Association of smoking with indicators of the structure and function of left ventricle of the heart in middle-aged men

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Aim. Evaluation of the association of smoking (status, intensity and duration) with indicators of the structure and function of the left ventricle of the heart in a sample of middle-aged men.

Material and methods. This study is part of a 32-year prospective cohort observation of men from childhood (11-12 years). 301 (30.0%) representatives of the original population sample aged 41-44 years were included in the study. The examination included a survey on intensity of smoking, anthropometry, measuring blood pressure, pulse rate, echocardiography, and blood lipid analysis.

Results. 301 men aged 41-44 included 92 (30.6%) men who had never smoked, 73 (24.3%) men smoked in the past and 136 (45.2%) men currently smoke. 75% of current smokers started smoking before age 19, of which 32.3% started smoking before age 15. The duration of smoking cessation among former smokers was 14.4 (12.5; 16.2) years. The average duration of smoking [average (95% confidence interval)] among former smokers was 14.4 (12.5; 16.2), for current smokers – 25.3 (24.6; 26.0) years. Current smoking was statistically significantly associated with higher mean values of the left ventricular myocardium mass (LVMM), the left ventricular myocardial mass index (LVMMI), the end-systolic and end-diastolic interventricular septum thickness (IVSTs/IVSTd), the end-systolic left ventricular posterior wall thickness (LVPWs), and the intensity and duration of current smoking were associated with higher values of the relative wall thickness of the left ventricle, the end-diastolic interventricular septum thickness, the end-systolic interventricular septum thickness, and with low values of the left ventricular stroke volume index (LV SVI). Multiple regression analysis showed that current smoking has an independent effect on the left ventricular myocardium mass, the left ventricular myocardial mass index and the end-diastolic interventricular septum thickness, and the duration and intensity of smoking has an effect on the index of the left ventricular stroke volume index.

Conclusion. Current smoking, duration and intensity in middle-aged men is associated with unfavorable changes in indicators of the structure and function of the left ventricle of the heart. Efforts for primary prevention of smoking should begin as early as childhood and continue into adolescence and young adulthood.

Key words: smoking, smoking index, left ventricle, myocardial function, men.

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Introduction

Smoking remains a pressing public health problem around the world and a leading cause of death that we could prevent. According to a report by the World Health Organization, 6 million people die annually from tobacco-related diseases, and this figure will increase to 10 million in the world by 2030 [1]. Tobacco smoke is one of the most significant risk factors for cardiovascular disease, including myocardial infarction, stroke, peripheral arterial disease and heart failure [2,3]. Smoking is also the second most common cause of cardiovascular disease after high blood pressure [4]. Large epidemiological studies on the effect of cardiac risk factors on the heart structure and function indicate the association of tobacco smoking with an increase in the left ventricular myocardium mass (LVMM) and a decrease in its systolic function [5-7]. But some studies have shown opposite results [8,9]. Cigarette smoking and tobacco use have generally been studied as a binary (yes/no) covariate, and few scientists have assessed the duration and intensity of smoking and their relationship to heart structure and function through echocardiography. Scientists hypothesized that a longer duration of smoking (starting at a younger age), a high smoking intensity (more daily cigarettes smoked) and an indicator of the cumulative duration and intensity of smoking are associated with the worst parameters of the heart structure and function [10]. The inconsistent and even contradictory results of previously published studies on the association of smoking with structural and functional disorders of the heart indicate the need for further study of this problem.

The aim of the present study is to assess of the association of smoking (status, intensity and duration) with indicators of the structure and function of the left ventricle of the heart in a sample of middle-aged men.

Material and methods

This study is part of a 32-year prospective cohort observation of men from childhood (11-12 years). After 32 years, 301 (30%) representatives of the original population sample were examinated for the continuation of this study. The average age of the surveyed men was 42.9 (41.0; 44.0) years. The examination included a survey on a standard questionnaire, measuring weight and height, measuring waist and hips, measuring blood pressure, pulse rate. The examination assessed levels of total cholesterol, high density lipoprotein cholesterol and triglycerides. Smoking status was assessed using a self-completed questionnaire. Smokers were defined as those who smoked one or more cigarettes per day. All study participants were divided according to their smoking status: 1st group - non-smokers and never smokers; 2nd group – former smokers (men who smoked in the past); 3rd group – current smokers (men who regularly smoke at present). Those who quit smoking were defined as those who smoking at least 1 year ago. The cumulative intensity and duration of smoking (cumulative effect of smoking) was assessed by the smoking person index (SPI), which was calculated using the formula:

SPI (pack of cigarettes/years)=number of cigarettes smoked per day × smoking experience (years)/20.

Four groups were formed: the first group included non-smokers and never smokers; the second group is the first tertile of the SPI (⩽14 packs/years); the third group is the second tertile of the SPI (15-27 packs/years); the fourth group – the third tertile of the SPI (⩾28 packs/years).

Ultrasound examination of the heart was performed on a SIM 5000 plus echocardiograph (Co. I, Japan) by the echo-pulse method in one- and two-dimensional examination modes with a transducer frequency of 2.7-3.5 MHz according to a generally recognized technique. The morphometric parameters were assessed – left atrial diameter, end-systolic and end-diastolic size (EDS) of the left ventricle, interventricular septum thickness (IVST) and left ventricular posterior wall thickness (LVPW) in diastole. LVMM was calculated using the formula of R. Devereux [11]: LVMM=1.04 × [(IVST+LVPW+EDS) 3 - (EDS) 3] – 13.6. The LVMM index was determined, that is, the ratio of the left ventricular myocardium mass to the body surface area. The relative wall thickness (RWT) of the myocardium was calculated using the formula: RWT=(IVST+LVPW)/EDS. The systolic function of the left ventricular myocardium was assessed according to the following parameters: end-diastolic volume and end-systolic volume of the left ventricle, ejection fraction, the degree of shortening of the anterior-posterior size of the left ventricle in systole, and stroke volume index as the ratio of the stroke volume to the body surface.

Statistical analysis

Statistical data processing was performed using SAS 9.0 software and IBM SPSS Statistics v.23. Descriptive statistics presented in tables and figures have the following designations: n – the absolute number of persons in the group; % – the share of persons from their total number in the group; M – arithmetic mean, 95% confidence interval. Descriptive statistics, histograms of residuals, and graphs of normal probability (Q-Q-plots) were used to check the normal

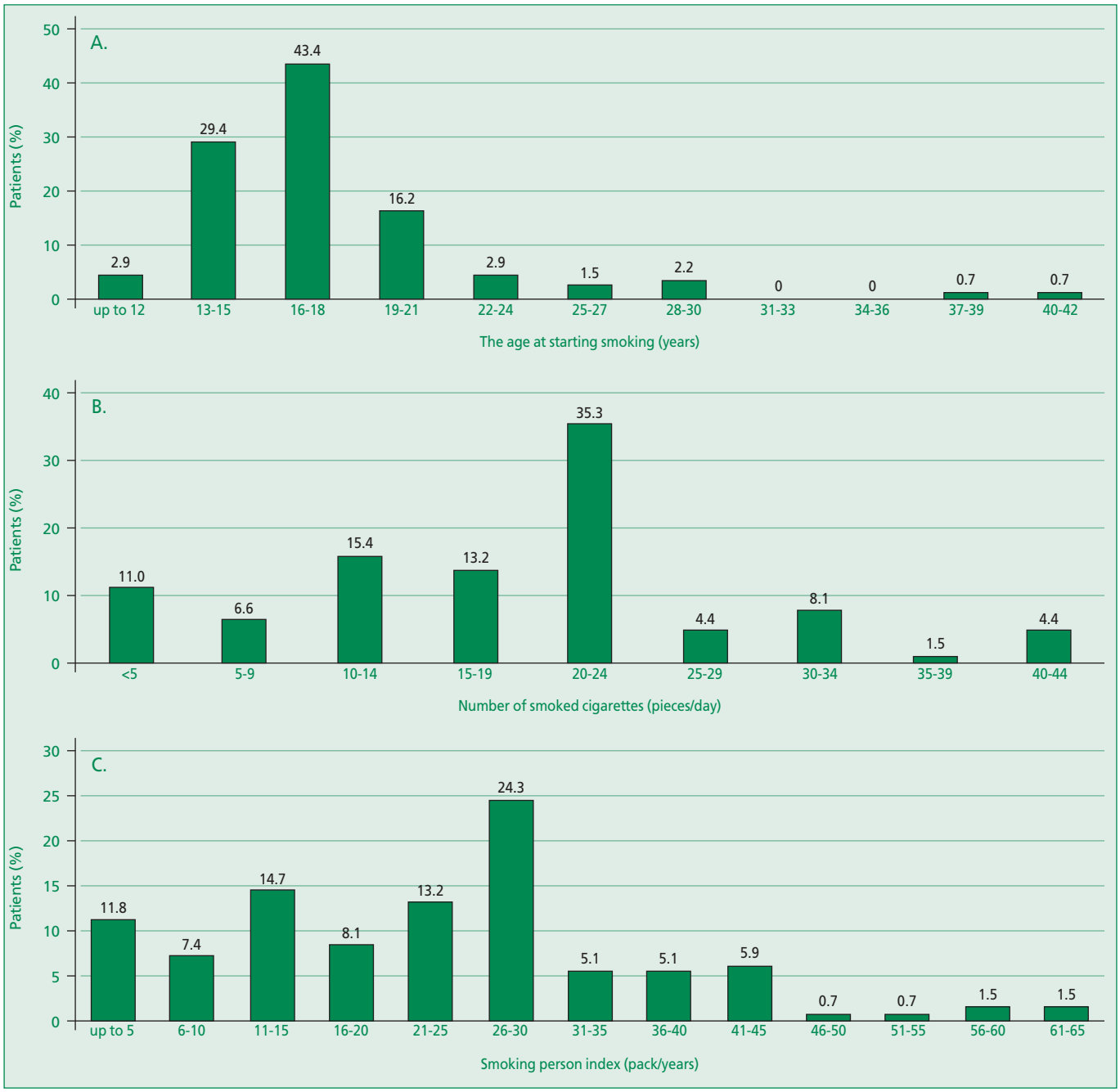

Figure 1. Distribution of current smokers by the age at starting smoking (A), daily number of cigarettes smoked (B), smoking person index (C)

distribution of quantitative variables. The homogeneity of the dispersions was checked using the Levene's test. Statistical comparison of means was performed using the two-sided Student's t-test for independent samples. The one-way analysis of variance (ANOVA) was used to check for a linear trend between the ordered categories of the factor variable and the dependent continuous variables. Post hoc comparisons were performed using a multiple t-test without alpha correction (LSD – Least Significant Difference test). Comparison of several groups with nominal data was performed using the Pearson's chi-square test (χ^2), followed by pairwise comparisons of proportions using the Z-test. The association of smoking status, smoking intensity and duration (cumulative effect of smoking) with structural and functional indicators of the left ventricle was assessed using simple (univariate) and multiple linear regression analyzes. Categorical predictors were converted to dummy variables with numeric labels 0 and 1. Continuous variables with skewed distributions were logarithmically transformed prior to regression analysis. The relationship between smoking and indicators of the structure and function of the left ventricle of the heart was assessed

taking into account the influence of confounders. Variance Inflation Factor (VIF) was used to test collinearity. The critical level of statistical significance was taken as p <0.05.

Results

The 301 men aged 41-44 included 92 (30.6%) men who never smoked, 73 (24.3%) men who smoked in the past and 136 (45.2%) men who currently smoke. 75% of 136 current smokers started smoking before age 19 (Fig. 1), of which 32.3% started smoking before age 15. The duration of smoking cessation among former smokers was 14.4 (12.5; 16.2) years. The average duration of smoking among former smokers was 14.4 (12.5; 16.2), and the average duration of smoking among current smokers was 25.3 (24.6; 26.0) years.

However, more than half of male smokers (about 54.0%) are heavy smokers (Fig. 1), that is, they smoke 20 or more cigarettes daily. The SPI for 44.8% of current smokers (Fig. 1) is 26 or more (packs/years).

The one-way ANOVA that we used to assess the trend between the ordered categories of the factor variable (smoking status) and group means (Table 1) showed an increase in the mean values of waist and hip circumference, diastolic blood pressure, pulse rate, triglycerides, and a decrease in high-density lipoprotein cholesterol from a group of men who have never smoked to current smokers. The linear relationship is confirmed by statistically significant weighted Fisher's F-tests. Pairwise comparison found that current smokers (group 3) had significantly higher mean values of waist and hip circumference, diastolic blood pressure, pulse rate and triglycerides, and the value of high-density lipoprotein cholesterol was lower compared with never smokers (group 1) but these values didn't differ significantly between current and former smokers. No statistically significant trend was found between group order and categorical variables on the χ^2 test. Current smokers compared with nonsmoking peers noted a trend towards a higher frequency of hypertension (p=0.06) and a lower adherence to hypertension treatment compared with other categories of smoking status.

A similar linear trend was observed (Table 2) between the ordered categories of smoking status and the mean values of LV morphometric parameters in middle-aged men. It showed an increase in the average values of LVMM, LVMMI, IVSTs, IVSTd, LVPWs, LVPWd from the group of never smokers men to current smokers. The linear trend is confirmed by statistically significant weighted Fisher's F-tests. The mean values of LVMM, LVMMI, IVSTs, LVPWs, LVPWd were significantly higher in current smokers compared with never smokers peers, but they didn't differ significantly between current and former smokers, with the exception of LVMMI, which was lower in former smokers.

The results of the one-way ANOVA (Table 3) also revealed a direct linear relationship between the ordered categories of the SPI (cumulative intensity and duration of smoking) of current smokers and morphometric indicators of the left ventricle (relative wall thickness of the LV, IVSTs, IVSTd, LVPWs, LVPWd), as well as inverse the relationship between the ordered

Table 1. Baseline indicators in middle-aged men depending on smoking status

Indicators	Total (n=301)	Group 1 (n=92)	Group 2 (n=73)	Group 3 (n=136)	p[a]
Age, years	42.9 (42.9; 43.0)	42.9 (42.8; 43.0)	42.9 (42.8; 43.1)	42.9 (42.9; 43.0)	0.579
Body mass index, kg/m^2	27.7 (27.1; 28.2)	26.8 (25.9; 27.8)	28.6 (27.5; 29.7)	27.7 (26.9; 28.5)	0.259
Waist circumference/ Hip circumference	0.93 (0.92; 0.94)	0.91 (0.90; 0.93)	0.94 (0.92; 0.96)	0.94 (0.93; 0.95) *	0.036
Systolic blood pressure, mm Hg	122 (120; 124)	120 (117; 123)	122 (119; 125)	124 (121; 126)	0.061
Diastolic blood pressure, mm Hg	82 (81; 84)	80 (78; 83)	82 (80; 85)	84 (82; 86) *	0.036
Pulse rate, beats/min	74 (73; 75)	73 (71; 75)	75 (72; 77)	75 (74; 77) *	0.052
Total cholesterol, mmol/l	5.7 (5.6; 5.9)	5.6 (5.4; 5.8)	5.8 (5.6; 6.1)	5.7 (5.5; 6.0)	0.472
High-density lipoprotein cholesterol, mmol/l	1.0 (0.96; 1.04)	1.07 (1.0; 1.13)	1.01 (0.93; 1.08)	0.95 (0.89; 1.0) **	0.006
Triglycerides, mmol/l	1.44 (1.34; 1.55)	1.19 (1.04; 1.34)	1.43 (1.26; 1.61) *	1.62 (1.43; 1.8) ***	0.001
Alcohol, g/week	130.5 (104.7; 156.2)	128.5 (70.4; 186.7)	103.5 (69.3; 137.7)	146.2 (108.7; 183.8)	0.492
Hypertension, n (%) of which: Antihypertensive therapy, n (%)	114 (37.9) 81 (71.1)	28 (30.4) 20 (71.4)	20 (38.4) 24 (85.7)	58 (42.7) 37 (63.8)	0.175 0.110

Data are presented as M (95% CI)

[a] test for continuous variables and χ^2 test for categorical variables

*p⩽0.05, **p⩽0.01, ***p⩽0.001 compared to those who have never smoked; †p⩽0.05 compared to those who smoked in the past

Table 2. Structural and functional indicators of the left ventricle in middle-aged men depending on smoking status

Indicators	Group 1 (n=92)	Group 2 (n=73)	Group 3 (n=136)	p (ANOVA) [a]
LVMM, g	165.7 (158.9; 172.4)	171.0 (164.7; 177.4)	176.8 (171.1; 182.4)**	0.009
LVMMI	80.9 (78.4; 83.3)	80.4 (78.3; 82.6)	84.8 (82.5; 87.0)*††	0.011
Relative wall thickness of the left ventricle	0.35 (0.34; 0.36)	0.36 (0.35; 0.37)	0.36 (0.35; 0.37)	0.088
IVSTd, cm	0.93 (0.91; 0.95)	0.96 (0.93; 0.98)	0.96 (0.94; 0.99)	0.039
IVSTs, cm	1.25(1.21; 1.28)	1.29 (1.25; 1.33)	1.31 (1.28; 1.34)**	0.013
LVPWd, cm	0.86 (0.84; 0.88)	0.88 (0.86; 0.90)	0.90 (0.88; 0.93)**	0.005
LVPWs, cm	1.53 (1.50; 1.56)	1.58 (1.55; 1.62)*	1.58 (1.55; 1.61)**	0.016
Left ventricular end-diastolic volume index	61.8 (59.9; 63.6)	60.3 (58.1; 62.6)	62.0 (60.2; 63.8)	0.662
left ventricular end-systolic volume index	21.0 (20.0; 22.0)	20.5 (18.7; 22.3)	21.4 (20.5; 22.4)	0.456
LV VMI	0.77 (0.75; 0.79)	0.75 (0.73; 0.78)	0.74 (0.72; 0.76)	0.054
LV SVI	40.8 (39.5; 42.1)	39.8 (38.0; 41.7)	40.4 (39.1; 41.8)	0.918
Left ventricular shortening fraction, %	36.80 (36.0; 37.6)	37.2 (35.6; 38.8)	36.6 (35.8; 37.4)	0.581
Left ventricular ejection fraction, %	66.2 (65.1; 67.2)	67.3 (66.2; 68.4)	65.9 (65.0; 66.9)	0.618
Left atrial diameter. cm	3.8 (3.8; 4.0)	4.0 (3.9; 4.1)	3.9 (3.9; 4.0)	0.381

Data are presented as M (95% CI)

atest for continuous variables with weighting of intergroup sums of squares by group size

*p⩽0,05, **p⩽0,01 compared to those who have never smoked

†p⩽0,05, ††p⩽0,01 compared to those who smoked in the past

LVMM – left ventricular myocardium mass, LVMMI – left ventricular myocardial mass index, IVSTd – end-diastolic interventricular septum thickness, IVSTs –end-systolic interventricular septum thickness, LVPWd – end- diastolic left ventricular posterior wall thickness, LVPWs – end-systolic left ventricular posterior wall thickness, LV VMI – volume/mass index of the left ventricular, LV SVI – left ventricular stroke volume index

categories (groups) of the SPI and the indicators of LV function (LV SVI and LV MMI), which is confirmed by statistically significant weighted Fisher's F-tests for a linear trend. The indicators of the relative wall thickness of the LV, IVSTs, IVSTd, LVPWs, LVPWd increased as the ordered categories (groups) of the SPI increased, and were minimal in the group of men who never smoked, and maximum in groups 3 and 4, whose the SPI, respectively, were ⩾15 and 28 packs/years (Table 3). But LV SVI and LV MMI decreased with an increase in the ordered categories (groups) of the SPI.

Consequently, the intensity and duration of current smoking are associated with higher values of the relative wall thicknesses of the LV, IVST and LVPWs, and with lower values of LV SVI.

Simple (univariate) and multiple (multivariate) linear regression analyzes were performed to quantify the association between smoking status, smoking intensity and duration, on the one hand, and echocardiographic parameters characterizing the structure and function of the left ventricle, on the other. Table 4 presents abbreviated models of linear regression analysis of the association between smoking status and indicators of the structure (morphometric indicators) of the LV. In a simple (univariate) linear regression analysis (Table 4), current smokers showed significantly higher rates of LVMMI, LVMMI, IVSTd, LVPWs and LVPWd by the value corresponding to the values of the regression coefficients for the specified explained (dependent) variables, compared with never smokers peers. And ex-smokers showed only higher values of LVPWs. Multiple linear regression analysis was performed to exclude the effect of the confounding factors on the relationship between smoking (past and current) and the LV morphometric indicators. Therefore, additional explanatory variables (body mass index, diastolic blood pressure, pulse rate, high-density lipoprotein cholesterol, triglycerides, and antihypertensive drugs) were included in the regression model. The results of multiple regression analysis showed (Table 4) that current smoking has an independent effect on LVMM, LVMMI and LVPWd also after correcting for the above confounders. However, a statistically significant association between past smoking and LVPWs found in univariate regression analysis disappeared after correcting for the confounders.

Table 5 shows an abbreviated linear regression analysis of the association between SPI (cumulative intensity and duration of current smoking) and left ventricular systolic function – SVI.

Those who were in the second (15-27 packs/years) and third (⩾28 packs/years) tertiles, according to the results of univariate analysis, had significantly lower SVI by the value corresponding to

Table 3. Structural and functional indicators of the left ventricle in middle-aged men, depending on the cumulative intensity and duration of smoking at present

Indicators	Group 1 (n=92)	Group 2 (n=45)	Group 3 (n=46)	Group 4 (n=45)	p (ANOVA)[a]
LVMM, g	165.7 (158.9; 172.4)	179.7 (168.6; 190.8)	177.6 (167.8; 187.4)	172.9 (163.9; 182.0)	0.116
LVMMI	80.9 (78.4; 83.3)	86.1 (81.5; 90.7)	84.7 (80.9; 88.4)	83.4 (79.9; 86.9)	0.175
Relative wall thickness of the left ventricle	0.35 (0.34; 0.37)	0.35 (0.33; 0.36)	0.37 (0.35; 0.39) *†	0.37 (0.35; 0.39) *†	0.012
IVSTd, cm	0.93 (0.91; 0.95)	0.94 (0.91; 0.98)	0.96 (0.93; 1.00)	0.99 (0.95; 1.02) **	0.008
IVSTs, cm	1.25 (1.21; 1.28)	1.29 (1.23; 1.34)	1.32 (1.27; 1.36) *	1.32 (1.27; 1.37) *	0.010
LVPWd, cm	0.86 (0.84; 0.88)	0.89 (0.85; 0.92)	0.93 (0.89; 0.97) *	0.89 (0.86; 0.93)	0.022
LVPWs, cm	1.53 (1.50; 1.56)	1.58 (1.53; 1.62)	1.59 (1.53; 1.65) *	1.58 (1.53; 1.63)	0.032
Left ventricular end-diastolic volume index	61.8 (59.9; 63.6)	65.2 (62.3; 68.2)	60.8 (57.9; 63.7)	60.0 (56.5; 63.5)	0.229
left ventricular end-systolic volume index	21.0 (20.0; 22.0)	21.7 (20.2; 23.2)	20.7 (19.0; 22.5)	21.8 (20.1; 23.6)	0.563
LV VMI	0.77 (0.75; 0.79)	0.77 (0.73; 0.81)	0.73 (0.69; 0.77) †	0.72 (0.68; 0.76) †	0.011
LV SVI	40.8 (39.5; 42.1)	43.5 (41.3; 45.8) *	39.6 (37.8; 41.5) ††	38.2 (35.5; 40.9) *†††	0.027
Left ventricular shortening fraction, %	36.8 (36.0; 37.6)	37.5 (36.1; 38.8)	36.7 (35.3; 38.2)	35.6 (34.2; 36.9)	0.150
Left ventricular ejection fraction, %	66.2 (65.1; 67.2)	66.5 (64.9; 68.2)	66.0 (64.1; 68.0)	65.2 (63.7; 66.)	0.370
Left atrial diameter, cm	3.9 (3.8; 4.0)	3.9 (3.8; 4.0)	3.9 (3.8; 4.0)	4.0 (3.8; 4.1)	0.190

Data are presented as M (95% CI)

1st group – don't smoke and have never smoked; 2nd group – ⩽14 packs/years; 3rd group – 15-27 packs/years; 4th group – ⩾28 packs/years

atest for continuous variables with weighting of intergroup sums of squares by group size

*p <0,05, **p <0,01, ***p <0,001 compared to group 1; †p <0,05 compared to group 2

LVMM – left ventricular myocardium mass, LVMMI – left ventricular myocardial mass index, IVSTd – end-diastolic interventricular septum thickness, IVSTs –end-systolic interventricular septum thickness, LVPWd – end- diastolic left ventricular posterior wall thickness, LVPWs – end-systolic left ventricular posterior wall thickness, LV VMI – volume/mass index of the left ventricular, LV SVI – left ventricular stroke volume index

the values of the regression coefficients for the specified explained (dependent) variables, compared with those who were in the first tertile (⩽14 packs/years). The relationship between cumulative intensity and duration of current smoking and SVI remained significant in the fully adjusted model.

Thus, the results of multiple regression analysis showed that current smoking in middle-aged men is directly related to morphometric indicators of LV myocardial hypertrophy, regardless of other interfering factors. And the intensity and duration of current smoking, its cumulative effect is directly related to a decrease in LV systolic function, regardless of traditional markers of cardiovascular disease risk. The structural and functional LV indicators in middle-aged men who quit smoking were similar to those in their peers who had never smoked.

Discussion

Our study is part of a 32-year prospective cohort observation of men starting in childhood. After 32 years, 301 representatives of the original population sample aged 41-44 years were examined for the continuation of this study. We found that most men have either smoked in the past or are currently smokers. Moreover, more than half of male smokers are heavy smokers, that is, they smoke 20 or more cigarettes daily.

The effect of smoking on the progression of atherosclerosis has been the most studied. Recent evidence suggests that smoking is associated with high blood pressure and increased vascular stiffness, suggesting hemodynamic and neurohumoral mechanisms of myocardial damage. The results of our study showed the presence of an association between smoking and abdominal obesity, atherogenic changes in the lipid spectrum of the blood, and a tendency towards a higher incidence of hypertension.

Contradictory data on the association of smoking with morphofunctional changes in the heart still exist despite the evidence for the effects of tobacco smoke on myocardial remodeling. The studies were carried out mainly among elderly patients with proven cardiovascular disease. In a study by W. Nadruz et al. [5] an examination of 4580 healthy elderly people using transthoracic echocardiography showed that active smoking is associated with changes in the LV structure and function. Our data are fully consistent with the results of the study by W. Nadruz et al. This study found that active smoking by current smokers was associated with higher LVMM and LVMMI values compared with never-smokers and former smokers. In addition, the SPI and smoking duration were associated with higher LVMMI and LV MMI indicators. These data indicate

Table 4. Linear regression analysis of the association between smoking status and structure indicators (morphometric indicators) of the left ventricle

Smoking status	Univariate analysis[a]		Multivariate analysis[b]	
	B (95% CI)	p	B (95% CI)	p
LVMM				
Those who don't smoke and have never smoked	referent category			
Former smokers	5.358 (-4.064; 14.779)	0.263	-0.927 (-8.107; 6.253)	0.799
Current smokers	11.107 (2.324-19.891)	0.013	8.669 (1.162; 16.175)	0.024
LVMMI				
Those who don't smoke and have never smoked	referent category			
Former smokers	-0.441 (-3.795; 2.914)	0.795	-1.651 (-4.814; 1.512)	0.304
Current smokers	3.879 (0.509; 7.249)	0.024	3.784 (0.457; 7.112)	0.026
IVSTd				
Those who don't smoke and have never smoked	referent category			
Former smokers	0.022 (-0.008; 0.053)	0.148	0.0008 (-0.026; 0.028)	0.951
Current smokers	0.031 (0.001; 0.061)	0.044	0.016 (-0.010; 0.042)	0.240
LVPWs				
Those who don't smoke and have never smoked	referent category			
Former smokers	0.057 (0.008; 0.105)	0.022	0.035 (-0.010; 0.080)	0.131
Current smokers	0.057 (0.012; 0.101)	0.012	0.038 (-0.003; 0.079)	0.072
LVPWd				
Those who don't smoke and have never smoked	referent category			
Former smokers	0.016 (-0.014; 0.047)	0.292	-0.004 (-0.031; 0.023)	0.750
Current smokers	0.042 (0.010; 0.073)	0.009	0.030 (0.001; 0.059)	0.041

[a] regression coefficients with the achieved level of statistical significance based on the results of simple linear regression analysis.

[b] regression coefficients with the achieved level of statistical significance based on the results of multiple linear regression analysis corrected for body mass index, diastolic blood pressure, pulse, high-density lipoprotein cholesterol, triglycerides and taking antihypertensive drugs

B – regression coefficient, CI – confidence interval, LVMM – left ventricular myocardium mass, LVMMI – left ventricular myocardial mass index, IVSTd – end-diastolic interventricular septum thickness, LVPWd – end- diastolic left ventricular posterior wall thickness, LVPWs – end-systolic left ventricular posterior wall thickness

that active smoking and cumulative exposure to cigarettes were associated with changes in the LV structure. Elevated LVMM and the LV hypertrophy are recognized risk factors for heart failure [12]. In our study, LVMM and the prevalence of the LV hypertrophy were higher in current smokers compared to never-smokers and former smokers, although previous studies have shown conflicting results [12,13]. The present study is consistent with data from other large population-based studies [14,15], which showed greater LVMM in active smokers.

Our study included many young men, and the group of smokers was statistically significantly different from the group of nonsmokers in terms of indicators characterizing the LV hypertrophy. At the same time, the indicators of cardiac output didn't differ significantly, which may reflect the compensatory stage without the development of heart failure in people without significant cardiac pathology and indicate the relationship between smoking and the LV hypertrophy.

Long-term smoking is believed to be associated with significant metabolic and morphological changes in the heart muscle, which can be characterized as «tobacco cardiomyopathy». Significant changes in the functions of the right and left chambers of the heart have been noted, which leads to diastolic or systolic dysfunction [16].

The influence of the smoking intensity and duration on morphofunctional changes in the heart was evaluated in isolated studies. The ECHO cardiographic Study of Hispanics/Latinos [17] found a statistically significant association between smoking duration and higher mean LVMM and lower stroke volume in the right ventricle. Daily smoking of a large number of cigarettes was associated with a higher LVMM value, worsening diastolic function, an increase in the LV relative wall thickness, and a decrease in the stroke volume of the right ventricle. The combined measure of the smoking intensity and duration was associated with higher LVMM, poorer LV geometry, worse diastolic function, greater RV dilatation, and worsening RV function. We concluded that there is a relationship between the intensity and duration of smoking cigarette tobacco with unfavorable changes in the structure of the left and right parts of the heart,

Table 5. Linear regression analysis of the association between cumulative intensity with duration of smoking and an indicator of left ventricular systolic function - stroke volume index

Tertiles of the SPI	Univariate analysis[a]		Multivariate analysis[b]	
	B (95% CI)	p	B (95% CI)	p
⩽14 packs/years	referent category			
15-27 packs/years	-3.918 (-6.792; -1.045)	0.008	-3.490 (-6.322; -0.657)	0.016
⩾28 packs/years	-4.688 (-7.704; -1.672)	0.003	-3.295 (-6.223; -0.367)	0.028

[a] regression coefficients with the achieved level of statistical significance based on the results of simple linear regression analysis

[b] regression coefficients with the achieved level of statistical significance based on the results of multi-ple linear regression analysis corrected for body mass index, diastolic blood pressure, pulse, high-density lipoprotein cholesterol, triglycerides and taking antihypertensive drugs

SPI – smoking person index, B – regression coefficient, CI – confidence interval, SVI – stroke vol-ume index

as well as impairment of their function. Our work has shown that prolonged and intense smoking leads not only to hypertrophy, but also to LV systolic dysfunction with a decrease in the stroke volume index, which may indicate the toxic effect of tobacco smoke and the development of «tobacco cardiomyopathy». Our findings are consistent with the results of a recent study in a small sample of 49 young patients without cardiovascular disease, in which statistically significant signs of systolic and diastolic dysfunction in the smokers group were found on morphometric and functional LV assessment. These data suggest that smoking can directly affect heart function even without severe atherosclerosis or other chronic comorbidities associated with an increased risk of cardiovascular disease [18,19].

An important fact is the possible reversibility of morphofunctional changes in the heart upon smoking cessation. The only study found showed that elderly former smokers had similar echocardiographic characteristics to those who had never smoked [5]. The data of our study indicate the potential reversibility of echocardiographic changes in the heart upon smoking cessation, since the structural and functional indicators of the LV in middle-aged men who quit smoking were similar to those in their peers who had never smoked.

These data confirm that smoking can directly affect the structure and function of the left ventricle of the heart, even without atherosclerosis or other chronic comorbidities associated with an increased risk of cardiovascular disease.

Limitations

The main limitations of this study are the peculiarities of the composition of the sample by sex and age (only men 41-44 years old were studied).

Conclusion

Current smoking in middle-aged men is associated with left ventricular myocardial hypertrophy even after correction for confounding factors, and the intensity and duration of current smoking and its cumulative effect are associated with a decrease in left ventricular systolic function.

The structural and functional indicators of the left ventricle in middle-aged men who quit smoking were similar to those in their peers who had never smoked.

Further studies should be aimed at studying the effect of long-term smoking on the structure and function of the right heart, as the most susceptible to the direct and indirect negative effects of smoking.

Efforts for primary prevention of smoking should begin as early as childhood and continue into adolescence and young adulthood.

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