The prevalence of obesity and its relationship to cardiometabolic risk factors in the LIPIDOGRAM studies

Dariusz Nowak[1]*, Martyna Fronczek[2]*, Tomasz Czapor[2], Tadeusz Osadnik[2], Marek Gierlotka[3], Tomasz Tomasik[4], Adam Windak[4], Agnieszka Kuras[5], Marcin Miga[6], Agata Kulkowska-Gaj[7], Gregory Y H Lip[8, 9], Dimitri P Mikhailidis[10], Peter P Toth[11, 12], Peter E Penson[13–15], Maciej Banach[16, 17]**, Jacek Jóźwiak[7]**

[1]Municipal Hospital, Częstochowa, Poland
[2]Department of Pharmacology, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland
[3]Department of Cardiology, Faculty of Medicine, University of Opole, Opole, Poland
[4]Department of Family Medicine, Jagiellonian University Medical College, Kraków, Poland
[5]Multiprofile Medical Simulation Center, Faculty of Medicine, University of Opole, Opole, Poland
[6]Clinical University Hospital, Opole, Poland
[7]Department of Family Medicine and Public Health, Faculty of Medicine, University of Opole, Opole, Poland
[8]Liverpool Centre for Cardiovascular Science at University of Liverpool, Liverpool John Moores University and Liverpool Heart and Chest Hospital, Liverpool, United Kingdom
[9]Department of Clinical Medicine, Danish Center for Health Services Research, Aalborg University, Aalborg, Denmark
[10]Department of Clinical Biochemistry, Royal Free Hospital Campus, University College London Medical School, University College London, London, United Kingdom
[11]Ciccarone Center for the Prevention of Cardiovascular Disease, Johns Hopkins University School of Medicine, Baltimore, Maryland, United States
[12]CGH Medical Center, Department of Preventive Cardiology, Sterling, Illinois, United States
[13]Clinical Pharmacy and Therapeutics Research Group, School of Pharmacy and Biomolecular Sciences, Liverpool John Moores University, Liverpool, United Kingdom
[14]Liverpool Centre for Cardiovascular Science, University of Liverpool, Liverpool, United Kingdom
[15]Department of Cardiovascular and Metabolic Medicine, Institute of Life Course and Medical Sciences, University of Liverpool, Liverpool, United Kingdom
[16]Department of Preventive Cardiology and Lipidology, Medical University of Lodz, Łódź, Poland
[17]Faculty of Medicine, John Paul II Catholic University of Lublin, Lublin, Poland
*Both authors equally contributed as first authors.
**Both authors equally contributed as senior authors.

Correspondence to:
Martyna Fronczek, PhD,
Department of Pharmacology,
Faculty of Medical Sciences in Zabrze,
Medical University of Silesia,
Jordana 38, 41–800 Zabrze,
Poland,
phone: +48 32 272 26 83,
e-mail: mfronczek@sum.edu.pl

DOI: 10.33963/v.phj.110885

Received:
October 30, 2025

Accepted:
January 26, 2026

Early publication date:
January 26, 2026

ABSTRACT

Background: High body mass index and abdominal obesity are associated with an increased risk of cardiometabolic diseases.

Aims: We aimed to investigate the prevalence of obesity in 45 591 individuals included in the LIPIDOGRAM cohort studies conducted in 2004, 2006, and 2015, and to examine the relationship between obesity and cardiometabolic risk factors such as diabetes, hypertension, and dyslipidemia.

Methods: To analyze the changes in the prevalence of central obesity, diabetes, hypertension, and dyslipidemia, age standardization was carried out according to the general population for the years 2004, 2006, and 2015.

Results: We observed a significant (P <0.001 in all groups) increase in the prevalence of abdominal obesity (39% of patients in 2004 to 51% in 2015), age-standardized obesity (26.8% in 2004 to 30.8% in 2015), and central obesity (30.1% in 2004 to 42.2% in 2015). Between 2004 and 2015, there was observed a significant increase in the age-standardized prevalence of diabetes (7.8% in 2004 to 9.8% in 2015) and an increase in the age-standardized prevalence of dyslipidemia (74.4% in 2004 to 76.4% in 2015). For hypertension, a decrease in age-standardized prevalence was observed (38.1% in 2004 to 37.8% in 2015).

Conclusions: An increase in body mass index and visceral obesity was observed among Polish primary health care patients included in the LIPIDOGRAM study from 2004 to 2015. As patients age, the number of cases of diabetes and dyslipidemia increases. These changes cause an increase in cardiometabolic risk.

Key words: diabetes mellitus, dyslipidemia, hypertension, obesity, prevalence

WHAT'S NEW?

This study provides valuable data on the prevalence of obesity, diabetes, hypertension, and dyslipidemia in the Polish population, based on a large and representative population of primary health care patients recruited within the nationwide cardiovascular risk factor LIPIDOGRAM study conducted in outpatient primary health care centers in Poland in 2004, 2006, and 2015. An increase in the prevalence of obesity, diabetes, and dyslipidemia was observed in the Polish primary health care patient population. Both unstandardized and standardized prevalence rates are presented, which provides a solid basis for future international comparisons. Recognition of the growing burden of obesity in Poland, along with the increased prevalence of diabetes and dyslipidemia, is crucial for optimizing healthcare policies and clinical practices for Eastern Europe. A thorough understanding of these epidemiological trends will help primary care physicians, health politicians, and prevention managers in the Polish health service to develop more effective health care strategies and care for patients at higher risk of cardiovascular events.

INTRODUCTION

Cardiometabolic syndrome (CMS) is a combination of numerous metabolic dysfunctions, including hypertension (HTN), central obesity, dyslipidemia, and insulin resistance with impaired glucose disposal. The components of CMS cause atherosclerotic cardiovascular disease (ASCVD) [1]. Lifestyle changes and socioeconomic factors undoubtedly contribute to the development of cardiometabolic disorders. An increased risk of developing cardiovascular disease (CVD) has been demonstrated in overweight and obese individuals, especially those with central obesity [2]. CVDs are one of the leading causes of death in Europe and worldwide [3]. On the other hand, there is still a lack of awareness in society of the relationship between CVD and atherosclerosis [4]. Morbidity and mortality related to sudden CV events will increase over the next few decades in both low- and middle-income countries, as well as in highly developed countries [5].

Obesity is a chronic disease characterized by disturbances in the body's energy homeostasis, resulting in excessive adipose tissue accumulation [5, 6]. Body mass index (BMI) is a value obtained by dividing body weight expressed in kilograms divided by the square of height (in meters) and can be used to diagnose obesity [6]. It is worth emphasizing that BMI may not reflect visceral and gluteal-femoral adipose tissue distribution within the body. Consequently, waist circumference (WC) measurements also diagnose central (abdominal) obesity. It should also be noted that for people aged 65 years and over, sarcopenia is a common condition, which directly affects the distribution of adipose tissue determined by BMI and WC [7]. Metabolic complications are considerably more likely to occur in individuals with a WC of 88 cm or greater in women and 102 cm or more in men [8]. High BMI and abdominal obesity are associated with an increased risk for cardiometabolic diseases, including insulin resistance, type 2 diabetes (DM), CVDs, HTN, and mixed dyslipidemias [9, 10]. Additionally, individuals with a higher BMI are more susceptible to developing metabolic syndrome and diabetes [10], and the occurrence of higher obesity classes and the duration of obesity are associated with a variety of CVDs [11]. Along with diseases such as insulin resistance, elevated serum levels of apolipoprotein B, hypertriglyceridemia, hyperuricemia, hypertension, chronic kidney disease, hyperglycemia, diabetes, metabolic syndrome, and obesity have been defined by the American Heart Association as "Life's Essential 8" [12]. Implementing prevention and treatment of these diseases and introducing changes to the patient's lifestyle related to following a healthy diet, avoiding smoking, regular physical activity, and ensuring adequate quality of sleep can significantly prevent the burden of CVD [12, 13].

Prevalence is one of the basic statistical methods for verifying the number of cases of a disease or the number of people with some other characteristic present during a given period [14]. In most European countries, the prevalence of obesity has increased since 2010 [15]. A particularly clear upward trend in the prevalence of obesity is visible in the countries of Eastern and Northern Europe. Although the prevalence of obesity varies from country to country, the predictions for the continent as a whole are alarming [15, 16]. The increase in the incidence of obesity strains healthcare resources and has negative economic consequences for the nations affected [16].

This study aimed to answer the question of how the prevalence of major ASCVD risk factors such as obesity, DM, HTN, and dyslipidemia change among adult primary health care patients consulted by Polish general practitioners for any reason in 2004, 2006, and 2015.

METHODS

Ethical considerations

The Bioethical Committee of the Polish Chamber of Physicians issued a positive opinion on the LIPIDOGRAM2004 and LIPIDOGRAM2006 study protocols (no. 51/2004/U), while the LIPIDOGRAM2015 study was accepted by the Bioethical Committee of the District Medical Chamber in Czestochowa (no. K.B.Cz.–0018/2015). All studies followed the Declaration of Helsinki. All participants provided written informed consent to participate in this study. Anonymity and confidentiality were ensured.

Study population

Three editions of the LIPIDOGRAM program were conducted in primary care centers in Poland. In 2006 and 2015, the

representativeness of the centers from the first edition in 2004 was maintained following the same methodology. All subsequent patients 18 years of age or older, consulting a general practitioner for any reason were eligible to participate. The number of recruited primary care patients in each voivodeship and the number of recruitment centers in Poland's 16 major administrative voivodeships was a direct function of each voivodeship's demographic size. In each edition, exclusion criteria included incomplete biochemical or clinical data, as well as dementia and/or mental illness resulting in the inability to provide informed consent. The methodology is described elsewhere [17–20].

Anthropometric measurement definitions of overall and central obesity

Healthcare staff recruiting patients for the LIPIDOGRAM study were responsible for collecting height, weight, and WC measurements. The measurements were performed on patients in underwear and barefoot. The detailed analysis methodology was presented in previous studies [17–19].

Biochemical analyses

Peripheral whole blood was used for biochemical analyses. Total cholesterol concentration in serum was determined using photometry. Triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) levels were measured using a homogeneous immune separation-based assay and a colorimetric glycerol-3-phosphate oxidase enzyme assay (DiaSys, Diagnostic Systems, Holzheim, Germany). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula (LIPIDOGRAM2004 and LIPIDOGRAM2006 studies) or measured directly (LIPIDOGRAM2015 study) [19]. For laboratory interpretation, both methods, Friedewald and direct, are equally valid, with the only limitation being that the Friedewald method loses linearity at TG concentrations >400 mg/dl. In the direct method, LDL concentration is always reported, because it is independent of TG concentration [21]. The detailed analysis methodology was presented in previous studies [17–19].

Definitions

Each patient was classified into the appropriate BMI category based on anthropometric measurements and mathematical calculations: underweight with BMI below 18.5 kg/m^2, normal weight with BMI 18.5 to 24.9 kg/m^2, overweight with BMI 25.0 to 29.9 kg/m^2, and individuals living with obesity with BMI above 30.0 kg/m^2 [6]. Additionally, obesity was categorized into three classes: class I obesity defined as a BMI from 30.0 to 34.9 kg/m^2, BMI between 35.0 to 39.9 kg/m^2 as class II obesity, and a BMI over 40.0 kg/m^2 classified as class III obesity or morbid obesity [10]. For further analyses, patients were classified into 3 groups based on BMI measurement: lean individuals with BMI <25.0 kg/m^2, overweight individuals with BMI 25.0–29.0 kg/m^2, and obese individuals with BMI ≥30.0 kg/m^2.

Central overweight was defined as a WC ≥94 cm for men and ≥80 cm for women; central obesity was defined as a WC above 102 cm for men and 88 cm for women [18].

Dyslipidemia was defined as: 1) previously diagnosed and medically documented dyslipidemia or lipid-lowering pharmacotherapy with statins of fibrates, or 2) on the basis of the result of a biochemical analysis performed as part of the ongoing LIPIDOGRAM study, when LDL-C level was above 2.97 mmol/l (115 mg/dl) and/or TG level was above 1.69 mmol/l (150 mg/dl) [22].

Statistical analysis

Continuous variables are presented as means and standard deviations. The Kruskal–Wallis test compared continuous variables across the three LIPIDOGRAM editions in 2004, 2006, and 2015. The dichotomous variables were compared using the χ^2 test and expressed in n (%). To ensure methodological consistency across time points, LDL-C was calculated for all 3 editions of the study (2004, 2006, and 2015) using the Martin-Hopkins equation. Hyperlipidemia was subsequently redefined using this harmonized LDL-C quantification measurement, which is now recommended in Polish (2021 and 2024) and international guidelines [21]. To analyze the actual changes in the prevalence of central obesity, diabetes, HTN, and dyslipidemia, standardization was carried out to the age of the general population for the years 2004, 2006, and 2015. Standardization was based on retrospective-year reference populations obtained from the Central Statistical Office in Poland. For each age group and sex, the expected number of cases of a given risk factor was calculated. Age- and sex-specific expected cases of each risk factor were calculated by multiplying the crude prevalence within each group by the respective population size. The standardized percentage of each risk factor occurrence was computed using the formula: (Σ expected cases / Σ population) × 100%. Missing data in specific subgroups (e.g., no cases for a risk factor in a particular group) were handled by including zero values to avoid population loss during standardization. Time trends were assessed using the Cochran–Armitage test for trends in BMI categories, central obesity categories, DM, HTN, and dyslipidemia, across the three study waves (2004, 2006, and 2015), both in the general population and in analyses stratified by sex. A value of 2-sided P <0.05 was considered significant, and all statistical analyses were performed using R, Version 3.4.3 (R Foundation for Statistical Computing, Vienna, Austria), with the following packages: dplyr for data manipulation, tidyr for handling missing values, gt for table generation, flextable for Word-compatible output, and ggplot2 for figures.

RESULTS

General clinical characteristics

The analysis included 45 591 Polish primary health care patients recruited from three editions of the LIPIDOGRAM

Table 1. Prevalence of obesity and cardiometabolic risk factors across LIPIDOGRAM 2004–2015

Category	2004, n = 16 577	2006, n = 15 427	2015, n = 13 587	*P*-value
Patient's sex				<0.001
Female	9910 (60%)	9620 (62%)	8614 (63%)	
Male	6667 (40%)	5807 (38%)	4973 (37%)	
BMI, kg/m^2	28.2 (4.7%)	28.4 (4.6%)	28.5 (5.1%)	<0.001
WC, cm	92 (14%)	93 (13%)	95 (14%)	<0.001
Age category				<0.001
18–34 years	445 (2.7%)	252 (1.6%)	1133 (8.3%)	
35–44 years	2367 (14%)	1825 (12%)	1729 (13%)	
45–54 years	5619 (34%)	4467 (29%)	2722 (20%)	
55–64 years	5093 (31%)	4844 (31%)	4270 (31%)	
65–74 years	2457 (15%)	3094 (20%)	2679 (20%)	
>75 years	596 (3.6%)	945 (6.1%)	1054 (7.8%)	
DM	1961 (12%)	1910 (12%)	1820 (13%)	<0.001
HTN	8411 (51%)	8389 (54%)	6702 (49%)	<0.001
Dyslipidemia	12606 (76%)	11 666 (76%)	9794 (72%)	0.703
Fibrate treatment	718 (4.3%)	417 (2.7%)	459 (3.4%)	<0.001
Statin treatment	4427 (27%)	4579 (30%)	4026 (30%)	<0.001
Central overweight/obesity				<0.001
Central obesity	6546 (39%)	6831 (44%)	6967 (51%)	<0.001
Central overweight	5285 (32%)	4939 (32%)	3775 (28%)	<0.001
No central obesity	4746 (29%)	3657 (24%)	2845 (21%)	<0.001
BMI categories				<0.001
Underweight	152 (0.9%)	71 (0.5%)	97 (0.7%)	0.496
Normal weight	4058 (24%)	3556 (23%)	3363 (25%)	0.105
Overweight	7146 (43%)	6629 (43%)	5349 (39%)	<0.001
Class 1 obese	3864 (23%)	3861 (25%)	3389 (25%)	0.015
Class 2 obese	1048 (6.3%)	1056 (6.8%)	1048 (7.7%)	< 0.001
Class 3 obese	309 (1.9%)	254 (1.6%)	341 (2.5%)	<0.001

Data are presented as n (%) or mean (SD)

Abbreviations: BMI, body mass index; DM, diabetes mellitus; HTN, hypertension; SD, standard deviation; WC, waist circumference

studies in 2004, 2006, and 2015 (Table 1). The mean age of the patients was 55 years (standard deviation 11 years). 24 942 (58%) patients had secondary or higher education. The majority, i.e., 23 640 (55%) lived in the city. 8402 (20%) of the recruited individuals were current smokers. In each study group, women comprised the majority of the analyzed groups: 9910 (60.0%) in 2004, 9620 (62.0%) in 2006, and 8614 (63.0%) in 2015. A small but significant increase in BMI was observed among individuals recruited between 2004 and 2015 (Table 1).

Among the LIPIDOGRAM 2004–2015 study population, the largest number of individuals with two (standardized percentage: 32.7% in 2004; 33.6% in 2006 and 31.2% in 2015) or one (standardized percentage: 28.3% in 2004; 27.1% in 2006 and 28.7% in 2015) risk factors for coronary heart disease was noted (Supplementary material, *Figure S1* and *Table S1*).

Analysis of obesity based on BMI in the 2004, 2006, and 2015 editions of the LIPIDOGRAM study

Over the years, overweight has declined (43% in 2004–2006 vs. 39% in 2015; P <0.001) at the expense of an increase in obesity in each class (class 1 obese: 23% in 2004 vs. 25% in 2006–2015 [P = 0.015]; class 2 obese: 6.3% in 2004 vs. 6.8% in 2006 vs. 7.7% in 2015 [P <0.001], and class 3 obese: 1.9% in 2004 vs. 1.6% in 2006 vs. 2.5% in 2015 [P <0.001]) (Supplementary material, *Figure S2*). Detailed data on the analyzed anthropometric data and cardiometabolic risk factors are presented in Table 1.

In the studied LIPIDOGRAM population of age-standardized BMI categories, we observed an increase in the prevalence of obesity from 26.8% in 2004 to 30.8% in 2015 (Supplementary material, *Table S2*). An increased prevalence of obesity was observed in class 1 (19.8 in 2004 and 22.0 in 2015) and in class 2 (5.0 in 2004 and 6.7 in 2015); P <0.001 (Table 2).

There were significant differences in the distribution of BMI categories (lean, overweight, and obese patients) in the 3 editions of the LIPIDOGRAM in 2004, 2006, and 2015. There were significant increases in the number of individuals living with obesity in the following age groups: 35–44 years (23% in 2004 vs. 25% in 2006 vs. 26% in 2015; P = 0.029); 55–64 years (36% in 2004 vs. 39% in 2006 vs. 39% in 2015; P <0.001); 65–74 years (33% in 2004 vs. 35% in 2006 vs. 44% in 2015; P <0.001); and patients above 75 years of age (30% in 2004 vs. 29% in 2006 vs. 36% in 2015; P = 0.006) (Figure 1). Detailed results regarding the unstandardized prevalence of BMI categories in 2004–2015 are presented in Supplementary material, *Table S3*.

It was also observed in the case of the LIPIDOGRAM 2004–2006 editions that the maximum number of cases

Table 2. Standardized prevalence estimates with estimated numbers by category across 2004, 2006, and 2015

	Total population			Estimated number of cases			Standardized prevalence (%)			*P*-value
	2004	2006	2015	2004	2006	2015	2004	2006	2015	
BMI categories										<0.001
Underweight	30 086 768	30 464 912	31 535 444	371 534	171 332	453 388	1.2	0.6	1.4	
Normal weight	30 086 768	30 464 912	31 535 444	9 612 990	8 989 441	9 259 118	32.0	29.5	29.4	
Overweight	30 086 768	30 464 912	31 535 444	12 024 592	12 531 799	12 109 820	40.0	41.1	38.4	
Class 1 obese	30 086 768	30 464 912	31 535 444	5 962 871	6 621 699	6 945 670	19.8	21.7	22.0	
Class 2 obese	30 086 768	30 464 912	31 535 444	1 514 262	1 685 879	2 123 900	5.0	5.5	6.7	
Class 3 obese	30 086 768	30 464 912	31 535 444	600 520	464 762	643 548	2.0	1.5	2.0	
Central overweight/obesity										<0.001
Central obesity	30 086 768	30 464 912	31 535 444	9 063 592	10 609 038	13 304 517	30.1	34.8	42.2	
Central overweight	30 086 768	30 464 912	31 535 444	8 714 232	9 874 725	8 881 318	29.0	32.4	28.2	
No central obesity	30 086 768	30 464 912	31 535 444	12 308 944	9 981 148	9 349 609	40.9	32.8	29.6	
DM	30 086 768	30 464 912	31 535 444	2 348 452	2 499 100	3 105 428	7.8	8.2	9.8	<0.001
Dyslipidemia	30 086 768	30 464 912	31 535 444	22 371 168	23 171 507	24 089 445	74.4	76.1	76.4	0.01
Fibrate treatment	30 086 768	30 464 912	31 535 444	973 784	756 032	986 007	3.2	2.5	3.1	<0.001
HTN	30 086 768	30 464 912	31 535 444	11 476 128	12 711 459	11 915 394	38.1	41.7	37.8	<0.001
Statin treatment	30 086 768	30 464 912	31 535 444	5 899 261	6 363 076	6 799 857	19.6	20.9	21.6	<0.001

Data are presented as n (%) or mean (SD)

Abbreviations: see Table 1

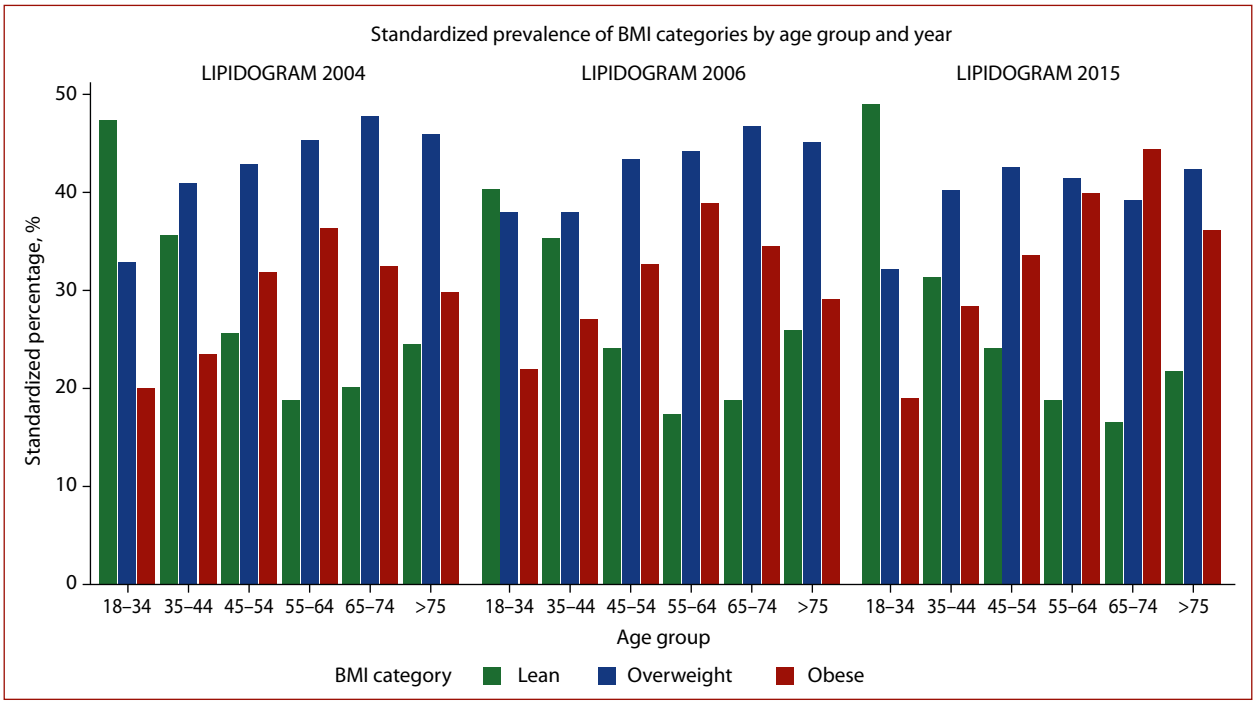


Figure 1. Standardized prevalence of body mass index categories by age group and year

of class 1, 2, and 3 obesity occurred among Polish primary health care patients in the 55–64 age group, while in the case of the LIPIDOGRAM 2015 edition it was recorded in the 65–74 age category (Supplementary material, *Figure S3*).

We observed reductions in the number of women over 2004–2015 in the overweight category according to BMI (39% in 2004 vs. 36% in 2015; P <0.001) and among men (48% in 2004 vs. 45% in 2015; P <0.001). Among both men and women, there was an increase in the prevalence of obesity in each class according to BMI assessment during the period 2004–2015. However, among those recruited over the period under study, there was an increase in women in the BMI category defined as normal weight (28% in 2004 vs. 30% in 2015; P = 0.001). Among men, there was a decrease in the number of individuals in this BMI category (19% in 2004 vs. 16% in 2015; P = 0.001) (Table 3).

Analysis of central obesity based on waist circumference in the 2004, 2006, and 2015 editions of the LIPIDOGRAM study

An increase in abdominal obesity was also observed in the analyzed groups, from 39% of individuals recruited in 2004 to 51% of primary health care patients recruited in 2015 (P <0.001). Over the 3 editions of the LIPIDOGRAM

Table 3. Prevalence of obesity and cardiometabolic risk factors across LIPIDOGRAM 2004–2015 in men and women subgroups

Category	All 45 591 (100%)							
	Men 17 447 (38.3%)				Women 28 144 (61.7%)			
	2004 n = 6667[a]	2006 n = 5807[a]	2015 n = 4973[a]	*P*-value[b]	2004 n = 9910[a]	2006 n = 9620[a]	2015 n = 8614[a]	*P*-value[b]
BMI, kg/m^2	28.5	28.8	29.1	<0.001	27.9	28.2	28.1	0.001
WC, cm	97	98	101	<0.001	89	90	91	<0.001
Age category				<0.001				<0.001
18–34 years	197 (3.0%)	108 (1.9%)	437 (8.8%)		248 (2.5%)	144 (1.5%)	696 (8.1%)	
35–44 years	1031 (15%)	732 (13%)	619 (12%)		1336 (13%)	1093 (11%)	1110 (13%)	
45–54 years	2332 (35%)	1761 (30%)	967 (19%)		3287 (33%)	2706 (28%)	1755 (20%)	
55–64 years	1994 (30%)	1796 (31%)	1541 (31%)		3099 (31%)	3048 (32%)	2729 (32%)	
65–74 years	911 (14%)	1090 (19%)	1048 (21%)		1546 (16%)	2004 (21%)	1631 (19%)	
>75 years	202 (3.0%)	320 (5.5%)	361 (7.3%)		394 (4.0%)	625 (6.5%)	693 (8.0%)	
DM	852 (13%)	809 (14%)	814 (16%)	<0.001	1109 (11%)	1101 (11%)	1006 (12%)	0.334
HTN	3,373 (51%)	3166 (55%)	2611 (53%)	0.370	5038 (51%)	5223 (54%)	4091 (47%)	<0.001
Dyslipidemia	5,175 (78%)	4477 (77%)	3758 (76%)	<0.001	7431 (75%)	7189 (75%)	6009 (70%)	0.007
Fibrate treatment	311 (4.7%)	196 (3.4%)	247 (5.0%)	<0.001	407 (4.1%)	221 (2.3%)	212 (2.5%)	<0.001
Statin treatment	1846 (28%)	1714 (30%)	1608 (32%)	<0.001	2581 (26%)	2865 (30%)	2418 (28%)	<0.001
Central overweight/obesity				<0.001				<0.001
Central obesity	1793 (27%)	1790 (31%)	2096 (42%)	<0.001	4753 (48%)	5041 (52%)	4871 (57%)	<0.001
Central overweight	2424 (36%)	2191 (38%)	1665 (33%)	<0.001	2861 (29%)	2748 (29%)	2110 (24%)	<0.001
No central obesity	2450 (37%)	1826 (31%)	1212 (24%)	<0.001	2296 (23%)	1831 (19%)	1633 (19%)	<0.001
BMI categories				<0.001				<0.001
Underweight	31 (0.5%)	14 (0.2%)	16 (0.3%)	0.439	121 (1.2%)	57 (0.6%)	81 (0.9%)	0.577
Normal weight	1263 (19%)	993 (17%)	811 (16%)	0.001	2795 (28%)	2563 (27%)	2552 (30%)	0.001
Overweight	3232 (48%)	2762 (48%)	2223 (45%)	<0.001	3914 (39%)	3867 (40%)	3126 (36%)	<0.001
Class 1 obese	1678 (25%)	1601 (28%)	1439 (29%)	<0.001	2186 (22%)	2260 (23%)	1950 (23%)	0.847
Class 2 obese	362 (5.4%)	359 (6.2%)	386 (7.8%)	<0.001	686 (6.9%)	697 (7.2%)	662 (7.7%)	0.054
Class 3 obese	101 (1.5%)	78 (1.3%)	98 (2.0%)	0.018	208 (2.1%)	176 (1.8%)	243 (2.8%)	<0.001

Data are presented as n (%) or mean (SD)

[a]Number of participants included in the analysis in a given edition of the LIPIDOGRAM study. [b]*P*-value for the comparison across the three editions of the LIPIDOGRAM study (2004, 2006, and 2015)

Abbreviations: see Table 1

study, a decrease in the number of central overweight individuals (32% in 2004 vs. 28% in 2015; *P* <0.001) and those without central obesity (29% in 2004 vs. 21% in 2015; *P* <0.001) was also observed (Table 1; Supplementary material, *Figure S4*).

After standardization a decrease in the prevalence of central overweight (29.0 in 2004 and 28.2 in 2015) and no central obesity (40.9 in 2004 and 29.6 in 2015) was observed (Table 2). In the LIPIDOGRAM population we observed an increase in the age standardized central obesity prevalence from 30.1% in 2004 to 42.2% in 2015. Detailed data on the analyzed age standardized central obesity prevalence with absolute differences and ratios are presented in Supplementary material, *Table S4*. There was a significant difference in the distribution of central obesity, central overweight, and no central obesity patients in the 3 editions of the LIPIDOGRAM in 2004, 2006, and 2015 in each group categories (Supplementary material, *Table S5*). In the analyzed 2004–2015 period there was an increase in the number of patients with central obesity at the expense of central obesity and non-obese individuals in all age categories: 18–34 years (central obesity 16% in 2004 vs. 24% in 2015; *P* = 0.001); 35–44 years (central obesity 23% in 2004 vs. 37% in 2015; *P* <0.001); 45–54 years (central obesity 35% in 2004 vs. 43% in 2015; *P* <0.001); 55–64 years (central obesity 47% in 2004 vs. 57% in 2015; *P* <0.001); 65–74 years (central obesity 51% in 2004 vs. 65% in 2015; *P* <0.001); and above 75 years (central obesity 52% in 2004 vs. 65% in 2015; *P* <0.001) (Figure 2; Supplementary material, *Table S5*).

There has been a significant increase in the number of men (27% in 2004 vs. 42% in 2015; *P* <0.001) over the years, as well as women (48% in 2004 vs. 57% in 2015; *P* <0.001) with abdominal obesity, and a significant decrease in the percentage of men (37% in 2004 vs. 24% in 2015; *P* <0.001) and women (23% in 2004 vs. 19% in 2015; *P* <0.001) without central obesity (Table 3).

Analysis of the frequency of diabetes mellitus in the 2004, 2006, and 2015 editions of the LIPIDOGRAM study

The cases of diagnosed DM increased among patients recruited in 2004–2015 (12% in 2004 vs. 13% in 2015; *P* <0.001) (Table 1). A similar trend was also observed in the subgroup analysis of men (13% in 2004 vs. 16% in 2015; *P* <0.001) (Table 3). In the case of diabetes, a greater number of recorded cases of diabetes were observed among patients recruited in the 2015 study after the age of 64 years compared with 2004 and 2006 (Figure 3).

In the LIPIDOGRAM population we observed an increase in the age-standardized DM prevalence, which was

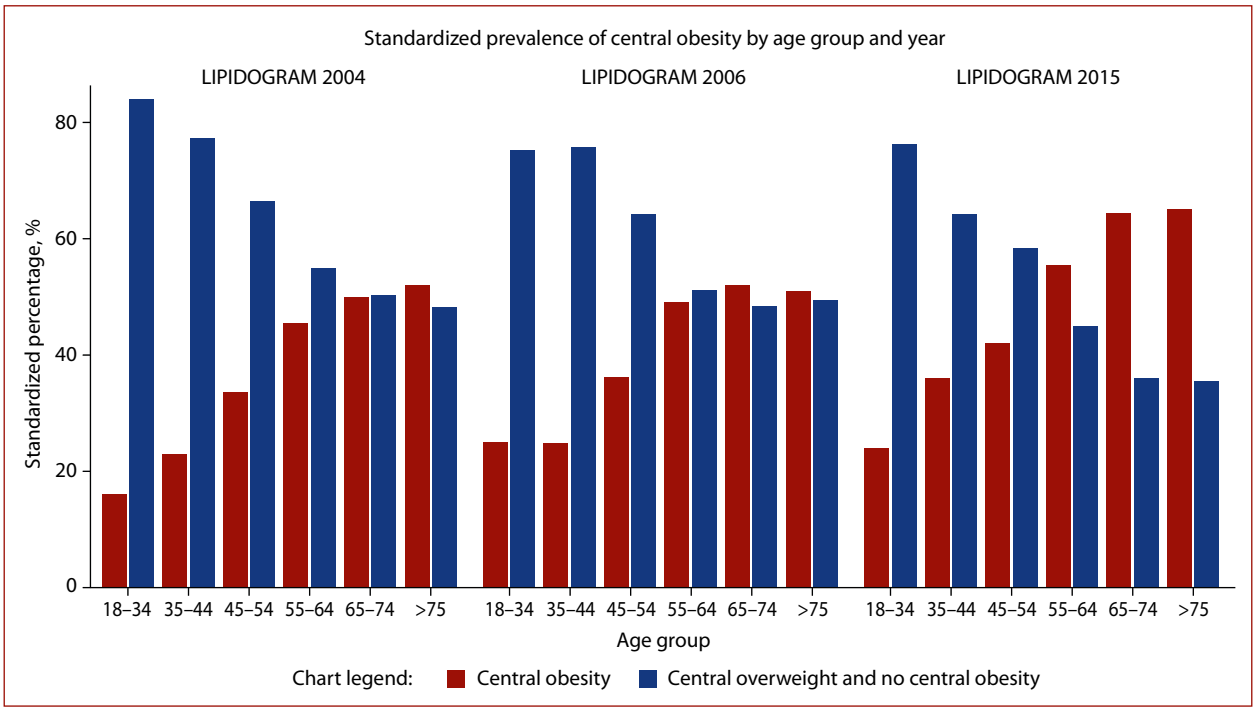


Figure 2. Standardized prevalence of central obesity by age and year

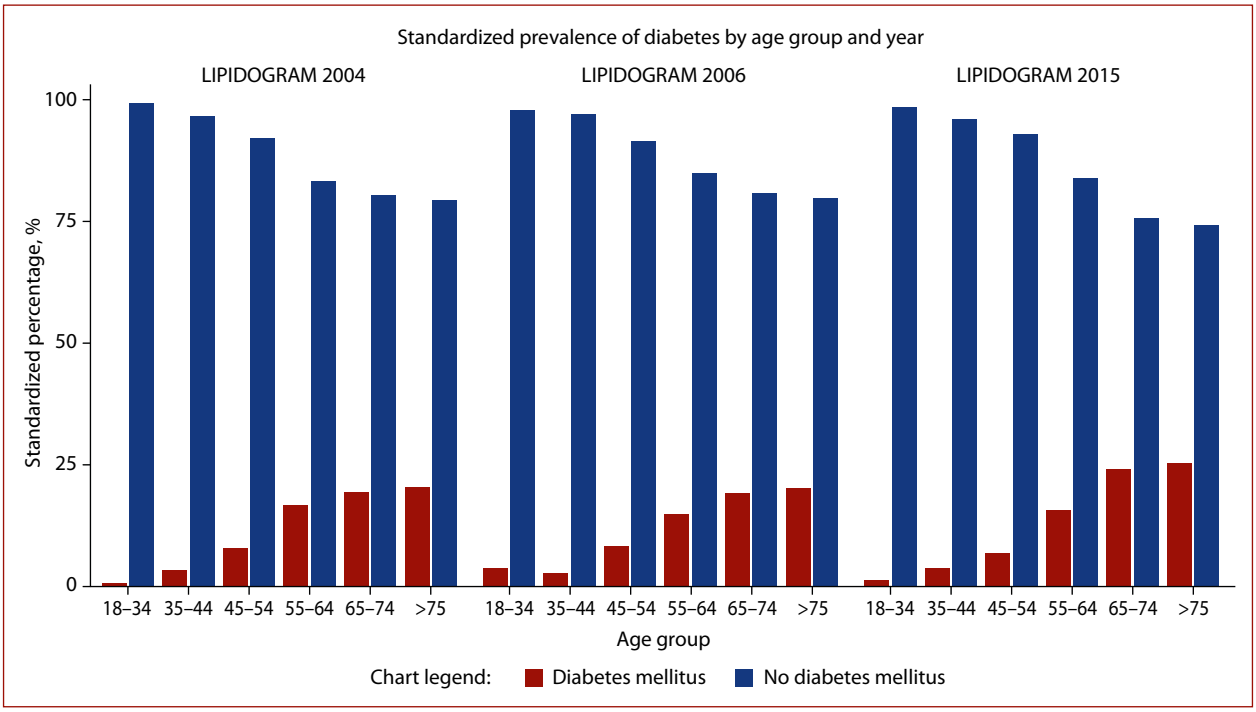


Figure 3. Standardized prevalence of diabetes mellitus by age group and year

7.8% in 2004, 8.2% in 2006, and 9.8% in 2015 with a prevalence ratio in 2004–2015 of 1.26 and absolute difference 2.04 (Supplementary material, *Table S6*). A reduction in the unstandardized prevalence of DM was observed between 2004 and 2015 in patients aged 45–54 years ($P = 0.031$) and 55–64 years ($P = 0.039$). In contrast, the incidence of DM increased during the same period among patients aged 65–74 ($P < 0.001$) and those aged 75 years and older ($P = 0.001$) in the 3 editions of the LIPIDOGRAM study (Supplementary material, *Table S7*).

Analysis of the frequency of hypertension in the 2004, 2006, and 2015 editions of the LIPIDOGRAM study

In the entire study population, a decrease in the number of primary health care patients with HTN was observed

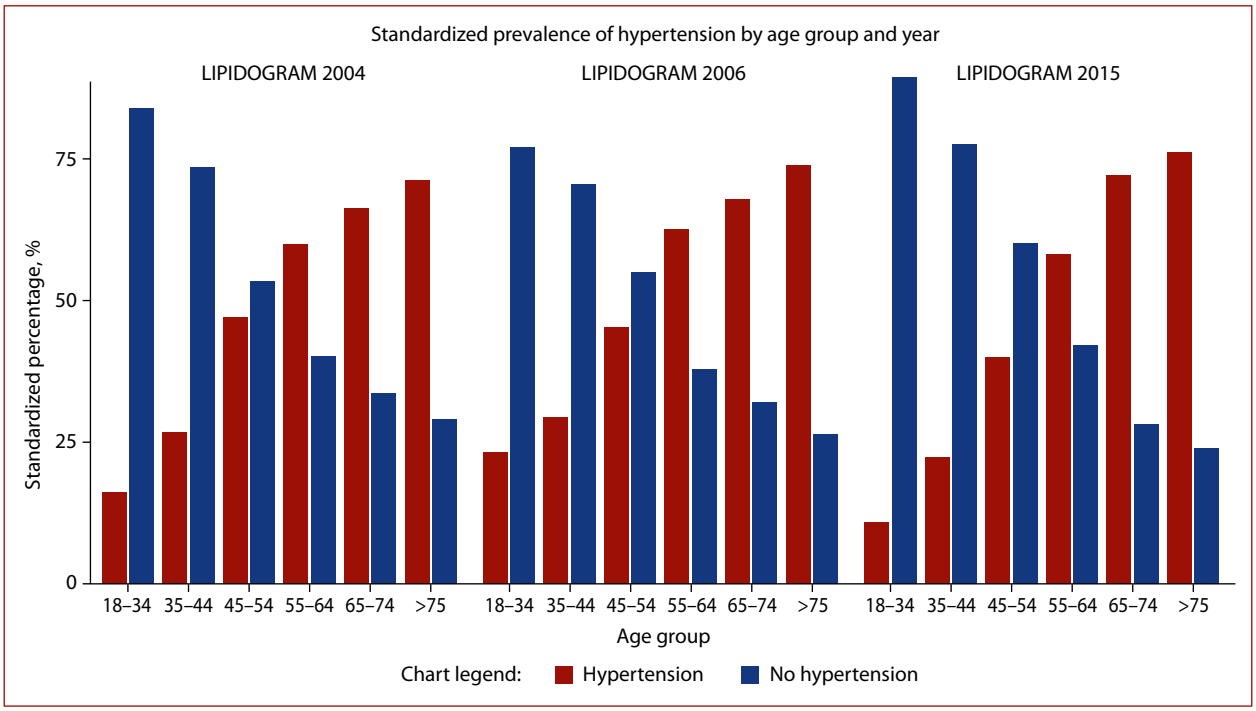


Figure 4. Standardized prevalence of hypertension by age group and year

between 2004 and 2015 (51% in 2004 vs. 54% in 2006 vs. 49% in 2015; P <0.001) (Table 1). In the case of the analysis of the entire population, which considered the division into age groups, an increase of HTN with age was observed in every LIPIDOGRAM edition (Figure 4).

In the LIPIDOGRAM study population, changes in the age-standardized prevalence of HTN were observed, which was 38.1% in 2004, 41.7% in 2006, and 37.8% in 2015, with the prevalence ratio in 2004–2015 being 0.99 and absolute difference 0.36 (Supplementary material, *Table S8*). A reduction of HTN was observed between 2004 and 2015 among patients aged 18–64 years (P <0.001). Additionally, the incidence of HTN increased significantly among patients aged 65–74 (P <0.001) and over 75 years (P = 0.048) in the studied editions of the LIPIDOGRAM program (Supplementary material, *Table S9*).

Analysis of the frequency of dyslipidemia in the 2004, 2006, and 2015 editions of the LIPIDOGRAM study

The number of recorded cases of dyslipidemia among patients recruited between 2004 and 2015 decreased from 76% in 2004 to 72% in 2015 (Table 1). A similar trend was also observed in the analysis of subgroups of men (78% in 2004 vs. 76% in 2015; P <0.001) and women (75% in 2004 vs. 70% in 2015; P = 0.007) (Table 3). The collected data show that the largest number of individuals with dyslipidemia in the LIPIDOGRAM 2004 edition was observed in the 55–64 age category (Figure 5).

In the LIPIDOGRAM population we observed an increase in the age standardized dyslipidemia prevalence from 74.4% in 2004 to 76.4% in 2015 with a prevalence ratio 2004–2015 of 1.03 and absolute difference 2.03 (Supplementary material, *Table S10*). An increase in the incidence of dyslipidemia was observed between 2004 and 2015 among patients aged 45–54 years (P = 0.005) and 55–64 years (<0.001) (Supplementary material, *Table S11*). In the study population, an increase in statin use was observed over the period 2004–2015 and a reduction in individuals with high LDL-C, TG, or both (Supplementary material, *Table S12*).

DISCUSSION

In this observational study, we conducted 3 cross-sectional analyses of patients attending primary health care offices in Poland in 2004, 2006, and 2015 (Graphical abstract). We observed an increased prevalence of obesity from 2004 to 2015. The percentage of primary health care patients with normal body weight increased, while the percentage of overweight patients decreased at the expense of obesity. The increase in patients with obesity occurred in every analyzed class. The highest percentage of obese patients was observed in the 55–74 age group and the lowest in the 18–34 age group. The number of patients with diabetes increased, with the greatest change occurring in older age groups. We observed decreased prevalence of HTN from 2004 to 2015. In addition, our study found an increase in the prevalence of dyslipidemia in primary care patients. All these changes, although significant, were small.

Cardiometabolic diseases, which include CVDs, dyslipidemia, HTN, and DM, are among the most important causes of morbidity and mortality worldwide. The incidence of cardiometabolic disorders has increased significantly in recent years, posing a significant burden on healthcare systems worldwide [23].

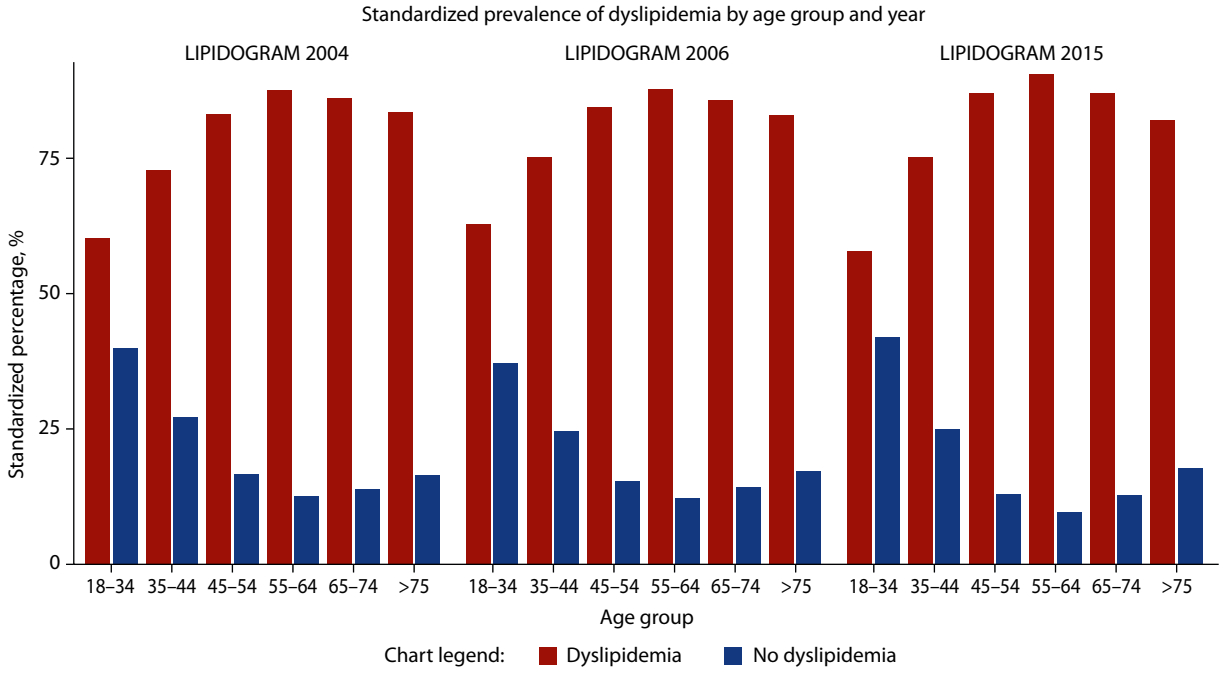
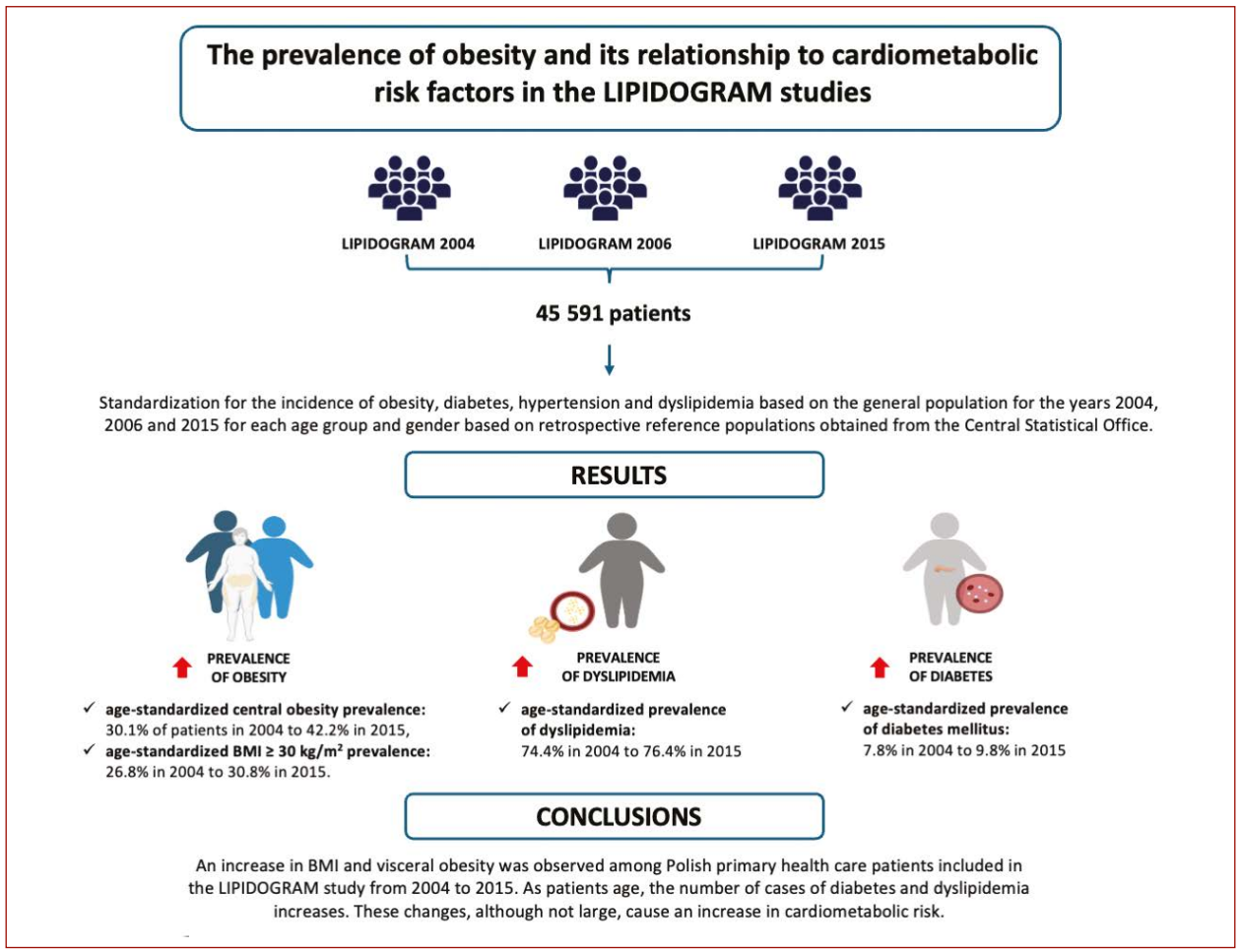


Figure 5. Standardized prevalence of dyslipidemia by age group and year



Graphical abstract

Abbreviation: BMI, body mass index

Obesity is an important risk factor for CVDs, and BMI may be a prognostic factor for these diseases [24]. However, it should be noted that BMI only reflects total body weight and not the distribution of fat tissue or the share of fat in body composition [25]. LIPIDOGRAM studies conducted in 2004, 2006, and 2015 on the general population show a slight increase in BMI and WC; the same applies to DM and the frequency of statin treatment. The percentage of lean individuals decreased (the highest percentage in the 18–34 age group in each study, the lowest in the 55–64 age group in 2004 and 2006, and 65–74 in 2015). The percentage of overweight individuals increased, with the highest percentage in the age group 65–74 in 2004 and 2006, and over 75 years in 2015. At the same time, the percentage of individuals living with central obesity increased in population. Additionally, pan-European studies show an increase in the percentage of overweight and obese individuals, particularly in Eastern European countries, with particular emphasis on men and individuals over 65 years of age [26, 27]. At the same time, two multi-center studies, WOBASZ and WOBASZ II, were conducted in Poland. These nationwide, cross-sectional studies sampled residents randomly from 16 Polish voivodeships: 13 545 individuals recruited in 2003–2005 [28], and 6170 in 2013–2014 [29]. Our analysis includes results based on 16 577 primary health care patients recruited in 2004, 15 427 in 2006, and 13 587 in 2015. Comparing WOBASZ and WOBASZ II, researchers observed an upward shift in body-weight distribution by BMI category and measured increases in the prevalence of overall and abdominal obesity between the first WOBASZ (2003–2005) and WOBASZ II (2013–2014). BMI increased for both men and women; the prevalence of overweight and obesity rose in both sexes. In men, increases occurred across all obesity classes; in women, increases were seen in classes 1 and 2, while class 3 showed a slight decrease. The percentage of men with normal body weight declined, whereas changes among women were nonsignificant [30]. In our study, we observed a decline in the prevalence of non–age-standardized overweight in both men and women, alongside increases in obesity classes 1, 2, and 3 for both sexes between 2004 and 2015. The number of men with normal weight decreased, while the number of women with normal weight increased. Differences between WOBASZ and LIPIDOGRAM findings may reflect sample-size differences and methodological choices: in LIPIDOGRAM we standardized overall and abdominal obesity to the combined populations of the three programs, but we did not standardize overweight and obesity by age group or sex, which may account for some discrepancies despite comparing populations recruited in the same years. Waist circumference and abdominal obesity increased in the entire LIPIDOGRAM sample and in both sex-specific subgroups. Conversely, the prevalence of abdominal overweight decreased over time in the overall LIPIDOGRAM population and in both men and women. WOBASZ and WOBASZ II similarly reported increases in abdominal obesity in men and women [30]. Additionally, central obesity occurred more often in women than in men. The observed differences in abdominal obesity between women and men contradict previous research that suggested that men tend to have more visceral fat (VAT), while women tend to have more subcutaneous fat [31]. These differences in adipose tissue distribution may be partially hormonally determined [32]. With a similar BMI, women may have a higher total fat mass compared to men, who, in turn, tend to accumulate larger amounts of VAT and muscle tissue. VAT may increase cardiometabolic risk [33]. Free fatty acids from VAT in the portal circulation support very low-density lipoprotein (VLDL) synthesis and increase serum TG concentrations [34]. Additionally, subcutaneous adipose tissue is associated with less inflammation and can absorb circulating free fatty acids and TGs, which may reduce the cardiometabolic risk associated with obesity [35]. Schorr et al. [33] found that, considering similar age and BMI, men exhibited a more unfavorable cardiometabolic risk profile compared to women. This profile was also associated with disturbances in serum lipid profiles (higher TG and ApoB/LDL levels and lower mean HDL-C levels), glucose homeostasis (higher HOMA-IR and mean fasting insulin levels), and higher fibrinogen levels, compared to women. In their study, the researchers indicated that VAT was associated with these cardiometabolic disturbances [33]. It is important to note that women are less likely to experience cardiometabolic disturbances before menopause because estrogens improve insulin sensitivity, increase LDL clearance, and are associated with promoting gynoid adipose tissue distribution. The reduction in sex hormone levels associated with menopause is associated with an increased risk of developing DM, dyslipidemia, and HTN [32, 33, 36].

In our study cardiometabolic risk factors, such as diabetes, HTN, and dyslipidemia, were more common among men compared with women. Perhaps this observation results from the smaller number of men recruited in the LIPIDOGRAM studies. Furthermore, the observed differences between men and women may be due to differences in biological factors, lifestyle, and approach to using primary health care [36]. Men are more likely to use stimulants such as tobacco [37] and alcohol [38]. Women, on the other hand, are at greater risk of obesity after menopause, which may also be related to hormonal changes and reduced physical activity. Additionally, men are less likely to use preventive healthcare than women, which contributes to the later diagnosis of cardiometabolic disorders [36]. These epidemiological differences may contribute to the earlier development of obesity, HTN, and dyslipidemia. Consideration of these differences is crucial for the prevention, diagnosis, and personalized treatment of metabolic diseases in women and men.

Comparing the results from LIPIDOGRAM 2004 with the results from 2015, we detected an increase in the prevalence of obesity as well as DM. The relationship between diabetes and obesity is well documented [39]. One of the most common metabolic disorders accompanying

obesity is hyperinsulinemia and insulin resistance, which may also lead to the development of dyslipidemia [40]. The literature describes a characteristic triad of lipid disorders in individuals with CMS and diabetes, characterized by high levels of TG and LDL-C, and a low level of HDL-C. These changes define atherogenic dyslipidemia and are an important factor leading to the development of ASCVD [1]. Insulin resistance is associated with increased lipolysis of TG in adipose tissue, which releases excess free fatty acids and leads to increased hepatic production and secretion of VLDL, resulting in hypertriglyceridemia and low HDL-C [41]. Postprandial lipemia may also lead to changes in the blood lipid profile, which may promote increased insulin resistance and be associated with increased oxidative stress and progress of endothelial dysfunction [42]. Postprandial lipemia is associated with an increase in TG-rich lipoproteins (TRL) [43]. TRL includes VLDL and chylomicrons, and their remnants. VLDL and their remnants are responsible for transporting TG in plasma during fasting, and chylomicrons and their remnants after a meal. Increased TRL blood levels, as well as low HDL-C levels, are associated with the occurrence of the so-called residual risk of CV events in some individuals despite achieving their target LDL-C levels [44]. In Western society this is an important element in determining the true "TG level status", which allows for a more accurate assessment of the risk of CV events and enables selection of optimal therapy for any given patient [42].

Christian et al. [45] showed that the association between BMI, WC, and major cardiometabolic risk factors varied by ethnicity and race. Selected anthropometric indicators, such as BMI or WC, may help assess cardiometabolic risk in specific subpopulations. Individuals with elevated WC and BMI may be significantly more likely to develop HTN, increased glucose, TGs, and decreased HDL-C levels compared with individuals with WC and BMI values within the normal range. Additionally, HTN was more frequently observed in individuals with both WC and BMI above the accepted levels compared with those with normal values [45]. In the study by Mohseni et al. [46], the risk of HTN was also associated with obesity and dyslipidemia. In our study, the prevalence of HTN among respondents by age group shows an age-related increase in the prevalence of HTN, with the lowest prevalence falling in the age group 18–34, while the highest prevalence occurs in the 65–74 and over 75 groups. In an American study analyzing trends in the incidence of HTN during 1999–2014 in individual age groups, a similar distribution of HTN rates was observed, with a peak at age 60+. Still, in that population, the rates of HTN incidence have remained constant over the years [47]. Our study indicates a slight decrease in the incidence of age-standardized HTN despite increases in the prevalence of other metabolic syndrome factors, such as BMI, abdominal obesity, and DM. In the present study, an increase in the prevalence of HTN with age was observed in each edition of the LIPIDOGRAM study. The percentage of patients with HTN over 65 years of age increased between 2004 and 2015. Furthermore, a 0.3% decrease in the prevalence of standardized HTN was observed between 2004 and 2015. The slight decrease in the prevalence of standardized HTN in the studied patient populations may be related to the effect of repeated testing in primary care facilities. Perhaps this can be treated as a kind of preventive and educational effect, where the dissemination of knowledge about CV risk factors and methods of their measurement in the same places for many years probably has a noticeable positive impact in the case of hypertension - a factor that can be measured in the simplest and most unlimited way.

Analyzing the general population of the LIPIDOGRAM program over the period from 2004 to 2015, a decrease in the number of cases of lipid disorders was observed. The same trend was observed in male and female groups. An increase in the age-standardized prevalence of dyslipidemia was observed between 2004 and 2015. Additionally, in the studied population, the lowest percentage of individuals with dyslipidemia was recorded in the of 18–34 age group in each edition of the LIPIDOGRAM program. It can be observed that the number of cases in each edition increased up to the age category of 55–64. The Polish cross-sectional study NATPOL 2011 showed hypercholesterolemia among 61.1% of the examined adults, including 60.8% of the examined men and 61.3% of the examined women, and in the individuals aged 40–59 years the highest incidence of elevated total cholesterol and LDL-C levels was observed, which constituted 71.6% of the examined [48]. The WOBASZ II study showed that 70% of the male population in the 35–44-year-old group had hypercholesterolemia. In women in the same age group, hypercholesterolemia occurred in 50% of people, and it peaked in the 55-year-old age group; then, a decrease in the percentage of hypercholesterolemia was observed with age 64 years [49]. With age, changes in adipose tissue distribution occur, which, unfortunately, are associated with an increase in the incidence of diseases typical of old age [50].

Strengths and limitations

The main strength of the presented study is the inclusion in the analyses of a large number of patients recruited from all 16 regions of Poland, which makes the population representative of the adult population using primary health care for various reasons. Additionally, biochemical tests were performed in one central laboratory in accordance with applicable quality control standards, and anthropometric measurements were performed by qualified medical personnel. Additionally, complete and statistically verified patient records containing the same variables were used in this publication. No missing data were found for variables included in the primary analyses (Supplementary material, *Table S13*).

The main limitation of the study is that it only includes the Polish primary health care patient population, which may create limitations when relating our observations to

other studies concerning other populations. Recruited individuals receiving primary care treatment potentially represent a group of individuals more vulnerable to health problems, which may be associated with the risk of selection bias. An additional limitation is the fact that we did not include in the manuscript information on the quantity and quality of meals consumed or information on the diet followed by the participants of our study. The study is also limited by impossibility performing a wider range of laboratory tests (due to financial and technical restrictions). For example, we did not perform a lipoprotein A test, which would have provided more information about risk factors.

Implications for research, clinical practice, education, and policy

The analysis of data collected within three editions of the LIPIDOGRAM program over the years 2004–2015 in the context of a population approach can serve as a basic instrument for assessing the occurrence and progression of obesity in the Polish primary health care patient population, taking into account the age of individuals. The conducted research may serve to develop new guidelines on cardiometabolic risk for the Polish and Eastern European primary health care patient population. Drawing doctors' attention to the high prevalence of obesity and the occurrence of diabetes, lipid disorders, and hypertension will create an opportunity to improve the quality of care in the area of primary and secondary prevention of CVDs in the Polish population.

CONCLUSIONS

An increase in BMI and visceral obesity was observed among Polish primary health care patients included in the LIPIDOGRAM study from 2004 to 2015. As patients age, the number of cases of diabetes increases. These changes cause an increase in cardiometabolic risk. The actions of primary care physicians, health politicians, and people managing prevention in Polish health care should take into account the results of this study.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/polish_heart_journal.

Article information

Acknowledgements: We would like to thank all volunteer LIPIDOGRAM 2004–2015 study investigators, all volunteer staff, and all participants.

Conflict of interest: TT has received fees from Bioferm, Adamed, Boehringer, Astra Zeneca, Novo-Nordisk, and Teva. AW has received consulting or speaking fees from Merck, Sanofi, Novo Nordisk, Astra Zeneca. MG has received consulting or speaking fees from: Novartis, Sanofi, Amgen, NovoNordisk, Bayer, AstraZeneca, Sandoz, Berlin-Chemie, has served on a Data Safety Monitoring Board or Advisory Board for: Novartis, Amgen; has held an officer or trustee position in: Polish Cardiac Society. PPT has received honoraria from Eli Lilly Pharmaceuticals for lectures, presentations, speakers' bureaus, and participation in educational events. PEP holds shares in AstraZeneca PLC; has received financial support for travel expenses to participate in the International Lipid Expert Panel (2024, Lodz; 2025, Warsaw) and the EU-RO Forum: Diabetes (2025, Bucharest). MB served on the speakers' bureau for: Amgen, Adamed, Daiichi Sankyo, Esperion, Kogen, KRKA, Lilly, MSD, Exceed Orphan, KRKA, Polpharma, Mylan, Novartis, Novo-Nordisk, Pfizer, Sanofi-Regeneron, Teva, Zentiva, Servier; consulted for Adamed, Amgen, Daiichi Sankyo, Esperion, NewAmsterdam, Novartis, NovoNordisk, Sanofi-Regeneron, Teva; Grants from Amgen, Daiichi Sankyo, Viatris, Sanofi, Mylan/Viatris, and Valeant. JJ has received honoraria for lectures, presentations, speakers' bureaus, educational events from: Novartis, Adamed, Amgen, Boehringer Ingelheim, Servier, Novo-Nordisk. Other authors declare no conflict of interest.

Data availability: The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of artificial intelligence use: During the preparation of this work the authors declare to use R, Version 3.4.3 (R Foundation for Statistical Computing, Vienna, Austria). The authors take full responsibility for the content after using this tool.

Funding: The authors disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: The Polish Lipid Association (PoLA) and the College of Family Physician in Poland (CFPIP) provided non-material support by endorsing the project. The present study was funded by an unrestricted educational grant from Valeant (Warsaw, Poland). Valeant had no role in the study design, data analysis, data interpretation, or writing of the report. The Medical University of Silesia in Katowice participated as part of the implementation of statutory agreements: PCN-1-211/N/2/K and PCN-1-113/N/1I.

REFERENCES

1. Hsiao YC, Wu CC. Dyslipidemia and cardiometabolic syndrome. Cardiometab Syndr J. 2021; 1(1): 18–23, doi: 10.51789/cmsj.2021.1.e2.
2. Teo KK, Rafiq T. Cardiovascular risk factors and prevention: A perspective from developing countries. Can J Cardiol. 2021; 37(5): 733–743, doi: 10.1016/j.cjca.2021.02.009, indexed in Pubmed: 33610690.
3. Cardiovascular diseases statistics – Statistics Explained. https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Cardiovascular_diseases_statistics (accessed: January 10, 2025).
4. Banach M, Fronczek M, Osadnik T, et al. Risk of adverse cardiovascular events based on common genetic variants in 8-year follow-up of the LIPIDOGEN2015 population using the polygenic risk score (PRS): Study design and methodology. Arch Med Sci. 2024; 20(5): 1452–1460, doi: 10.5114/aoms/192147, indexed in Pubmed: 39649254.
5. Sun J, Qiao Y, Zhao M, et al. Global, regional, and national burden of cardiovascular diseases in youths and young adults aged 15–39 years in 204 countries/territories, 1990–2019: A systematic analysis of Global Burden of Disease Study 2019. BMC Med. 2023; 21(1): 222, doi: 10.1186/s12916-023-02925-4, indexed in Pubmed: 37365627.
6. Purnell JQ. Definitions, classification, and epidemiology of obesity South Dartmouth (MA), 2023.
7. Chen Y, Zhong Z, Gue Y, et al. Impact of surrogates for insulin resistance on mortality and life expectancy in primary care: A nationwide cross-sectional study with registry linkage (LIPIDOGRAM2015). Lancet Reg Health Eur. 2025; 49: 101182, doi: 10.1016/j.lanepe.2024.101182, indexed in Pubmed: 39759579.
8. Dobrowolski P, Prejbisz A, Kuryłowicz A, et al. Metabolic syndrome — a new definition and management guidelines: A joint position paper by the Polish Society of Hypertension, Polish Society for the Treatment of Obesity, Polish Lipid Association, Polish Association for Study of Liver, Polish Society of Family Medicine, Polish Society of Lifestyle Medicine, Division of Prevention and Epidemiology Polish Cardiac Society, "Club

30" Polish Cardiac Society, and Division of Metabolic and Bariatric Surgery Society of Polish Surgeons. Arch Med Sci. 2022; 18(5): 1133–1156, doi: 10.5114/aoms/152921, indexed in Pubmed: 36160355.
9. Powell-Wiley TM, Poirier P, Burke LE, et al. Obesity and cardiovascular disease: A scientific statement from the American Heart Association. Circulation. 2021; 143(21): e984–e1010, doi: 10.1161/CIR.0000000000000973, indexed in Pubmed: 33882682.
10. Weir CB, Weir CB, Jan A. BMI classification percentile and cut off points 2023, Treasure Island (FL).
11. Valenzuela PL, Carrera-Bastos P, Castillo-García A, et al. Obesity and the risk of cardiometabolic diseases. Nat Rev Cardiol. 2023; 20(7): 475–494, doi: 10.1038/s41569-023-00847-5, indexed in Pubmed: 36927772.
12. Lloyd-Jones DM, Allen NB, Anderson CAM, et al. Life's essential 8: Updating and enhancing the American Heart Association's construct of cardiovascular health: A presidential advisory from the American Heart Association. Circulation. 2022; 146(5): e18–e43, doi: 10.1161/CIR.0000000000001078, indexed in Pubmed: 35766027.
13. Banach M, Toth PP, Bielecka-Dąbrowa A, et al. Primary and secondary cardiovascular prevention: Recent advances. Pol Heart J. 2024; 82(12): 1200–1210, doi: 10.33963/v.phj.103997, indexed in Pubmed: 39775556.
14. Centers for Disease Control and Prevention. Prevalence – Health, United States. https://www.cdc.gov/nchs/hus/sources-definitions/prevalence.htm (accessed: January 10, 2025).
15. Stival C, Lugo A, Odone A, et al. Prevalence and correlates of overweight and obesity in 12 European countries in 2017–2018. Obes Facts. 2022; 15(5): 655–665, doi: 10.1159/000525792, indexed in Pubmed: 35917801.
16. Pineda E, Sanchez-Romero LM, Brown M, et al. Forecasting future trends in obesity across Europe: The value of improving surveillance. Obes Facts. 2018; 11(5): 360–371, doi: 10.1159/000492115, indexed in Pubmed: 30308509.
17. Osadnik T, Nowak D, Osadnik K, et al. Association of body mass index and long-term mortality in patients from nationwide LIPIDOGRAM 2004–2015 cohort studies: No obesity paradox? Cardiovasc Diabetol. 2023; 22(1): 323, doi: 10.1186/s12933-023-02059-0, indexed in Pubmed: 38017465.
18. Osadnik K, Osadnik T, Gierlotka M, et al. Metabolic syndrome is associated with similar long-term prognosis in those living with and without obesity: an analysis of 45 615 patients from the nationwide LIPIDOGRAM 2004–2015 studies. Eur J Prev Cardiol. 2023; 30(12): 1195–1204, doi: 10.1093/eurjpc/zwad101, indexed in Pubmed: 37039119.
19. Jóźwiak JJ, Kasperczyk S, Tomasik T, et al. Design and rationale of a nationwide screening analysis from the LIPIDOGRAM2015 and LIPIDOGEN2015 studies. Arch Med Sci. 2022; 18(3): 604–616, doi: 10.5114/aoms.2020.96052, indexed in Pubmed: 35591817.
20. Kaess BM, Jóźwiak J, Nelson CP, et al. The relation of rapid changes in obesity measures to lipid profile — insights from a nationwide metabolic health survey in 444 Polish cities. PLoS One. 2014; 9(1): e86837, doi: 10.1371/journal.pone.0086837, indexed in Pubmed: 24497983.
21. Solnica B, Sygitowicz G, Sitkiewicz D, et al. 2024 Guidelines of the Polish Society of Laboratory Diagnostics and the Polish Lipid Association on laboratory diagnostics of lipid metabolism disorders. Arch Med Sci. 2024; 20(2): 357–374, doi: 10.5114/aoms/186191, indexed in Pubmed: 38757022.
22. Studziński K, Tomasik T, Windak A, et al. The differences in the prevalence of cardiovascular disease, its risk factors, and achievement of therapeutic goals among urban and rural primary care patients in Poland: Results from the LIPIDOGRAM 2015 study. J Clin Med. 2021; 10(23): 5656, doi: 10.3390/jcm10235656, indexed in Pubmed: 34884357.
23. Fox CS, Golden SH, Anderson C, et al. Update on prevention of cardiovascular disease in adults with type 2 diabetes mellitus in light of recent evidence: A scientific statement from the American Heart Association and the American Diabetes Association. Diabetes Care. 2015; 38(9): 1777–1803, doi: 10.2337/dci15-0012, indexed in Pubmed: 26246459.
24. Coral DE, Smit F, Farzaneh A, et al. Subclassification of obesity for precision prediction of cardiometabolic diseases. Nat Med. 2025; 31(2): 534–543, doi: 10.1038/s41591-024-03299-7, indexed in Pubmed: 39448862.
25. Gómez-Ambrosi J, Silva C, Galofré JC, et al. Body adiposity and type 2 diabetes: Increased risk with a high body fat percentage even having a normal BMI. Obes (Silver Spring). 2011; 19(7): 1439–1444, doi: 10.1038/oby.2011.36, indexed in Pubmed: 21394093.
26. Marques A, Peralta M, Naia A, et al. Prevalence of adult overweight and obesity in 20 European countries, 2014. Eur J Public Health. 2018; 28(2): 295–300, doi: 10.1093/eurpub/ckx143, indexed in Pubmed: 29036436.
27. Janssen F, Bardoutsos A, Vidra N. Obesity prevalence in the long-term future in 18 European countries and in the USA. Obes Facts. 2020; 13(5): 514–527, doi: 10.1159/000511023, indexed in Pubmed: 33075798.
28. Rywik S, Kupść W, Piotrowski W, et al. Multicenter nationwide health survey of the population – the WOBASZ project. Methodological assumptions and logistics [in Polish]. Kardiol Pol. 2005; 63(Suppl IV).
29. Drygas W, Niklas AA, Piwońska A, et al. Multi-centre National Population Health Examination Survey (WOBASZ II study): Assumptions, methods, and implementation. Kardiol Pol. 2016; 74(7): 681–690, doi: 10.5603/KP.a2015.0235, indexed in Pubmed: 26620680.
30. Stepaniak U, Micek A, Waśkiewicz A, et al. Prevalence of general and abdominal obesity and overweight among adults in Poland. Results of the WOBASZ II study (2013–2014) and comparison with the WOBASZ study (2003–2005). Pol Arch Med Wewn. 2016; 126(9): 662–671, doi: 10.20452/pamw.3499, indexed in Pubmed: 27535012.
31. Nauli AM, Matin S. Why do men accumulate abdominal visceral fat? Front Physiol. 2019; 10: 1486, doi: 10.3389/fphys.2019.01486, indexed in Pubmed: 31866877.
32. Muscogiuri G, Verde L, Vetrani C, et al. Obesity: A gender-view. J Endocrinol Invest. 2024; 47(2): 299–306, doi: 10.1007/s40618-023-02196-z, indexed in Pubmed: 37740888.
33. Schorr M, Dichtel LE, Gerweck AV, et al. Sex differences in body composition and association with cardiometabolic risk. Biol Sex Differ. 2018; 9(1): 28, doi: 10.1186/s13293-018-0189-3, indexed in Pubmed: 29950175.
34. Després JP, Moorjani S, Lupien PJ, et al. Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. Arteriosclerosis. 1990; 10(4): 497–511, doi: 10.1161/01.atv.10.4.497, indexed in Pubmed: 2196040.
35. de Mutsert R, Gast K, Widya R, et al. Associations of abdominal subcutaneous and visceral fat with insulin resistance and secretion differ between men and women: The Netherlands epidemiology of obesity study. Metab Syndr Relat Disord. 2018; 16(1): 54–63, doi: 10.1089/met.2017.0128, indexed in Pubmed: 29338526.
36. Kim H, Kim SE, Sung MK. Sex and gender differences in obesity: Biological, sociocultural, and clinical perspectives. World J Mens Health. 2025; 43(4): 758–772, doi: 10.5534/wjmh.250126, indexed in Pubmed: 40676890.
37. Tobacco consumption statistics - Statistics Explained - Eurostat. https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Tobacco_consumption_statistics (accessed: October 29, 2025).
38. Alcohol consumption statistics - Statistics Explained - Eurostat. https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Alcohol_consumption_statistics (accessed: October 29, 2025).
39. Al-Goblan AS, Al-Alfi MA, Khan MZ. Mechanism linking diabetes mellitus and obesity. Diabetes Metab Syndr Obes. 2014; 7: 587–591, doi: 10.2147/DMSO.S67400, indexed in Pubmed: 25506234.
40. Vekic J, Zeljkovic A, Stefanovic A, et al. Obesity and dyslipidemia. Metabolism. 2019; 92: 71–81, doi: 10.1016/j.metabol.2018.11.005, indexed in Pubmed: 30447223.
41. Ginsberg HN, Zhang YL, Hernandez-Ono A. Metabolic syndrome: Focus on dyslipidemia. Obesity (Silver Spring). 2006; 14 (Suppl 1): 41S–49S, doi: 10.1038/oby.2006.281, indexed in Pubmed: 16642962.
42. Zhao Y, Liu L, Yang S, et al. Mechanisms of atherosclerosis induced by postprandial lipemia. Front Cardiovasc Med. 2021; 8: 636947, doi: 10.3389/fcvm.2021.636947, indexed in Pubmed: 33996937.
43. Dias CB, Moughan PJ, Wood LG, et al. Postprandial lipemia: Factoring in lipemic response for ranking foods for their healthiness. Lipids Health Dis. 2017; 16(1): 178, doi: 10.1186/s12944-017-0568-5, indexed in Pubmed: 28923057.
44. Ginsberg HN, Packard CJ, Chapman MJ, et al. Triglyceride-rich lipoproteins and their remnants: Metabolic insights, role in atherosclerotic cardiovascular disease, and emerging therapeutic strategies — a consensus statement from the European Atherosclerosis Society. Eur Heart J. 2021; 42(47): 4791–4806, doi: 10.1093/eurheartj/ehab551, indexed in Pubmed: 34472586.
45. Christian AH, Mochari H, Mosca LJ. Waist circumference, body mass index, and their association with cardiometabolic and global risk. J Cardiometab

Syndr. 2009; 4(1): 12–19, doi: 10.1111/j.1559-4572.2008.00029.x, indexed in Pubmed: 19245511.
46. Mohseni P, Khalili D, Djalalinia S, et al. The synergistic effect of obesity and dyslipidemia on hypertension: Results from the STEPS survey. Diabetol Metab Syndr. 2024; 16(1): 81, doi: 10.1186/s13098-024-01315-x, indexed in Pubmed: 38566160.
47. Zhang Y, Moran AE. Trends in the prevalence, awareness, treatment, and control of hypertension among young adults in the United States, 1999 to 2014. Hypertension. 2017; 70(4): 736–742, doi: 10.1161/HYPERTENSIONAHA.117.09801, indexed in Pubmed: 28847890.
48. Zdrojewski T, Solnica B, Cybulska B, et al. Prevalence of lipid abnormalities in Poland. The NATPOL 2011 survey. Kardiol Pol. 2016; 74(3): 213–223, doi: 10.5603/KP.2016.0029, indexed in Pubmed: 27004543.
49. Pająk A, Szafraniec K, Polak M, et al. Changes in the prevalence, treatment, and control of hypercholesterolemia and other dyslipidemias over 10 years in Poland: The WOBASZ study. Pol Arch Med Wewn. 2016; 126(9): 642–652, doi: 10.20452/pamw.3464, indexed in Pubmed: 27452484.
50. Jura M, Kozak LP. Obesity and related consequences to ageing. Age (Dordr). 2016; 38(1): 23, doi: 10.1007/s11357-016-9884-3, indexed in Pubmed: 26846415.