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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Alcohol intake and cardiovascular risk factor profile in men participating in the WOBASZ study

Anna Waśkiewicz, Elżbieta Sygnowska

Department of Epidemiology, Cardiovascular Diseases Prevention, and Health Promotion, Institute of Cardiology, Warsaw, Poland

Abstract

Background: Studies showed that alcohol intake affects various biomarkers (lipids, blood pressure [BP], homocysteine, diabetes, haemostatic factors) associated with the risk of coronary heart disease.

Aim: To determinate cardiovascular (CV) risk factor profile in a population Polish men stratified according to alcohol intake.

Methods: Within the frame of the National Multicentre Health Survey (WOBASZ), a sample of 6912 men aged 20–74 years representative for the general population in Poland was screened in 2003–2005. A wide range of CV risk factors was assessed in all participants on the basis of questionnaires, laboratory tests, anthropometric studies, and BP measurements. Annual beer, wine and vodka intake was assessed using a standardised questionnaire, and daily pure ethanol intake was calculated. The studied subjects were divided into 4 groups: abstainers (A), light drinkers (L; ≤ 15 g ethanol/day), moderate drinkers (M; 15–30 g ethanol/day), and heavy drinkers (H; > 30 g ethanol/day).

Results: A positive association between alcohol consumption and systolic BP (A: 134.0, L: 136.9, M: 139.7, H: 141.3 mm Hg), diastolic BP (81.1, 83.3, 85.9, 87.1 mm Hg, respectively), high-density lipoprotein cholesterol (HDL-C) level (1.25, 1.34, 1.45, 1.61 mmol/L, respectively), and triglyceride level (1.59, 1.63, 1.82, 2.00 mmol/L, respectively) was observed. After adjustment for confounding factors, moderate drinkers were found to have a 37% higher risk of hypertension, a 25% higher risk of elevated trigliceride level, a 40% lower risk of low HDL-C level and a 35% lower risk of diabetes compared to light drinkers. Heavy alcohol consumption increased the likelihood of hypertension by 52%, elevated triglycerides by 46% and hyperhomocysteinaemia by 95%, and decreased the likelihood of low HDL-C by 44%.

Conclusions: In the Polish population, negative consequences of alcohol intake were seen among men who consumed more than 15 g of ethanol daily. A potential positive effect of alcohol consumption, as manifested by higher HDL-C level a lower likelihood of diabetes (only with moderate alcohol intake), was counterbalanced by a negative effect on BP, homocysteinaemia, and triglycerides.

Key words: alcohol intake, blood pressure, HDL-cholesterol, triglycerides, homocysteine, diabetes, Polish population

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INTRODUCTION

Studies published during the last several years [1–12] suggested that moderate alcohol intake of 1–3 drinks per day might contribute to a lower risk of cardiovascular diseases (CVD).

Alcohol affects the cardiovascular system by modifying CVD risk factors including lipid levels, blood pressure (BP), homocysteine, diabetes, and haemostatic factors, but these effects may be negative or positive depending on the specific factor and alcohol dose.

Although moderate alcohol intake may have some beneficial effects leading to lower CVD mortality [1, 4, 6, 9, 11, 13–15], regular or episodic intake of large amounts of alcohol is associated with a higher coronary artery disease risk compared to abstinence [16, 17].

The purpose of this study was to evaluate CVD risk factor profile among adult men reporting varied alcohol intake.

METHODS

We analysed data from the National Multicentre Health Survey (Wieloośrodkowe Ogólnopolskie Badanie Stanu Zdrowia Ludności, WOBASZ) performed in 2003–2005. The study involved a representative sample of Polish citizens aged

Address for correspondence:
Anna Waśkiewicz, M.Sc., PhD, Department of Epidemiology, Cardiovascular Disease Prevention, and Health Promotion, Institute of Cardiology, ul. Alpejska 42, 04–628 Warszawa, Poland, tel: +48 22 815 65 56, e-mail: awaskiewicz@ikard.pl
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20–74 years. The study was approved by an appropriate ethics committee, and all patients gave informed consent for participation in the study. Details regarding sampling and study conduct were published previously [18]. In the present study, we report on 6912 men participating in the WOBASZ study for whom biochemical testing results and reliable data on alcohol intake were available. For homocysteine levels, the analysis involved 3359 men as this biochemical parameter was measured in 50% of the study sample.

Information regarding alcohol intake was obtained using a standardised questionnaire that included questions on usual consumption of wine, beer, and vodka during the previous year. Taking into account the usual dose, frequency of intake, and mean alcohol content in specific alcoholic beverages, daily intake of pure ethanol was calculated for each respondent.

The studied subjects were divided into 4 groups: abstainers (persons reporting no alcohol intake during the previous year), light drinkers (≤ 15 g ethanol/day), moderate drinkers (15.01–30 g ethanol/day), and heavy drinkers (> 30 g ethanol/day).

Data on socioeconomic characteristics and habits regarding smoking and drinking were self-reported using a special patient questionnaire. BP was measured 3 times on the right arm using a automated Omron M-51 device with an appropriately sized cuff, and BP was defined as the mean of the second and third measurement. Lipid levels, glucose, and homocysteine were measured in a single core laboratory that participates in standardisation programs run by the Centres for Disease Control (Atlanta, GA, USA) and in the European RANDOX project. High-density lipoprotein cholesterol (HDL-C) was measured by homogenous colorimetric immunoassay (PEG), and triglyceride level by the enzymatic colorimetric method (GPO/PAD). Lipid levels were measured using the INTEGRA 400 analyser and commercial ROCHE kits. Blood glucose level was measured by the enzymatic method with hexokinase, and homocysteine level by the immunoenzymatic method using the IMMULITE 1 analyser and DPC reagents.

Statistical analysis

Statistical analyses were performed using the Statistical Analysis System (SAS) software, version 9.2. To evaluate levels of socioeconomic factors and CVD risk factors in groups with varying alcohol intake, we used age-adjusted analysis of covariance (GLM procedure) for continuous variables, and χ^2 test (FREQ procedure) for categorical variables. Probability of abnormal levels of selected CVD risk factors in groups with varying alcohol intake was evaluated using a logistic regression model (LOGISTIC procedure) adjusted for age (Model 1) and for age, education, net family income per person, body mass index, smoking status, and physical activity (Model 2). Men consuming small amounts of alcohol (0.1–15.0 g ethanol/day) were the reference group.

RESULTS

Among adult Polish men, the largest group (72%) consumed beer, wine, and vodka for a total of 0.01–15.0 g of pure ethanol per day, followed by men consuming 15.01–30.0 g of ethanol per day (13% of the respondents) and those who reported abstinence (8% of the respondents). Consumption exceeding 30 g of ethanol per day was reported by 7% of the respondents. The most commonly consumed alcoholic beverages were beer (providing 54% to 63% of the consumed ethanol depending on the analysed group) and vodka (providing 30% to 36% of the consumed ethanol).

The abstainer group was characterised by the highest proportion of older and married men, and the lowest proportion of men with higher education and net family income per person above 1000 PLN per month. Proportions of divorced men and men reporting elementary education rose with increasing alcohol consumption. Heavy drinkers had relatively low income, as the net family income per person did not exceed 500 PLN per month in 58% of families (Table 1).

The abstainer group also showed highest rates of hypertension, overweight and obesity, low HDL-C level, diabetes, and a history of smoking. Alcohol intake was related to levels of some CVD risk factors, with a positive association between alcohol consumption and mean systolic and diastolic BP, HDL-C and triglyceride levels, and the proportion of hypertensives, subjects with hypertriglyceridaemia, and smokers, and a negative association with the proportion of subjects with low HDL-C and overweight or obese subjects. The lowest proportion of diabetics was noted among moderate drinkers (Table 2).

Alcohol intake was significantly associated with the probability of abnormal levels of the analysed CVD risk factors in both employed logistic regression models (Table 3). After adjustment for confounding variables (Model 2), moderate drinkers (intake of 15.01–30.0 g ethanol per day) were found to have a 37% higher risk of hypertension, a 25% higher risk of elevated triglyceride level, a 40% lower risk of low HDL-C level, and a 35% lower risk of diabetes compared to light drinkers (intake of 0.01–15.0 g ethanol per day; reference group). Among heavy drinkers (intake of > 30 g ethanol per day), the risk of hypertension was increased by 52%, the risk of elevated triglyceride level increased by 46%, the risk of elevated homocysteine level increased by 95%, and the risk of low HDL-C level reduced by 44%. The risk of elevated low-density lipoprotein cholesterol (LDL-C) level was not associated with alcohol intake. The probability of abnormal levels of the analysed CVD risk factors among abstainers did not differ significantly as compared to the reference group.

DISCUSSION

Our findings indicate that among adult men in Poland, alcohol intake of more than 15 g of ethanol per day (as compared to the daily intake of < 15 g of ethanol) was associated with

Table 1. Characteristics of the study population

	Ethanol consumption [g/day]				
	Abstainers[1]	Light drinkers (0.01–15.00)	Moderate drinkers (15.01–30.00)	Heavy drinkers (> 30.00)	P
Number of subjects	561	4998	875	478	
Mean ethanol consumption [g/day]	0	4.7	20.0	54.3	
including:					
as vodka [g/day]	0	1.7	5.9	17.1	
as wine [g/day]	0	0.3	1.5	8.0	
as beer [g/day]	0	2.7	12.6	29.2	
Mean age [years] including:	52.9	45.5	42.3	43.4	< 0.0001
20–40 (% of subjects)	21.9	38.3	45.7	40.0	< 0.0001
41–60 (% of subjects)	40.3	42.4	44.6	50.4	
> 60 (% of subjects)	37.8	19.3	9.7	9.6	
Marital status:					< 0.0001
married (% of subjects)	75.3	74.2	68.9	61.1	
divorced (% of subjects)	3.0	2.5	3.2	7.3	
Education:					< 0.0001
elementary (% of subjects)	67.4	58.2	59.7	71.3	
higher (% of subjects)	6.4	11.0	10.5	6.5	
Family income per person[2]:					< 0.0001
< 500 PLN (% of subjects)	51.3	49.5	47.2	57.7	
> 1000 PLN (% of subjects)	10.4	12.3	13.5	11.2	

[1]Subjects reporting no alcohol intake during the last year before the study; [2]Only those subjects who answered the question regarding net family income per persons were included.

an increased level of elevated BP and triglycerides, and daily intake of > 30 g of ethanol was also associated with elevated homocysteine levels.

Previous studies regarding the effect of alcohol intake on BP gave inconsistent results, with some studies showing no association of BP with moderate drinking, or increase in BP only above a certain threshold of alcohol intake, and some other studies showing increased rates or risk of hypertension with increasing alcohol intake. The former include studies in young Americans [19] in whom no association was found between alcohol intake and the incidence of hypertension during a 20-year follow-up, and in somewhat older inhabitants of Amsterdam in whom no relation between alcohol intake and hypertension was found during 4 years of follow-up [20].

In the Kaiser Permanente Health Screening Survey [21], a relation was noted between alcohol dose and BP in white Americans who consumed alcohol daily, with highest BP values found for the intake of 6–8 drinks per day. A similar relation was also found in our study, as both systolic and diastolic BP and proportion of hypertensives rose with increasing alcohol intake. In the United States, alcohol intake above 210 g per week was an independent risk factor for the development of hypertension in men during a 6-year follow-up [22]. Also in men followed up for nearly 22 years in the Physicians' Health Study [23], moderate alcohol intake was associated with an increased risk of hypertension. Finally, a metaanalysis of 156 studies including nearly 117,000 subjects [2] showed that any alcohol intake was associated with an increased risk of hypertension.

Alcohol also affects lipid profile [3, 7], increasing HDL-C levels, and in some studies also triglyceride levels. In our study, an increase in daily alcohol dose was associated with higher HDL-C and triglyceride levels, as well as the likelihood of abnormal triglyceride level, similarly to findings reported in French subjects [3] in whom levels of these lipids were lowest among abstainers, and Japanese subjects [5] in whom the frequency of alcohol consumption affected HDL-C level regardless of the weekly alcohol dose.

A metaanalysis of 25 studies [9] showed that compared to total abstinence, daily intake of 30 g of ethanol was associated with an increase in HDL-C level by 4 mg/dL. This relation was also confirmed in a metaanalysis of 44 intervention trials [13] assessing changes in selected biomarkers that occurred with alcohol intake vs. a period of no alcohol intake. In this study, alcohol intake of 12.5–29.9 g per day was associated with an increase in HDL-C level by 0.07 mmol/L, intake of 30–60 g per day with an increase in HDL-C level by 0.10 mmol/L, and intake above 60 g per day with an increase in HDL-C level

Table 2. Mean levels of cardiovascular disease risk factors (age-adjusted) and their rates in the study population

Parameter	Ethanol consumption [g/day]				
	Abstainers[1]	Light drinkers (0.01–15.00)	Moderate drinkers (15.01–30.00)	Heavy drinkers (> 30.00)	P
Systolic BP [mm Hg]	134.0	136.9	139.7	141.3	< 0.0001
Diastolic BP [mm Hg]	81.1	83.3	85.9	87.1	< 0.0001
HDL cholesterol [mmol/L]	1.25	1.34	1.45	1.61	< 0.0001
LDL cholesterol [mmol/L]	3.24	3.28	3.33	3.24	NS
Triglycerides [mmol/L]	1.59	1.63	1.82	2.00	< 0.0001
Homocysteine[2] [μmol/L]	10.69	10.16	10.05	11.33	< 0.0001
Glucose [mmol/L]	5.07	5.12	5.13	5.19	NS
Hypertension[3] (% of subjects)	45.3	39.1	40.8	44.1	0.0082
HDL cholesterol < 1 mmol/L (% of subjects)	19.6	16.3	11.1	10.7	0.0001
LDL cholesterol ≥ 3 mmol/L (% of subjects)	60.6	59.0	57.7	54.6	NS
Triglycerides > 1.7 mmol/L (% of subjects)	30.5	31.5	35.5	39.2	0.0039
Homocysteine ≥ 12 μmol/L (% of subjects)	36.7	25.5	22.3	37.6	< 0.0001
Overweight and obesity: BMI ≥ 25 kg/m^2 (% of subjects)	64.1	61.0	59.9	54.7	0.0173
Diabetes[4] (% of subjects)	12.20	7.65	4.21	6.86	< 0.0001
Smokers (% of subjects)	28.1	36.9	47.5	62.1	< 0.0001
Former smokers[5] (% of subjects)	35.9	29.4	26.9	23.8	

[1]Subjects reporting no alcohol intake during the last year before the study; [2]Geometrical mean; [3]Defined as systolic BP ≥ 140 mm Hg and/or diastolic BP ≥ 90 mm Hg or antihypertensive drug treatment; [4]Defined as fasting blood glucose > 7 mmol/L and/or previous diagnosis of diabetes; [5]Defined as currently non-smoking subjects who reported previous regular smoking for at least 1 year; BP — blood pressure; BMI — body mass index; HDL — high-density lipoprotein; LDL — low-density lipoprotein; NS — nonsignificant

by 0.14 mmol/L. In contrast, these 2 metaanalyses yielded inconsistent results in regard to the effect of alcohol on triglycerides: in the former, daily intake of 30 g of ethanol was associated with an increase in triglyceride level by 5.7 mg/dL, while no such relationship was found in the latter. Both our study and the above mentioned metaanalysis of 44 intervention trials [13] showed no association between alcohol intake and LDL-C level.

In our study, a negative effect of alcohol consumption on homocysteine level become evident only with daily intakes above 30 g. In other studies, an association between alcohol intake and homocysteine level was not entirely clear, with most studies indicating such a relationship [24, 25], but one study showed an inverse relation between these 2 parameters [26]. A study performed in German men [24] found that regardless of the type of alcoholic beverage (beer, wine, vodka), daily consumption of 30 g of ethanol during 6 weeks was associated with an increase in homocysteine level by about 17–25%. In a Japanese study [25], homocysteine level was higher among subjects consuming various types of alcoholic beverages (whisky, sake, wine, beer) compared to abstainers but the likelihood of hyperhomocysteinaemia was found to increase only with increasing consumption of whisky. In contrast, a German study [26] found a linear decrease in homocysteine level with increasing alcohol consumption. Of note, 75% of ethanol intake in this study population came from consumption of beer which a source of B vitamins which may lower homocysteine level.

Both in our study and in French [3], Danish [4] and U.S. populations [23, 27] highest proportions of diabetic subjects were noted among abstainers which may indicate that some men stopped drinking due to the disease. Data regarding the relationship between alcohol dose and the proportion of diabetics are not that clear, and lowest rates of diabetes are usually seen among light to moderate drinkers [3, 4, 23]. Our findings, similarly to a Norwegian study [28], show that the likelihood of diabetes was lowest with moderate alcohol consumption. Similar results were obtained in a metaanalysis of 13 epidemiological studies [29] in which alcohol intake of 5–30 g per day was associated with a 28% lower risk of diabetes compared to light drinking and abstinence.

Reference group

In our study, we chose subjects reporting alcohol intake of up to 15.0 g per day and not complete abstainers as the reference group, as the abstainer group might have included subjects abstaining for life, subjects who abused alcohol in the past but developed diseases necessitating abstinence,

Table 3. Likelihood of abnormal levels of cardiovascular disease risk factors in relation to the level of alcohol consumption.

Variable	Ethanol consumption [g/day]			
	Abstainers[1]	Light drinkers (0.01–15.00)	Moderate drinkers (15.01–30.00)	Heavy drinkers (> 30.00)
Hypertension[2]:				
Model 1	0.88 (0.73–1.06)	1	1.33 (1.13–1.55)	1.45 (1.19–1.77)
Model 2	0.90 (0.73–1.11)	1	1.37 (1.15–1.63)	1.52 (1.21–1.90)
HDL cholesterol < 1 mmol/L:				
Model 1	1.22 (0.97–1.53)	1	0.65 (0.52–0.82)	0.62 (0.46–0.85)
Model 2	1.22 (0.94–1.57)	1	0.60 (0.47–0.77)	0.56 (0.39–0.78)
LDL cholesterol ≥ 3 mmol/L:				
Model 1	0.80 (0.66–0.97)	1	1.06 (0.90–1.24)	0.88 (0.72–1.08)
Model 2	0.81 (0.66–1.00)	1	0.99 (0.84–1.17)	0.82 (0.66–1.03)
Triglycerides > 1.7 mmol/L:				
Model 1	0.87 (0.72–1.06)	1	1.24 (1.06–1.45)	1.44 (1.18–1.76)
Model 2	0.84 (0.67–1.04)	1	1.25 (1.05–1.48)	1.46 (1.17–1.83)
Homocysteine ≥ 12 μmol/L:				
Model 1	1.36 (1.04–1.78)	1	0.95 (0.73–1.22)	1.90 (1.42–2.53)
Model 2	1.19 (0.89–1.60)	1	0.95 (0.72–1.26)	1.95 (1.42–2.66)
Diabetes[3]:				
Model 1	1.11 (0.83–1.49)	1	0.67 (0.47–0.97)	1.11 (0.75–1.64)
Model 2	1.07 (0.77–1.47)	1	0.65 (0.44–0.97)	1.25 (0.83–1.89)

[1]Subjects reporting no alcohol intake during the last year before the study; [2]Defined as systolic blood pressure ≥ 140 mm Hg and/or diastolic blood pressure ≥ 90 mm Hg or antihypertensive drug treatment; [3]Defined as fasting blood glucose > 7 mmol/L and/or previous diagnosis of diabetes; Model 1 — adjusted for age; Model 2 — adjusted for age, education, net family income per person, body mass index, smoking status, and physical activity; HDL — high-density lipoprotein; LDL — low-density lipoprotein

and subjects who stopped drinking moderate amounts of alcohol due to age, poor health status, or medications. For these reasons, abstainers more often show adverse levels of CVD risk factors [27] and 2 separate metaanalyses showed that a protective effect of alcohol disappeared when subjects who stopped drinking or reported some occasional alcohol consumption were excluded from the abstainer group [30, 31]. Also in our study, the abstainer group was the oldest and characterised by a highest proportion of former smokers which may indicate that some men stopped drinking alcohol for health reasons. The proportion of hypertensives was also the highest in this group although mean systolic and diastolic BP was low. This resulted from the fact that proportion of subjects taking antihypertensive medications was highest in this group (27% among abstainers compared to 7–14% in the remaining groups) which qualified them as hypertensives. In addition, abstinence may be inconsistently reported, as showed in a U.S. cohort study with 3 serial evaluations, in which 53% of subjects reporting lifelong abstinence had in fact reported some alcohol intake in a previous questionnaire [32]. For these reasons, we believed that choosing the abstainer group as the reference group in our study might result in unreliable assessment of the effect of alcohol intake on CVD risk factors among drinkers.

Limitations of the study

Information regarding alcohol dose was obtained from questionnaires in which respondents reported the frequency and amounts of consumed alcoholic beverages, which might have led to errors in quantification of alcohol intake. As alcohol abuse is widely criticised, some respondents might have chosen not to disclose alcohol consumption or underreport its intake. In addition, preferences regarding types of alcoholic beverages and their amounts consumed correlate with socioeconomic and lifestyle factors such as smoking and physical activity which also affect CVD risk factors. We attempted to eliminate bias related to some of these factors (which were also measured in the WOBASZ study) by statistical adjustments and inclusion of these variables into the logistic regression model.

In summary, moderate alcohol consumption was found to have a negative effect on BP and triglyceride levels in a population of Polish men, and higher alcohol consumption also adversely affected homocysteine levels. Positive effects of alcohol consumption included a beneficial effect on HDL-C level, and with low intakes also a lower likelihood of diabetes.

If we take into account other negative effects of alcohol consumption [7, 33, 34] including addiction potential and an increased risk of numerous neoplasms, stroke, hepatitis, and pancreatitis [2], the issue whether any potential benefits

related to protective effects of alcohol on the cardiovascular system outweigh its other negative health effects calls for a critical reassessment.

CONCLUSIONS

In the Polish population, negative consequences of alcohol intake were seen among men who consumed more than 15 g of ethanol daily. A potential positive effect of alcohol consumption, as manifested by higher HDL-C level a lower likelihood of diabetes (only with alcohol intake of 15–30 g per day), was counterbalanced by a negative effect on BP, homocysteinaemia, and triglycerides.

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Spożycie alkoholu a profil czynników ryzyka chorób sercowo-naczyniowych wśród mężczyzn uczestniczących w badaniu WOBASZ

Anna Waśkiewicz, Elżbieta Sygnowska

Zakład Epidemiologii, Prewencji Chorób Układu Krążenia i Promocji Zdrowia, Instytut Kardiologii, Warszawa

Streszczenie

Wstęp: Alkohol wpływa na układ sercowo-naczyniowy poprzez modyfikację czynników ryzyka: stężenia lipidów, ciśnienia tętniczego, homocysteiny, cukrzycy oraz procesów krzepnięcia. W zależności od analizowanego czynnika i dawki alkoholu może to być negatywne lub pozytywne oddziaływanie.

Cel: Celem pracy była ocena profilu czynników ryzyka chorób sercowo-naczyniowych (CVD) w dorosłej populacji mężczyzn o zróżnicowanym spożyciu alkoholu.

Metody: W ramach Wieloośrodkowego Ogólnopolskiego Badania Stanu Zdrowia Ludności (WOBASZ) przebadano w latach 2003–2005 reprezentatywną próbę mieszkańców całej Polski w wieku 20–74 lat. U wszystkich respondentów, na podstawie badań ankietowych, laboratoryjnych, antropometrycznych i pomiaru ciśnienia krwi, określono szeroki zakres czynników ryzyka CVD. W niniejszej pracy wykorzystano dane 6912 mężczyzn, a w przypadku analiz dotyczących homocysteiny — 3359 mężczyzn. Konsumpcję piwa, wina i wódki oszacowano na podstawie wystandaryzowanego kwestionariusza i przeliczono na dzienne spożycie czystego etanolu. Badanych podzielono na 4 grupy: A — abstynenci oraz 3 grupy w zależności od ilości spożytego etanolu: N — niskie spożycie (≤ 15 g), S — średnie spożycie (15–30 g), W — wysokie spożycie (> 30 g etanolu na dzień). Za referencyjną uznano grupę mężczyzn o niskiej konsumpcji alkoholu (N).

Wyniki: W populacji polskiej udział grup mężczyzn w zależności od ilości spożywanego alkoholu przedstawiał się następująco — A: 8%, N: 72%, S: 13%, W: 7%. We wszystkich analizowanych grupach podstawowym źródłem etanolu było piwo, które dostarczało ponad 50% tego składnika, a następnie wódka — ok. 30%. Wielkość spożycia alkoholu różnicowała poziom niektórych czynników ryzyka CVD. Zanotowano dodatni związek między konsumpcją alkoholu a średnią wartością ciśnienia skurczowego (A: 134,0; N: 136,9; S: 139,7; W: 141,3 mm Hg) i rozkurczowego (odpowiednio 81,1; 83,3; 85,9; 87,1 mm Hg), cholesterolu frakcji HDL (1,25; 1,34; 1,45; 1,61 mmol/l) i triglicerydów (1,59; 1,63; 1,82; 2,00 mmol/l). Wśród abstynentów w odniesieniu do pozostałych grup zaobserwowano najwyższy udział mężczyzn z nadciśnieniem tętniczym (45%), nadwagą i otyłością (64%), cukrzycą (12,2%) i byłych palaczy (36%). Ze wzrostem dawki alkoholu notowano wzrost odsetka osób z nadciśnieniem tętniczym (N: 39%; S: 41%; W: 44%), z hipertriglicerydemią (N: 32%; S: 36%; W: 39%), z hiperhomocysteinemią (N: 26%; S: 22%; W: 38%) oraz osób palących tytoń (N: 37%; S: 48%; W: 62%), natomiast spadek udziału osób z niskim stężeniem cholesterolu frakcji HDL (N: 16%; S: 11%; W: 11%) oraz z nadwagą i otyłością (N: 61%; S: 60%; W: 55%). Po skorygowaniu o zmienne zakłócające (wiek, wykształcenie, dochód na osobę w rodzinie, wskaźnik masy ciała, status palenia tytoniu, aktywność fizyczna) spożycie alkoholu wpływało na wystąpienie nieprawidłowych wartości analizowanych czynników ryzyka CVD. W porównaniu z grupą referencyjną (N), grupa S charakteryzowała się o 37% wyższym ryzykiem wystąpienia nadciśnienia tętniczego i o 25% wyższym ryzykiem podwyższonego stężenia triglicerydów, a o 40% niższym ryzykiem wystąpienia niskiego stężenia cholesterolu frakcji HDL i o 35% niższym ryzykiem cukrzycy. W przypadku grupy o wysokiej konsumpcji alkoholu (> 30 g etanolu) ryzyko nadciśnienia tętniczego było wyższe o 52%, hipertriglicerydemii o 46%, hiperhomocysteinemii o 95%, natomiast o 44% mniejsze było ryzyko niskiego cholesterolu frakcji HDL. Ryzyko wystąpienia podwyższonego stężenia cholesterolu frakcji LDL nie wiązało się z dawką alkoholu. U abstynentów ryzyko pojawienia się nieprawidłowych wartości analizowanych czynników nie różniło się istotnie od grupy referencyjnej.

Wnioski: W populacji polskiej negatywne skutki działania alkoholu wystąpiły w grupie mężczyzn spożywających powyżej 15 g etanolu dziennie. Potencjalnie korzystny efekt konsumpcji alkoholu związany z podwyższaniem stężenia HDL i obniżaniem ryzyka wystąpienia cukrzycy (tylko przy dawce 15–30 g etanolu dziennie) był niwelowany przez jego niekorzystny wpływ na wartość ciśnienia tętniczego, stężenia homocysteiny i triglicerydów.

Słowa kluczowe: spożycie alkoholu, ciśnienie tętnicze, cholesterol frakcji HDL, triglicerydy, homocysteina, cukrzyca, populacja polska

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Adres do korespondencji:
dr n. roln. Anna Waśkiewicz, Zakład Epidemiologii, Prewencji Chorób Układu Krążenia i Promocji Zdrowia, Instytut Kardiologii, ul. Alpejska 42, 04–628 Warszawa, tel: +48 22 815 65 56, e-mail: awaskiewicz@ikard.pl
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