

## Prevalence of diseases and pathological conditions in young people under 45 years of age with abdominal obesity in Siberia

Ragino Yu.I., Khudyakova A.D., Striukova E.V., Denisova D.V.,  
Shcherbakova L.V.

*Research Institute of Internal and Preventive Medicine, Branch of the Institute of Cytology and Genetics,  
Siberian Branch of the Russian Academy of Sciences (IIPM – Branch of IC&G SB RAS)  
175/1, B. Bogatkova Str., Novosibirsk, 630089, Russian Federation*

### ABSTRACT

**Aim.** To study the prevalence of abdominal obesity in young people aged 25–44 years in Novosibirsk, as well as the prevalence of diseases and pathological conditions in individuals with abdominal obesity.

**Materials and methods.** We conducted a cross-sectional, population-based study of the population of Novosibirsk aged 25–44 years. The screening examined 1,415 people, including 670 men and 745 women. For all individuals, we evaluated the presence of such conditions as abdominal obesity (AO), arterial hypertension (AH), increased body mass index (BMI), coronary heart disease (according to validated epidemiologic and functional criteria with ECG findings classified according to the Minnesota Code), diabetes mellitus (DM), reduced glomerular filtration rate (GFR), chronic bronchitis (CB), increased blood levels of total cholesterol (hypercholesterolemia) and low-density lipoprotein (LDL) cholesterol (hyper-LDL-cholesterolemia).

**Results.** The prevalence of AO in the population of Novosibirsk aged 25–44 years was 42.4%: in men – 42.7%, in women – 42.1%. We found that AO had a significant direct effect on the development of AH (odds ratio (OR) = 2.550, 95% confidence interval (CI) 1.899–3.422,  $p = 0.0001$ ), CB (OR = 1.830, CI 1.326–2.527,  $p = 0.0001$ ), hypercholesterolemia (OR = 1.486, CI 1.193–1.851,  $p = 0.0001$ ), hyper-LDL-cholesterolemia (OR = 1.527, CI 1.222–1.907,  $p = 0.0001$ ) and a reverse effect on reduced GFR (OR = 0.603, CI 0.427–0.852,  $p = 0.004$ ). In the male population under 45 years of age, AO had a significant direct effect on the development of AH, CB, hypercholesterolemia, and hyper-LDL-cholesterolemia. In the female population under the age of 45, AO had a significant direct effect on the development of DM, AH, CB, and hyper-LDL-cholesterolemia and a reverse effect on the reduced GFR development.

**Conclusion.** Therefore, in the young Siberian population under 45 years of age, abdominal obesity is associated with the development of common diseases and pathological conditions.

**Key words:** abdominal obesity, population under 45 years of age, arterial hypertension, chronic bronchitis, hypercholesterolemia, diabetes mellitus.

**Conflict of interest.** The authors declare the absence of obvious or potential conflict of interest related to the publication of this article.

**Source of financing.** The study was carried out within the state assignment No. AAAA-A17-117112850280-2; statistical processing and analysis were supported by the Russian Science Foundation grant No. 21-15-00022.

**Conformity with the principles of ethics.** An informed consent to examination and processing of personal data was obtained from all individuals. The study was approved by the local Ethics Committee at IIPM – Branch of IC&G SB RAS (Protocol No. 6/2013 of 25.06.2013).

**For citation:** Ragino Yu.I., Khudyakova A.D., Striukova E.V., Denisova D.V., Shcherbakova L.V. Prevalence of diseases and pathological conditions in young people under 45 years of age with abdominal obesity in Siberia. *Bulletin of Siberian Medicine*. 2021; 20 (4): 39–48. <https://doi.org/10.20538/1682-0363-2021-4-39-48>.

## Распространенность заболеваний и патологических состояний у молодых людей до 45 лет с абдоминальным ожирением в Сибири

Рагино Ю.И., Худякова А.Д., Стрюкова Е.В., Денисова Д.В., Щербакова Л.В.

Научно-исследовательский институт терапии и профилактической медицины – филиал Федерального исследовательского центра «Институт цитологии и генетики Сибирского отделения Российской академии наук» (НИИТПМ – филиал ИЦиГ СО РАН)

Россия, 630089, г. Новосибирск, ул. Б. Богаткова, 175/1

### РЕЗЮМЕ

**Цель** – изучение распространенности абдоминального ожирения (АО) в популяции молодых людей 25–44 лет г. Новосибирска, а также распространенности терапевтических заболеваний и патологических состояний у лиц с АО.

**Материалы и методы.** Проведено одномоментное популяционное обследование населения 25–44 лет г. Новосибирска. На скрининге обследованы 1 415 человек, из них 670 мужчин (47,3%) и 745 женщин (52,7%). Беременные и женщины в декретном отпуске не включались в исследование. У обследуемых оценивалось наличие таких заболеваний и патологических состояний, как АО, артериальная гипертензия (АГ), повышенный индекс массы тела, ишемическая болезнь сердца (по валидизированным эпидемиологическим и функциональным критериям с расшифровкой электрокардиограммы по Миннесотскому коду), сахарный диабет (СД), сниженная скорость клубочковой фильтрации (СКФ), хронический бронхит (ХБ), повышенный уровень в крови общего ХС (гиперХСемия), повышенный уровень в крови ХС-ЛНП (гиперХС-ЛНПемия).

**Результаты.** Распространенность АО в популяции 25–44 лет г. Новосибирска составила 42,4%, у мужчин – 42,7%, у женщин – 42,1%. Обнаружено, что в молодой популяции до 45 лет АО оказывает прямое влияние на развитие АГ (отношение шансов (ОШ) 2,550, 95%-й доверительный интервал (95%-й ДИ) 1,899–3,422,  $p = 0,0001$ ), ХБ (ОШ = 1,830, 95%-й ДИ 1,326–2,527,  $p = 0,0001$ ), гиперХСемии (ОШ = 1,486, 95%-й ДИ 1,193–1,851,  $p = 0,0001$ ), гиперХС-ЛНПемии (ОШ = 1,527, 95%-й ДИ 1,222–1,907,  $p = 0,0001$ ), обратное влияние на развитие сниженной СКФ (ОШ = 0,603, 95%-й ДИ 0,427–0,852,  $p = 0,004$ ). В мужской популяции до 45 лет АО оказывает прямое влияние на развитие АГ, ХБ, гиперХСемии, гиперХС-ЛНПемии. В женской популяции до 45 лет АО оказывает прямое влияние на развитие СД, АГ, ХБ, гиперХС-ЛНПемии, и обратное – на развитие сниженной СКФ.

**Заключение.** Таким образом, в популяции до 45 лет абдоминальное ожирение ассоциировано с развитием распространенных терапевтических заболеваний и патологических состояний.

**Ключевые слова:** абдоминальное ожирение, популяция в возрасте до 45 лет, артериальная гипертензия, хронический бронхит, гиперхолестеринемия, сахарный диабет.

**Конфликт интересов.** Авторы заявляют об отсутствии конфликта интересов, связанных с публикацией настоящей статьи.

**Источник финансирования.** Исследование выполнено в рамках бюджетной темы по государственному заданию № ААААА 17-117112850280-2, статистическая обработка и анализ материала проведены при финансовой поддержке гранта Российского научного фонда № 21-15-00022.

**Соответствие принципам этики.** От всех лиц получено информированное согласие на обследование и обработку персональных данных. Исследование одобрено локальным этическим комитетом НИИТПМ – филиала ИЦиГ СО РАН (протокол № 6/2013 от 25.06.2013).

**Для цитирования:** Рагино Ю.И., Худякова А.Д., Стрюкова Е.В., Денисова Д.В., Щербакова Л.В. Распространенность заболеваний и патологических состояний у молодых людей до 45 лет с абдоминальным ожирением в Сибири. *Бюллетень сибирской медицины*. 2021; 20 (4): 39–48. <https://doi.org/10.20538/1682-0363-2021-4-39-48>.

## INTRODUCTION

Currently, obesity is a topical issue worldwide. The disease is associated with progressive spread and severe complications, which often cause death of patients at a young age [1, 2].

Recent research on abdominal obesity (AO) around the world has been devoted to the study of its impact on endocrine and cardiovascular pathologies [3–5].

Recent studies show that visceral adipose tissue serves not only for accumulation of energy substrates. It is also a kind of endocrine gland producing many different substances, which act at both local and systemic level. The products of adipocyte (visceral adipose tissue cells) secretion are hormones (leptin, adiponectin, and resistin), proinflammatory cytokines (tumor necrosis factor- $\alpha$ , interleukin (IL)-6, IL-8, etc.), and proteins of the renin-angiotensin system. Some of them are involved in the complement system and vascular hemostasis (plasminogen activator inhibitor-1 and others) [6–9]. Based on the known pathological effects of various biological substances secreted by visceral adipocytes, an increased blood level of adipokines/cytokines in AO is assumed to be a significant etio-pathogenetic link in the development of many common diseases and pathological conditions.

Novosibirsk (Russian Federation) shows high prevalence of AO and metabolic syndrome (MS) in the population over 45 years of age [10]. The problem of AO is poorly studied in the young working-age population of reproductive age. Therefore, the study is dedicated to the prevalence of AO in the population of Novosibirsk aged 25–44-years, as well as to the prevalence of diseases and pathological conditions in individuals with AO.

## MATERIALS AND METHODS

A cross-sectional, population-based study of the population of Novosibirsk was carried out. The study was approved by the local Ethics Committee at IIPM – Branch of IC&G SB RAS (Protocol No. 6/2013 of 25.06.2013). To build a population sample, we used a database of the Territorial Federal Compulsory Medical Insurance Fund for persons aged 25–44 years in one of the districts of Novosibirsk. The district was typical in terms of industrial, social, population, demographic, and transport structures and the level of population migration. Using a random number generator, a random representative sample of 2,500 people was built. Young age groups are known to be among the most rigid ones regarding response, so methods of gradual epidemiological stimulation were used: mail invitations, phone calls, information messages in the mass media. 1,415 people were examined at the screening – 670 men (47.3%) and 745 women (52.7%). The study did not include pregnant women and wo-

men on maternity leave. The response was 56.6%. An informed consent to the examination and personal data processing was obtained from all individuals participating in the study.

A team of doctors trained in standardized epidemiological screening methods conducted the screening procedure. The survey program included demographic and social data collection, a survey on smoking habits and alcohol use, a socioeconomic survey, a dietary survey, history of chronic diseases and medication use, Rose questionnaire, anthropometry, triplicate measurement of blood pressure (BP), spirometry, an ECG with findings interpreted according to the Minnesota code, etc.

Waist circumference (WC) was determined with a measuring tape applied horizontally in the middle between the lower edge of the costal arch and the sacral part of the ilium. AO was determined with  $WC \geq 94$  cm in men and  $\geq 80$  cm in women [11, 12]. BP was measured three times with an interval of two minutes on the right arm in a sitting position after a 5-minute rest using an automatic digital blood pressure monitor Omron M5-I (Japan). The average value for 3 measurements was registered. Arterial hypertension (AH) was determined at systolic blood pressure (SBP)  $\geq 140$  mmHg and / or diastolic blood pressure (DBP)  $\geq 90$  mmHg [12]. The body mass index (BMI) was calculated using the formula  $I = m/h^2$ , where  $m$  – body weight (kg),  $h$  – height. BMI was considered increased at  $> 25$  kg/m<sup>2</sup> [12]. Individuals who smoked at least one cigarette a day were considered smokers.

The epidemiological diagnosis of coronary artery disease (CAD) was made using validated epidemiological (the Rose Angina Questionnaire) and functional criteria (an ECG with findings classified according to the Minnesota Code (MC)). We used the following ECG determination of CAD based on the mentioned MC classification system for electrocardiographic findings (WHO guidelines):

- ECG changes indicative of new ischemia (new ST-T changes or new left bundle branch block (LBBB) – the Minnesota codes: ST-depression 4.1; 4.2; ST-elevation 9.2; LBBB 7.1);

- development of pathological Q waves in the ECG (the Minnesota codes: 1.1.1–1.2.5, 1.2.7), including the absence of unequivocal pathological Q waves in the first ECG or in the set of ECGs followed by a record with a pathological Q wave, or any Q

wave in leads V2–V3  $\approx 0.2$  s, or a QS complex in leads V2 and V3, or Q  $\approx 0.03$  s and  $\approx 0.01$  mV deep, or a QS complex in leads I, II, aVL, aVF or V4–V6 in any two leads of a contiguous lead grouping (I, aVL, V6: V4–V6: II, III, aVF).

Diabetes mellitus (DM) was established according to the epidemiological criteria at fasting plasma glucose levels  $\geq 7.0$  mmol/l [13] and/or normoglycemia in individuals with a medical history of established DM. The glomerular filtration rate (GFR) was calculated using the CKD-EPI formula (Chronic Kidney Disease Epidemiology Collaboration), which takes into account race, sex, age, and serum creatinine [14]. A decrease in the GFR was recorded at  $\text{GFR} < 90$  ml/min/1.73 cm<sup>2</sup>, GFR was considered normal at  $\text{GFR} \geq 90$  ml/min/1.73 cm<sup>2</sup>. Microalbuminuria was not determined.

The WHO Respiratory Diseases questionnaire and the European Community Respiratory Health Survey (ECRHS) were used to detect respiratory symptoms. The epidemiological diagnosis of chronic bronchitis (CB) was established in the presence of cough with mucus for at least three months a year or if there was a medical history of the disease. In addition, the respiratory function was studied with the help of spirometry according to the recommendations for performing spirometry on the Spiro USB Micro spirometer (Medical Limited, Great Britain).

Three reproducible attempts were selected for the analysis. The spirometry results were recorded and processed by the Spida 5 PC-based diagnostic software. We selected the best parameters of forced expiratory volume in 1 second, forced vital capacity, and their ratio to assess the respiratory function. The calculation of the respiratory function indices was carried out using comparative equations of proper values obtained in the Third National Health and Nutrition Examination Survey (NHANES III) [15].

A single blood sampling from the ulnar vein was performed on an empty stomach after 12-hour fasting. Blood parameters of the lipid profile, glucose, and creatinine were measured by the enzymatic method using standard TermoFisher reagents on the automatic biochemistry analyzer KoneLab 30i (Finland). Conversion of serum glucose to plasma glucose was performed according to the formula: plasma glucose (mmol/l) =  $-0.137 + 1.047 \times$

serum glucose (mmol/l). The values  $\geq 5.0$  mmol/l were considered increased blood levels of total cholesterol (hypercholesterolemia), the values  $\geq 3.0$  mmol/l were considered increased blood levels of LDL-C [12].

Statistical processing of the obtained results was performed using the SPSS software package (version 13.0). In the tables and text, the obtained data are presented as absolute and relative values  $n$  (%) for categorical variables and as  $Me (Q_{25}; Q_{75})$  for continuous variables due to non-normal distribution of most variables. The Kolmogorov – Smirnov test was used to check the normality of distribution. The nonparametric Mann – Whitney U-test was used to assess the differences between two independent samples. The Pearson's chi-squared test was used to compare the differences between the sets of data. Associations were evaluated with the help of multiple logistic regression analysis, performed under the following conditions: the dependent variable is dichotomous; independence of observations; absence of multicollinearity, i.e., situations when the independent variables strongly correlate with one other ( $r > 0.9$ ); the linear dependence between each independent variable and the logarithm of the odds ratio (OR) (log odds); independence of the residuals. The results of the multiple logistic regression analysis were presented as OR and 95% confidence interval (95% CI) for OR. The critical significance level of the null hypothesis ( $p$ ) was equal to 0.05.

## RESULTS

1,415 individuals were examined, and AO was determined in 600 people (42.4%). 286 of the 670 examined men had AO (42.7%) and 314 of the 745 examined women had AO (42.1%). The prevalence of AO in the population of Novosibirsk aged 25–44 years was 42.4%: 42.7% in men and 42.1% in women.

Table 1 shows the characteristics of the studied factors depending on the presence of AO in the population of Novosibirsk aged 25–44 years. The individuals with AO, both men and women, demonstrated higher values of SBP, DBP, hip circumference (HC), BMI, age, blood glucose levels, total cholesterol, and LDL-C. Male population did not show any differences in the blood levels of creatinine depending on the presence of AO. Female population with

AO had lower blood creatinine levels than women without AO.

Table 2 shows the prevalence of certain diseases and pathological conditions in people under 45 years of age with AO. In the population of individuals with AO, the prevalence of DM was 2.3 times higher, AH was 2.3 times higher, CB was 1.5 times higher, hypercholesterolemia was 1.3 times higher, and hyper-LDL-cholesterolemia was 1.2 times higher than in individuals without AO. Similar results were found in women. Thus, in women with AO, the prevalence of DM was 13.0 times higher,

AH was 4.4 times higher, CB was 1.9 times higher, hypercholesterolemia was 1.2 times higher, and hyper-LDL-cholesterolemia was 1.3 times higher than in individuals without AO. Men showed significant differences only for hypertension and lipid disorders. So, in men with AO, the prevalence of hypertension was 1.8 times higher, hypercholesterolemia – 1.4 times higher, and hyper-LDL-cholesterolemia – 1.2 times higher than in men without AO. No differences were detected in the prevalence of CAD and reduced GFR depending on the presence of AO, both in men and women.

Table 1

Characteristics of the studied factors depending on the presence of abdominal obesity in the population of Novosibirsk aged 25–44 years, $Me (Q_{25}; Q_{75})$									
Continuous variables	Population, $n = 1.415$			Men, $n = 670$			Women, $n = 745$		
	АО есть, $n = 600$	АО нет, $n = 815$	$p$	АО есть, $n = 286$	АО нет, $n = 384$	$p$	АО есть, $n = 314$	АО нет, $n = 431$	$p$
SBP	123.0 (114.0; 134.0)	116.5 (107.5; 125.5)	<0.001	128.0 (120.0; 137.5)	122.5 (115.5; 131.5)	<0.001	118.3 (109.5; 129.0)	110.3 (103.5; 118.5)	<0.001
DBP	82.5 (75.0; 90.0)	76.0 (69.5; 82.5)	<0.001	85.5 (79.5; 92.5)	80.0 (74.5; 88.0)	<0.001	79.0 (72.0; 86.0)	72.0 (66.5; 77.9)	<0.001
WC	97.8 (88.8; 103.5)	77.0 (71.0; 85.0)	<0.001	101.1 (98.0; 107.9)	85.9 (81.0; 90.0)	<0.001	89.6 (84.9; 98.0)	72.0 (67.6; 76.0)	<0.001
Glucose	5.83 (5.41; 6.18)	5.62 (5.20; 5.94)	<0.001	5.94 (5.52; 6.25)	5.73 (5.41; 6.15)	<0.001	5.73 (5.41; 6.04)	5.41 (5.10; 5.73)	<0.001
Total cholesterol (TC)	5.14 (4.52; 5.79)	4.83 (4.21; 5.43)	<0.001	5.27 (4.65; 5.50)	4.87 (4.26; 5.50)	<0.001	5.01 (4.39; 5.68)	4.78 (4.19; 5.39)	0.001
LDL-C	3.26 (2.66; 3.83)	3.02 (2.45; 3.60)	<0.001	3.34 (2.82; 4.01)	3.15 (2.55; 3.70)	0.001	3.15 (2.58; 3.72)	2.89 (2.36; 3.50)	<0.001
Creatinine	74.0 (66.5; 82.0)	75.0 (68.0; 82.0)	0.117	81.0 (73.0; 87.0)	79.0 (73.0; 86.0)	0.485	69.0 (64.0; 74.0)	71.0 (66.0; 77.0)	0.002
HC	108.7 (104.0; 114.0)	96.2 (92.9; 100.0)	<0.001	101.1 (104.5; 113.0)	97.1 (94.0; 100.4)	<0.001	109.0 (103.8; 116.0)	95.0 (91.8; 99.0)	<0.001
BMI	29.50 (27.07; 32.43)	22.60 (20.66; 24.66)	<0.001	29.90 (28.02; 32.41)	23.78 (21.85; 25.57)	<0.001	29.0 (25.81; 32.44)	21.78 (20.09; 23.44)	<0.001
Age	39.0 (33.8; 42.8)	35.8 (31.0; 41.1)	<0.001	38.5 (33.0; 42.4)	35.0 (30.4; 40.4)	<0.001	39.5 (34.8; 43.1)	36.4 (31.4; 41.4)	<0.001

Table 2

Prevalence of diseases and pathological conditions depending on the presence of abdominal obesity in the population of Novosibirsk aged 25–44 years, %									
Categorical variables	Population, $n = 1.415$			Men, $n = 670$			Women, $n = 745$		
	АО (+), $n = 600$	АО (–), $n = 815$	$p$	АО (+), $n = 286$	АО (–), $n = 384$	$p$	АО (+), $n = 314$	АО (–), $n = 431$	$p$
Detected CAD	3.8	3.0	0.430	2.9	2.6	0.839	4.7	3.4	0.396
DM	3.5	1.5	0.022	4.9	11.0	0.260	2.6	0.2	0.004
AH	28.1	12.3	<0.0001	37.9	21.1	<0.0001	19.2	4.4	<0.001

Table 2 (continued)

Categorical variables	Population, <i>n</i> = 1.415			Men, <i>n</i> = 670			Women, <i>n</i> = 745		
	AO (+), <i>n</i> = 600	AO (–), <i>n</i> = 815	<i>p</i>	AO (+), <i>n</i> = 286	AO (–), <i>n</i> = 384	<i>p</i>	AO (+), <i>n</i> = 314	AO (–), <i>n</i> = 431	<i>p</i>
CB	26.5	17.7	<0.001	31.5	24.4	0.072	21.8	11.7	0.001
Hypercholesterolemia	56.2	43.5	<0.0001	61.4	44.7	<0.0001	51.4	42.5	0.016
Hyper-LDL-cholesterolemia	62.5	50.4	<0.0001	66.3	56.2	0.009	59.1	45.3	0.001
Reduced GFR	21.3	24.4	0.254	9.8	9.9	0.983	30.5	37.3	0.166

At the next stage of the study, a logistic regression analysis was performed to assess the impact of AO on the development of diseases and pathological conditions (Table 3). The categorical variables

of CAD, DM, AH, CB, lipid disorders, and reduced GFR were included in individual models as dependent variables, whereas AO, sex, age, and some other parameters were taken as independent variables.

Table 3

**Logistic regression analysis of the impact of abdominal obesity on the development of diseases and pathological conditions in the population of Novosibirsk aged 25–44 years**

Categorical variables	Population, <i>n</i> = 1,415			Men, <i>n</i> = 670			Women, <i>n</i> = 745		
	OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>	OR	95% CI	<i>p</i>
Detected CAD	1.158	0.624 – 2.147	0.642	1.048	0.384 – 2.856	0.928	1.194	0.538 – 2.652	0.663
DM	1.971	0.950 – 4.087	0.068	1.255	0.545 – 2.890	0.593	10.765	1.316 – 88.057	0.027
AH	2.550	1.899 – 3.422	0.0001	2.070	1.450 – 2.956	0.0001	4.074	2.343 – 7.082	0.0001
CB	1.830	1.326 – 2.527	0.0001	1.655	1.069 – 2.563	0.024	2.130	1.311 – 3.459	0.002
Hypercholesterolemia	1.486	1.193 – 1.851	0.0001	1.805	1.313 – 2.483	0.0001	1.293	0.957 – 1.746	0.094
Hyper-LDL-cholesterolemia	1.527	1.222 – 1.907	0.0001	1.439	1.040 – 1.990	0.028	1.595	1.180 – 2.156	0.002
Reduced GFR	0.603	0.427 – 0.852	0.004	0.708	0.364 – 1.376	0.309	0.573	0.382 – 0.861	0.007

We found a significant impact of AO on the development of AH in the population, including men and women (Table 3). In the general population, age (OR = 1.089, 95% CI 1.062–1.117, *p* = 0.0001), male sex (OR = 3.632, 95% CI 2.677–4.928, *p* = 0.0001), and smoking (OR = 1.689, 95% CI 1.188–2.402, *p* = 0.003) significantly influenced the development of AH along with AO. In men, only age, along with AO, influenced the development of hypertension (OR = 1.074, 95% CI 1.041–1.107, *p* = 0.0001). In women, age (OR = 1.128, 95% CI 1.077–1.180, *p* = 0.0001) and smoking (OR = 2.102, 95% CI 1.166–3.789, *p* = 0.014), along with AO, had a significant impact on the development of hypertension.

We found a significant impact of AO on the development of CB in the population, including men and women (Table 3). In the general population, age (OR = 1.038, 95% CI 1.010–1.067, *p* = 0.008), male sex (OR = 1.457, 95% CI 1.062–2.001, *p* = 0.020),

and smoking (OR = 6.284, 95% CI 4.242–9.308, *p* = 0.0001) significantly influenced the development of CB along with AO. In men, the development of CB was significantly influenced by age (OR = 1.040, 95% CI 1.002–1.080, *p* = 0.039) and smoking (OR = 7.268, 95% CI 3.981–13.270, *p* = 0.0001), along with AO. In women, only smoking, along with AO, significantly influenced the development of CB (OR = 5.230, 95% CI 3.062–8.933, *p* = 0.0001).

A significant effect of AO on the development of hyper-LDL-cholesterolemia in the population, including men, was detected (Table 3). In the general population, age (OR = 1.037, 95% CI 1.019–1.056, *p* = 0.0001) and male sex (OR = 1.541, 95% CI 1.247–1.905, *p* = 0.0001) significantly influenced the development of hyper-LDL-cholesterolemia along with AO. In men, only age, along with AO, influenced the development of hyper-LDL-cholesterolemia (OR = 1.032, 95% CI 1.005–1.060,

$p = 0.020$ ). In women, only age had a significant effect on the development of hyper-LDL-cholesterolemia (OR = 1.042, 95% CI 1.016–1.067,  $p = 0.001$ ).

A significant effect of AO on the development of hypercholesterolemia in the population, including men, was identified (Table 3). In the general population, age (OR = 1.046, 95% CI 1.028–1.065,  $p = 0.0001$ ) and male sex (OR = 1.310, 95% CI 1.064–1.613,  $p = 0.011$ ), along with AO, significantly influenced the development of hypercholesterolemia. In men, only age, along with AO, influenced the development of hypercholesterolemia (OR = 1.048, 95% CI 1.021–1.076,  $p = 0.001$ ). In women, the development of hypercholesterolemia was affected only by age (OR = 1.045, 95% CI 1.021–1.069,  $p = 0.0001$ ).

A significant influence of AO on the development of DM in women was also detected (Table 3). In the general population, the development of DM was influenced by age (OR = 1.113, 95% CI 1.045–1.185,  $p = 0.001$ ) and male sex (OR = 2.976, 95% CI 1.440–6.151,  $p = 0.003$ ); in men – only by age (OR = 1.152, 95% CI 1.065–1.247,  $p = 0.0001$ ) and in women – only by AO.

Finally, a significant reverse effect of AO on the development of reduced GFR in the population, including women, was established (Table 3). In the general population, age (OR = 1.083, 95% CI 1.054–1.13,  $p = 0.0001$ ) and female sex (OR = 0.183, 95% CI 0.125–0.268,  $p = 0.0001$ ), along with AO, significantly influenced the development of reduced GFR. In men, the development of reduced GFR was influenced only by age (OR = 1.099, 95% CI 1.035–1.167,  $p = 0.002$ ). In women, age, along with AO, also had a significant effect on the development of reduced GFR (OR = 1.078, 95% CI 1.046–1.12,  $p = 0.0001$ ). No effect of AO on the CAD development in the population, including men and women, was found.

In the young population under the age of 45, AO had a significant direct effect on the development of AH, CB, hypercholesterolemia, hyper-LDL-cholesterolemia and a reverse effect on the development of reduced GFR. In the male population under 45 years of age, AO had a significant direct effect on the development of AH, CB, hypercholesterolemia, and hyper-LDL-cholesterolemia. In the female population under the age of 45, AO had a significant direct effect on the development of DM, AH, CB,

and hyper-LDL-cholesterolemia and a reverse effect on the development of reduced GFR.

## DISCUSSION

Our results confirming the direct effect of AO on the AH development in young people under 45 years of age correspond with the known data on the relationship between AO and AH, also as criteria/signs of metabolic syndrome (MS), as well as to data from other studies of recent years. Thus, Y. Zhao et al. in the cohort study of rural Chinese residents of a broad age group showed that AO increased a 6-year risk of developing AH in both men and women [16]. J. B. Almeida et al. found that young women with AO aged 20–59 years demonstrated a two times higher AH prevalence than women without AO [17].

Our results regarding the direct effect of AO on the development of CB in young people under 45 years of age do not contradict the data of other studies. E. Pekkarinen et al. concluded that even mild AO in healthy non-smoking adults was associated with obstructive changes in the lungs and a decrease in the vital capacity of the lungs according to spirometry [18]. Similar data were obtained by A. Vatrella et al. in the cohort study of women in Italy [19]. Discussing a possible mechanism of the association between AO and CB, it is essential to note the etiopathogenetic synergy of proinflammatory biomolecules secreted by visceral adipocytes in AO and factors of chronic inflammation that potentiate the development of chronic inflammatory diseases, including CB [6, 20].

The obtained results regarding the direct effect of AO on the development of lipid disorders (hypercholesterolemia, hyper-LDL-cholesterolemia) in young people under 45 years of age have not come as unexpected, since the data on the relationship between AO and lipid disorders, also as criteria/signs of MS, have also been known for a long time. Z. Hertelyova et al. also found a positive association of non-HDL-C with increased WC and BMI in students. However, unlike us, they did not find an association of WC with the level of total blood cholesterol [21].

MS based on AO and insulin resistance is known to play a potentiating role in the development of type 2 diabetes. In this respect, the expected results regarding the association of AO and DM were not obtained, since this association was identified only



in women. However, it should be noted that our study determined DM only with the help of epidemiological criteria (fasting plasma glucose level) [13] and did not take into account the type of DM. In the 12-year cohort study, F. Salehinia et al. also identified an association of AO with the development of type 2 diabetes only in women over 20 years of age, but not in men [22].

Our results regarding the reverse effect of AO on the development of reduced GFR in young people under 45 years of age, including women, do not correspond with the data of other studies. Several studies demonstrated a direct association of AO with reduced GFR and kidney pathology [23–25]. On the other hand, A. Shahali et al. found no association of AO with an increased risk of kidney failure in either men or women in the cohort study of 7,002 people over the age of 20 [26]. It is important to note that our study considered  $GFR < 90 \text{ ml/min/1.73 cm}^2$  as reduced GFR, since there were only 4 young people with reduced  $GFR < 60 \text{ ml/min/1.73 cm}^2$  (this criterion for reduced GFR is used in the majority of studies), which was not enough for a correct statistical analysis of the results.

Finally, the expected association of AO with early CAD (according to epidemiological criteria) in young people aged 25–44 years was not identified. The obtained data differ from the known results of other numerous studies showing a direct association of AO with CAD development and its complications. It should be noted that most of these studies were conducted on populations, cohorts or selective clinical groups of people over 45 years of age. Our results presented in Tables 2 and 3 reflect higher prevalence of CAD in individuals with AO and a direct association of CAD with AO, but their statistical significance has not been achieved. This is probably due to the low number of CAD cases in the examined young population under the age of 45.

## CONCLUSION

Therefore, it is important to note that AO, including AO in young people, probably causes and triggers the development of not only endocrine and cardiovascular diseases, but also a broad range of other socially sensitive diseases and pathological conditions. These studies will undoubtedly continue, including the search for pathogenetic associations of AO with the development of a wide range of diseases.

## REFERENCES

1. Ural D., Kiliçkap M., Göksülük H., Karaaslan D., Kayıkçioğlu M., Özer N., Barçın C., Yılmaz M.B., Abacı A., Şengül Ş., Arınoy T., Erdem Y., Sanisoğlu Y., Şahin M., Tokgözoğlu L. Data on prevalence of obesity and waist circumference in Turkey: Systematic review, meta-analysis and meta-regression of epidemiological studies on cardiovascular risk factors. *Türk Kardiyol. Dern. Ars.* 2018; 46: 577–590. DOI: 10.5543/tkda.2018.62200.
2. Triggiani A.I., Valenzano A., Trimigno V., Di Palma A., Moscatelli F., Cibelli G., Messina G. Heart rate variability reduction is related to a high amount of visceral adiposity in healthy young women. *PLoS One.* 2019; 14. (2019). DOI: 10.1371/journal.pone.0223058.
3. Kim H.Y., Kim J.K., Shin G.G., Han J.A., Kim J.W. Association between Abdominal Obesity and Cardiovascular Risk Factors in Adults with Normal Body Mass Index: Based on the Sixth Korea National Health and Nutrition Examination Survey. *J. Obes. Metab. Syndr.* 2019; 28: 262–270. DOI: 10.7570/jomes.2019.28.4.262.
4. Melin E.O., Thulesius H.O., Hillman M., Landin-Olsson M., Thunander M. Abdominal obesity in type 1 diabetes associated with gender, cardiovascular risk factors and complications, and difficulties achieving treatment targets: A cross sectional study at a secondary care diabetes clinic. *BMC Obes.* 2018; 5. DOI: 10.1186/s40608-018-0193-5.
5. Mohammadi H., Ohm J., Discacciati A., Sundstrom J., Hambræus K., Jernberg T., Svensson P. Abdominal obesity and the risk of recurrent atherosclerotic cardiovascular disease after myocardial infarction. *Eur. J. Prev. Cardiol.* 2020; 27: 1944–1952. DOI: 10.1177/2047487319898019.
6. Alexopoulos N., Katritsis D., Raggi P. Visceral adipose tissue as a source of inflammation and promoter of atherosclerosis. *Atherosclerosis.* 2014; 233: 104–112. DOI: 10.1016/j.atherosclerosis.2013.12.023.
7. Ragino Y.I., Stakhneva E.M., Polonskaya Y.V., Kashtanova E.V. The role of secretory activity molecules of visceral adipocytes in abdominal obesity in the development of cardiovascular disease: A review. *Biomolecules.* 2020; 10. DOI: 10.3390/biom10030374.
8. Dutheil F., Gordon B.A., Naughton G., Crendal E., Courteix D., Chaplais E., Thivel D., Lac G., Benson A.C. Cardiovascular risk of adipokines: a review. *J. Int. Med. Res.* 2018; 46: 2082–2095. DOI: 10.1177/0300060517706578.
9. Neeland I.J., Ross R., Després J.P., Matsuzawa Y., Yamashita S., Shai I., Seidell J., Magni P., Santos R.D., Arsenault B., Cuevas A., Hu F.B., Griffin B., Zambon A., Barter P., Fruchart J.C., Eckel R.H. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol.* 2019; 7: 715–725. DOI: 10.1016/S2213-8587(19)30084-1.
10. Vikhireva O., Pajak A., Broda G., Malyutina S., Tamosiunas A., Kubinova R., Simonova G., Skodova Z., Bobak M., Pikhart H. SCORE performance in central and eastern Europe and former Soviet Union: MONICA and HAPIEE results. *Eur. Heart J.* 2014; 35. DOI: 10.1093/eurheartj/eh189.



11. Alberti K.G.M.M., Eckel R.H., Grundy S.M., Zimmet P.Z., Cleeman J.I., Donato K.A., Fruchart J.C., James W.P.T., Loria C.M., Smith S.C. Harmonizing the metabolic syndrome: A joint interim statement of the international diabetes federation task force on epidemiology and prevention; National heart, lung, and blood institute; American heart association; World heart federation; International atherosclerosis society; And international association for the study of obesity. *Circulation*. 2009; 120: 1640–1645. DOI: 10.1161/CIRCULATIONAHA.109.192644.
12. Mach F., Baigent C., Catapano A.L., Koskinas K.C., Casula M., Badimon L., Chapman M.J., De Backer G.G., Delgado V., Ference B.A., Graham I.M., Halliday A., Landmesser U., Mihaylova B., Pedersen T.R., Riccardi G., Richter D.J., Sabatine M.S., Taskinen M.R., Tokgozoglu L., Wiklund O., Mueller C., Drexel H., Aboyans V., Corsini A., Doehner W., Farnier M., Gigante B., Kayikcioglu M., Krstacic G., Lambrinou E., Lewis B.S., Masip J., Moulin P., Petersen S., Petronio A.S., Piepoli M.F., Pinto X., Raber L., Ray K.K., Reiner Z., Riesen W.F., Roffi M., Schmid J.P., Shlyakhto E., Simpson I.A., Stroes E., Sudano I., Tselepis A.D., Viigimaa M., Vindis C., Vonbank A., Vrablik M., Vrsalovic M., Gomez J.L.Z., Collet J.P., Windecker S., Dean V., Fitzsimons D., Gale C.P., Grobbee D.E., alvorsen S., Hindricks G., Iung B., Jüni P., Katus H.A., Leclercq C., Lettino M., Merkely B., Sousa-Uva M., Touyz R.M., Nibouche D., Zelveian P.H., Siostrzonek P., Najafav R., Van De Borne P., Pojskic B., Postadzhiyan A., Kypris L., Spinar J., Larsen M.L., Eldin H.S., Strandberg T.E., Ferrières J., Agladze R., Laufs U., Rallidis L., Bajnok L., Gudjonsson T., Maher V., Henkin Y., Gulizia M.M., Mussagaliyeva A., Bajraktari G., Kerimkulova A., Latkovskis G., Hamoui O., Slapikas R., Visser L., Dingli P., Ivanov V., Boskovic A., Nazzi M., Visseren F., Mitevska I., Retterstøl K., Jankowski P., Fontes-Carvalho R., Gaita D., Ezhov M., Foscoli M., Giga V., Pella D., Fras Z., De Isla L.P., Hagström E., Lehmann R., Abid L., Ozdogan O., Mitchenko O., Patel R.S. 2019 ESC/EAS guidelines for the management of dyslipidaemias: Lipid modification to reduce cardiovascular risk. *Eur. Heart J.* 2020; 41: 111–188. DOI: 10.1093/eurheartj/ehz455.
13. Alberti K.G.M.M., Zimmet P.Z. Definition, diagnosis and classification of diabetes mellitus and its complications part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Original articles. 1997. DOI: 10.1002/(SICI)1096-9136(199807)15:7.
14. Levin A., Stevens P.E. Summary of KDIGO 2012 CKD Guideline: Behind the scenes, need for guidance, and a framework for moving forward. *Kidney Int.* 2014; 85: 49–61. DOI: 10.1038/ki.2013.444.
15. Hankinson J.L., Odencrantz J.R., Fedan K.B. Spirometric reference values from a sample of the general U.S. Population. *Am. J. Respir. Crit. Care Med.* 1999; 159: 179–187. DOI: 10.1164/ajrccm.159.1.9712108.
16. Sun H., Liu Y., Liu D., Zhou Q., Guo C., Li Q., Tian G., Wu X., Hu D., Sun X., Zhang M. Metabolically healthy general and abdominal obesity are associated with increased risk of hypertension. *Br. J. Nutr.* 2020; 123: 583–591. DOI: 10.1017/S0007114519003143.
17. Almeida J.B., Kian K.O., Lima R.C., De Souza M.C.C. Total and abdominal adiposity and hypertension in indigenous women in midwest Brazil. *PLoS One*. 2016; 11: 1–12. DOI: 10.1371/journal.pone.0155528.
18. Pekkarinen E., Vanninen E., Länsimies E., Kokkarinen J., Timonen K.L. Relation between body composition, abdominal obesity, and lung function. *Clin. Physiol. Funct. Imaging*. (2012; 32: 83–88. DOI: 10.1111/j.1475-097X.2011.01064.x.
19. Vatrella A., Calabrese C., Mattiello A., Panico C., Costigliola A., Chiodini P., Panico S. Abdominal adiposity is an early marker of pulmonary function impairment: Findings from a Mediterranean Italian female cohort. *Nutr. Metab. Cardiovasc. Dis.* 2016; 26: 643–648. DOI: 10.1016/j.numecd.2015.12.013.
20. Borges M.D., Franca E.L., Fujimori M., Silva S.M.C., de Marchi P.G.F., Deluque A.L., Honório-Franca A.C., de Abreu L.C. Relationship between proinflammatory cytokines/chemokines and adipokines in serum of young adults with obesity, endocrine, metab. immune disord. *Drug Targets*. 2018; 18: 260–267. DOI: 10.2174/1871530318666180131094733.
21. Hertelyova Z., Salaj R., Chmelarova A., Dombrovsky P., Dvorakova M.C., Kruzliak P. The association between lipid parameters and obesity in university students. *J. Endocrinol. Invest.* 2016; 39: 769–778. DOI: 10.1007/s40618-015-0240-8.
22. Salehinia F., Abdi H., Hadaegh F., Serahati S., Valizadeh M., Azizi F., Hosseiniapanah F. Abdominal obesity phenotypes and incident diabetes over 12 years of follow-up: The Tehran Lipid and glucose study. *Diabetes Res. Clin. Pract.* 2018; 144: 17–24. DOI: 10.1016/j.diabres.2018.07.021.
23. Sarathy H., Henriquez G., Abramowitz M.K., Kramer H., Rosas S.E., Johns T., Kumar J., Skversky A., Kaskel F., Melamed M.L. Abdominal obesity, race and chronic kidney disease in young adults: Results from NHANES 1999–2010. *PLoS One*. 2016; 11. DOI: 10.1371/journal.pone.0153588.
24. Dias R.S.C., Calado I.L., De Alencar J.D., Hortegal E.V., Santos E.J.F., De Araújo Brito D.J., Lages J.S., Dos Santos A.M., Filho N.S. Abdominal obesity and reduction of glomerular filtration. *Rev. Assoc. Med. Bras.* 2018; 64: 346–353. DOI: 10.1590/1806-9282.64.04.346.
25. Tsao Y.C., Chen J.Y., Yeh W.C., Li W.C. Gender- and age-specific associations between visceral obesity and renal function impairment. *Obes. Facts*. 2019; 12: 67–77. DOI: 10.1159/000496626.
26. Shahali A., Tasdighi E., Barzin M., Mahdavi M., Valizadeh M., Niroomand M., Azizi F., Hosseiniapanah F. Abdominal obesity phenotypes and risk of kidney function decline: Tehran Lipid and Glucose Study. *Obes. Res. Clin. Pract.* 2020;(14):168–175. DOI: 10.1016/j.orcp.2020.03.006.

## Authors contribution

Ragino Yu. I. – conception, methodology, visualization, drafting of the manuscript. Khudyakova A.D – carrying out of the research. Striukova E.V. – carrying out of the research, drafting of the manuscript, review and editing of the article. Denisova D.V. – provision of resources. Shcherbakova L.V. – carrying out of formal analysis. All authors have reviewed the article and agreed with its contents.

---

## Authors information

**Ragino Yulia I.**, Dr. Sci. (Med.), Professor, Corresponding Member of the RAS, Head of the IIPM – Branch of IC&G SB RAS, Novosibirsk, Russian Federation. ORCHID 0000-0002-4936-8362.

**Khudyakova Alyona D.**, Cand. Sci (Med.), Head of Laboratory of Genetic and Environmental Determinants of the Human Life Cycle, IIPM – Branch of IC&G SB RAS, Novosibirsk, Russian Federation. ORCHID 0000-0001-7875-1566.

**Striukova Evgeniia V.**, Junior Researcher, Laboratory of Genetic and Environmental Determinants of the Human Life Cycle, IIPM – Branch of IC&G SB RAS, Novosibirsk, Russian Federation. ORCHID 0000-0001-5316-4664.

**Denisova Diana V.**, Dr. Sci. (Med.), Leading Researcher, Laboratory of Preventive Medicine, IIPM – Branch of IC&G SB RAS, Novosibirsk, Russian Federation. ORCID 0000-0002-2470-2133.

**Shcherbakova Liliia V.**, Senior Researcher, Laboratory of Clinical-Populational and Prophylactic Studies on Internal and Endocrine Diseases, IIPM – Branch of IC&G SB RAS, Novosibirsk, Russian Federation. ORCID 0000-0001-9270-9188.

(✉) **Striukova Evgeniia V.**, e-mail: stryukova.j@mail.ru

Received 16.07.2021

Accepted 10.09.2021