of adiponectin (µg/ml) and selected biochemical parameters in the study and control group

Parameters	Study group (n=40)		Control group (n=25)	
	X±SD	Me	X±SD	Me
Adiponectin (µg/ml)	5.0± 2.2‡	4.6	8.9±3.3	7.9
Glucose (mg/dl)	176.8±49.6‡	166.0	82.6±8.2	82.8
HbA1C (%)	8.7±2.1	7.9	Not performed	
Total cholesterol (mg/dl)	190.3±47.3	183.0	193.9± 35.6	189.0
HDL-cholesterol (mg/dl)	43.2±10.8‡	43.0	57.6± 12.9	57.0
LDL-cholesterol (mg/dl)	108.4±40.2	99.0	114.8± 31.2	119.0
Triglicerydes (mg/dl)	204.6±89.2‡	182.0	106.9± 62.3	90.0
Urea (mg/dl)	36.7±12.9	35.2	32.6±8.6	31.5
Creatinine (mg/dl)	0.9±0.2†	0.9	0.8±0.2	0.7
Alanine *aminotransferase* (U/l)	32.4±19.3†	27.5	20.7±14.5	17.3
Aspartate *aminotransferase* (U/l)	27.3±12.7*	24.0	21.2±9.24	18.9

* p< 0.01; † p< 0.001; ‡ p< 0.0001

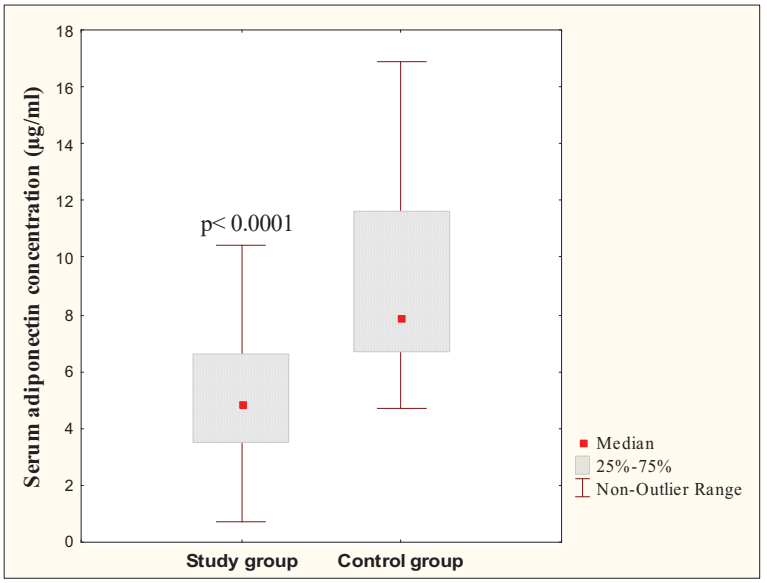

Figure 1. Serum concentration of adiponectin (µg/ml) in the study and control group

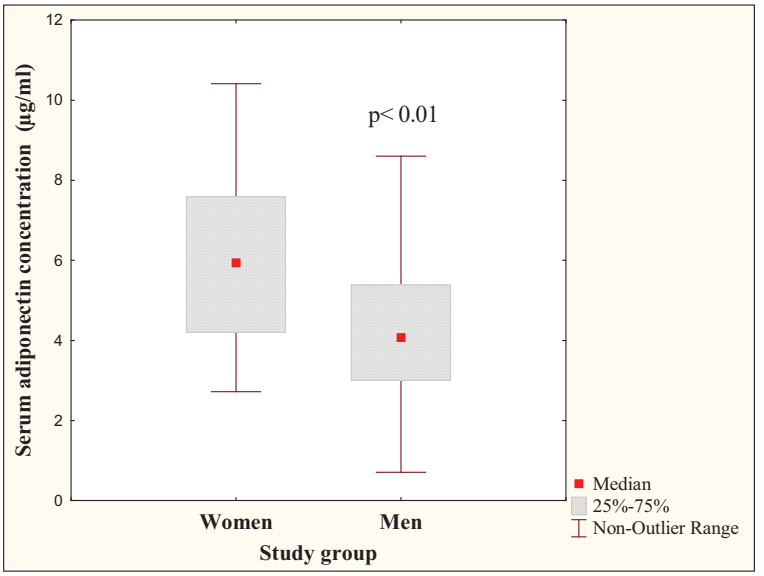

Figure 2. Serum concentration of adiponectin (µg/ml) in the study group depending on gender

In the present study, we also examined the relationship between serum adiponectin concentration and age and the values of selected demographic, anthropometric features and biochemical parameters in the group of diabetic subjects (Table 2).

Based on statistical analysis we observed significant positive correlations between adiponectin concentration and age and level of HDL-cholesterol in the study group. Furthermore, significant negative correlations were found between adiponectin concentration and body mass, BMI, level of glucose, HbA_1C, triglicerydes and activity of ALT, AST. However, no significant relationships were observed between the values of biochemical parameters such as total cholesterol, LDL-cholesterol, urea, creatinine and adiponectin concentration in diabetic patients

Table 2. Correlation coefficients of the studied variables with adiponectin concentration in the group of diabetic patients

PARAMETERS		STUDY GROUP (n=40)	
		Correlation coefficient r	Level p
ADIPONECTIN (µg/ml)	Age (yaers)	0.346	0.019
	Body mass (kg)	-0.514	0.000
	BMI (kg/m^2)	-0.310	0.038
	Glucose (mg/dl)	-0.495	0.000
	HbA_1C (%)	-0.430	0.005
	Total cholesterol (mg/dl)	-0.0412	NS
	HDL-cholesterol (mg/dl)	0.409	0.012
	LDL-cholesterol (mg/dl)	0.004	NS
	Triglicerydes (mg/dl)	-0.549	0.000
	Urea (mg/dl)	0.105	NS
	Creatinine (mg/dl)	-0.069	NS
	Alanine *aminotransferase* (U/l)	-0.388	0.009
	Aspartate *aminotransferase* (U/l)	-0.307	0.043

p- level of statistical significance ($p \leq 0.05$); NS- statistically insignificant

DISCUSSION

A growing body of literature has demonstrated that in many ethnic groups adiponectin gene expression and circulating adiponectin levels are lower in patients with type 2 diabetes than in nondiabetic individuals [1, 13, 16, 26]. It has been hypothesized that reduced adiponectin concentrations observed in obese subjects are involved in the development of type 2 diabetes, atherosclerosis and cardiovascular disease [11, 27]. We also revealed significant decrease in adiponectin concentration in serum of patients with type 2 diabetes compared to the control group, which remains consistent with the results of other authors [4, 31].

In the present study, we examined the relationship between serum adiponectin concentration and selected demographic, anthropometric and biochemical parameters in the group of diabetic subjects.

As adiponectin is secreted primarily by the adipocytes, it is not surprising that adiponectin concentrations in the serum are affected by changes in adipose tissue mass [28]. However, unlike most adipose-derived hormones and secreted proteins, levels of adiponectin are paradoxically reduced in obese animals and humans [1, 11, 13, 14]. Several studies have shown that oobesity, in general, is associated with decreased adiponectin expression in adipose tissue and plasma levels [1,15-17]. Furthermore, recent research has reported that in both men and women, overall obesity, assessed by parameters such as body mass index (BMI) and fat mass, is negatively correlated to adiponectin concentration, although prolonged weight reduction leads to increased adiponectin levels [1,12,31,32]. Indeed, weight loss (>20% of body weight) via bariatric surgery results in significant increases in circulating adiponectin levels within 6-12 months after surgery [28,3 2]. Other human clinical studies also support these outcomes [22, 28, 31].

In this study, adiponectin serum concentrations were found to be markedly reduced in overweight and obese patients with type 2 diabetes, in comparison to healthy participants with normal weight. Furthermore, significant negative correlation was observed between adiponectin concentration and body mass and BMI in diabetic patients in our research. Similar results were obtained by other authors [4, 5, 19], also in different rodent models of obesity and in rhesus monkeys [13,14].

Numerous studies have underlined that not only fat mass but the distribution of adipose tissue determines adiponectin levels [28]. There is a strong negative correlation between adiponectin levels and visceral or central fat, compared to subcutaneous fat [4,10,23]. A cross-sectional study conducted by Cnop et al. [5] demonstrates that in obese and lean men and women, the negative relationship between plasma adiponectin and visceral fat (measured by computed tomography scan) is significantly stronger than that with subcutaneous fat. One explanation is that adiponectin is primarily produced by visceral adipose tissue, but that large triglyceride-filled visceral adipocytes produce less adiponectin. This conclusion agrees with a result published by Motoshima et al. [19] indicating that adiponectin levels were associated with intra-abdominal fat distribution. Furthermore, in the experiment performed by Lihn et al. [17] comparison of adiponectin gene expression in different groups from lean, obese, and obese diabetic subjects shows adiponectin mRNA levels were lower in visceral versus subcutaneous adipose tissue in all groups and adiponectin expression was even lower in visceral adipose tissue from obese subjects with diabetes.

Data from the literature [3, 4, 6] and results of this research have shown that circulating adiponectin levels increase with age and are higher in women compared with men, independent of body composition. Interestingly, the relationship between fat mass and adiponectin does not explain the sexual dimorphism in total adiponectin levels, suggesting that additional factors beyond fat mass are responsible for the regulation of adiponectin [6]. It is suggested that this sexual dimorphism may be due to a selective increase in high molecular weight oligomers in women. Moreover, numerous studies have revealed that androgens inhibit the production of adiponectin in animal models and tissue culture of adipocytes [3, 24, 26].

There is a growing body of evidence that adiponectin is involved in the regulation of both lipid and carbohydrate metabolism. Available data have suggested the role of adiponectin in the regulation of insulin action, and that it might reduce hepatic glucose production and increase muscle glucose utilization [11, 24, 25, 28].

One of the significant cardiovascular risk factors in type 2 diabetes is dyslipidemia [24]. Recent reports show that circulating adiponectin may exert anti-atherogenic effects through the modulation of lipid metabolism [11, 28]. Evidence from animal and in vitro studies suggests adiponectin protects against the development of atherosclerosis [7, 18]. Although it is tempting to speculate that adiponectin may modulate serum lipids through direct effects on the liver and/or adipose tissue, the mechanisms mediating the relationship between adiponectin and lipid metabolism are unknown [28].

Data from two large cross-sectional studies conducted by Cnop et al. [4] and Tschritter et al. [29] indicate that after adjusting for both sex and body adiposity, circulating adiponectin concentrations are negatively correlated with triglycerides levels and strongly positively correlated with plasma high-density lipoprotein (HDL) cholesterol concentrations. In contrast, several clinical studies show low-density lipoprotein (LDL) cholesterol and total cholesterol do not have significant independent

relationships to adiponectin levels [2,1 0]. These *findings* are in agreement *with our* and other authors [12]. Furthermore, reports in the literature show that the relationship between adiponectin and plasma lipids is independent of age, gender, BMI and insulin sensitivity [2, 4, 33].

It is notable that in our study we observed significant negative correlations between adiponectin concentration and activity of ALT and AST in diabetic patients, thus suggesting a role for adiponectin in the maintenance of liver integrity. Numerous studies have underlined that adiponectin might be able to preserve liver function by preventing lipid accumulation in hepatocytes [21]. Hoveower, Ezenwaka et al. [8] suggest that low serum adiponectin level may not be a suitable marker for impaired liver function in diabetic patients. Although the available evidence indicates that adiponectin stimulates fatty acid oxidation in liver and skeletal muscle, it is still unknown whether circulating adiponectin levels are altered in disorders of hepatic metabolism of energy substrates [11, 21, 25, 29]. Current data suggest that adiponectin may have a protective role in liver injury in nonalcoholic fatty liver disease in mice, but direct evidence of the role of adiponectin in human is lacking [21].

The available literature [14, 20, 23-28] and results of this study confirm that adiponectin is a key adipokine with multiple actions in the human body. Prospective studies are required to better elucidate the role of adiponectin in metabolic and cardiovascular diseases.

CONCLUSION

On the basis of the results obtained, it can be concluded that in overweight and obese patients with type 2 diabetes adiponectin serum concentrations are markedly lower in comparison to healthy participants. Moreover, obese women with type 2 diabetes have higher serum levels of adiponectin than men in the study group. Serum adiponectin concentration correlates positively with age and level of HDL-cholesterol and negatively with body mass, BMI, level of glucose, HbA_1C, triglicerydes and activity of ALT, AST in diabetic patients. In conclusion, we report that decreased adiponectin concentration in overweight and obese patients with type 2 diabetes in comparison to healthy subjects and the observed correlations of adiponectin with lipid metabolism parameters may suggest antiatherogenic effects of this protein.

However, further studies are needed to clarify the clinical significance of adiponectin for the prevention and treatment of the pathological states associated with obesity such as type 2 diabetes and cardiovascular disease.

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ABSTRACT

Adiponectin, a recently discovered protein secreted exclusively and abundantly from adipose tissue, known as Acrp30 (adipocyte complement-related protein of 30kDa), AdipoQ, apM1 protein (adipose most abundant gene transcript-1), and GBP28 (gelatin-binding protein of 28kDa), comprises approximately 0.01% of the total plasma protein in healthy humans. Recent studies have suggested that circulating adiponectin is a bioactive protein with multivalent functions such as insulin sensitizing, anti-inflammatory, anti-atherogenic activities and also prevents endothelial dysfunction. Although its structure and source are known, the role and clinical implications of adiponectin have not been definitively established. The aim of this study was to evaluate serum adiponectin concentration in overweight and obese patients with type 2 diabetes, in comparison to healthy participants, and also assess the possible relationship of this cytokine

with selected demographic, anthropometric and biochemical parameters. The study was conducted in 40 patients with diabetes type 2 with the average duration *of the disease from diagnosis 9.4*±8.1 years. Among the enrolled subjects were 17 women and 23 men with mean age 58.9±11.1 years. The control group was composed of 25 healthy subjects (11 women and 14 men) with the mean age of 55.3±7.1 years, with no disturbances in carbohydrate metabolism and with normal body mass. The mean adiponectin concentration in the study group was significantly lower compared to the control group. However, in the examined group the females had a significantly higher adiponectin level than males. We observed significant positive correlations between adiponectin concentration and age and level of HDL-cholesterol in the study group. Furthermore, significant negative correlations were found between adiponectin concentration and body mass, BMI, level of glucose, HbA_1C, triglicerydes and activity of ALT, AST. In conclusion, we report that decreased adiponectin concentration in overweight and obese patients with type 2 diabetes in comparison to healthy subjects and the observed correlations of adiponectin with lipid metabolism parameters may suggest antiatherogenic effects of this protein.

Keywords: adiponectin, adipose tissue, diabetes

STRESZCZENIE

Adiponektyna, stosunkowo niedawno odkryte białko wydzielane wyłącznie przez tkankę tłuszczową, znane również pod nazwami: Acrp30 (adipocyte complement related protein of 30 kDA), AdipoQ, apM1 (adipose tissue most abundant gene transcripte) oraz GBP28 (gelatin-binding protein of 28kDA), występuje w organizmie w dość dużej ilości, stanowi bowiem 0,01% wszystkich białek osocza. Badania sugerują, że adiponektyna jest białkiem aktywnym metabolicznie ze względu na swoje potencjalne działanie insulinouwrażliwiające, przeciwmiażdżycowe, przeciwzapalne oraz protekcyjne na śródbłonek naczyń. Jednakże rola i znaczenie adiponektyny w wielu procesach patologicznych nie są do końca jednoznacznie wyjaśnione. Celem niniejszej pracy była ocena stężenia adiponektyny oraz wybranych parametrów biochemicznych u chorych z cukrzycą typu 2 z towarzyszącą nadwagą i otyłością oraz osób zdrowych bez zaburzeń gospodarki węglowodanowej i z prawidłową masą ciała oraz ocena zależności pomiędzy stężeniem adiponektyny a wybranymi parametrami demograficznymi, antropometrycznymi i biochemicznymi. Badania przeprowadzono u 40 chorych, u których rozpoznano cukrzycę typu 2 (17 kobiet i 23 mężczyzn w wieku 58,9±11,1 lat). Średni czas od rozpoznania cukrzycy u tych chorych wynosił 9,4±8,1 lat. Grupę kontrolną stanowiło 25 osób zdrowych (11 kobiet i 14 mężczyzn w wieku 55,3±7,1 lat). Średnie stężenie adiponektyny u chorych z cukrzycą typu 2 z towarzyszącą nadwagą i otyłością było istotnie statystycznie niższe niż u osób zdrowych. W grupie pacjentów stwierdzono znamiennie wyższe stężenie adiponektyny u kobiet niż u mężczyzn. Stężenie adiponektyny korelowało dodatnio z wiekiem oraz stężeniem cholesterolu HDL u chorych z cukrzycą typu 2. Zaobserwowano ponadto istotne ujemne zależności pomiędzy stężeniem adiponektyny a masą ciała i BMI oraz wartościami glukozy, HbA_1C, triglicerydów, a także aktywnością ALT i AST. Obniżone stężenie adiponektyny u chorych z cukrzycą typu 2 z towarzyszącą nadwagą i otyłością oraz obserwowane zależności pomiędzy stężeniem adiponektyny a parametrami odzwierciedlającymi metabolizm lipidów mogą sugerować jej przeciwmiażdżycowe właściwości.

Słowa kluczowe: adiponektyna, tkanka tłuszczowa, cukrzyca