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Associations Between Body Mass Index (BMI) and Dyslipidemia: Results From the PERSIAN Guilan Cohort Study (PGCS)

Jahangir Shahraz[1] | Farahnaz Joukar[1] | Fateme Sheida[1] | Sara Yeganeh[1] | Saman Maroufizadeh[2] | Massood Baghaee[1] | Mohammadreza Naghipour[1] | Fariborz Mansour-Ghanaei[1]

[1]Gastrointestinal and Liver Diseases Research Center, Guilan University of Medical Sciences, Rasht, Iran | [2]Department of Biostatistics and Epidemiology, School of Health, Guilan University of Medical Sciences, Rasht, Iran

Correspondence: Fariborz Mansour-Ghanaei (ghanaie@yahoo.com; fmansourghanaei@gmail.com)

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ABSTRACT

Background: Obesity and dyslipidemia are interconnected complex conditions and their prevalence differs across different geographical regions. As a major risk factor for cardiovascular diseases, dyslipidemia is often misdiagnosed and inadequately treated, highlighting the need for region-specific public health policies. Therefore, the objective of this study was to examine the associations between BMI and dyslipidemia in the Prospective Epidemiological Research Studies in Iran (PERSIAN) Guilan Cohort study (PGCS) population.

Methods: This cross-sectional study analyzed the demographic and biochemical data from 10,519 participants of the PGCS population. Participants were divided into two groups with and without dyslipidemia and were compared based on BMI. Data analysis was performed in SPSS v16 with a significance level of < 0.05.

Results: The average age of the participants was 51.52 ± 8.90 years. The prevalence of dyslipidemia in all participants was equal to 75.83%. Among those with dyslipidemia, 41.18% and 35.39% had overweight and obesity, respectively. There was a positive association between BMI and the prevalence of dyslipidemia (unadjusted OR = 1.09, 95% confidence interval (CI): 1.08–1.10) ($p < 0.01$), indicating that for a one-unit increase in participants' BMI, the probability of having dyslipidemia increased by 9%, which remained statistically significant even after adjusting. Analysis of dyslipidemia components and BMI revealed a significant association between elevated TG and cholesterol, as well as low HDL levels and higher BMI (unadjusted OR = 1.04, 1.01, and 1.09, respectively) ($p < 0.01$). However, this was not statistically significant for high LDL levels (unadjusted OR = 1.01) ($p = 0.05$).

Conclusion: Given the high prevalence of dyslipidemia in our studied region and its strong association with obesity, prioritizing obesity management in public health decision-making is vital. Greater focus should be given on accessing and modifying the components of dyslipidemia, particularly LDL particles, as a potentially significant research target to prevent the mismanagement of dyslipidemia in individuals with obesity.

Jahangir Shahraz and Farahnaz Joukar Co-first author.

1 | Introduction

According to global data, in 2022, 2.5 billion adults (aged ≥ 18 years) were overweight, with over 890 million suffering from obesity [1, 2]. The prevalence of overweight and obesity in Middle East countries is 54.2% and 31.4% in women and men, respectively [3, 4]. Based on a cohort study in northwestern Iran, 42.7% and 45% of participants had overweight and obesity, respectively [5]. There are different classifications for obesity, but according to the World Health Organization (WHO), the most important definition is based on body mass index (BMI), which is obtained by dividing a person's weight in kilograms by the square of height in meters (kg/m^2). BMI more than or equal to 25 and 30 is categorized as overweight and obesity, respectively [6].

Obesity is associated with increased mortality and morbidity, by its link with many non-communicable diseases (NCDs), including cardiovascular diseases [7], cancer, type 2 diabetes, liver disease like non-alcoholic fatty liver disease (NAFLD), and other metabolic disorders [8–11], mostly through promoting insulin resistance and pro-inflammatory cytokines. However, not all persons with obesity are at an increased risk of cardiovascular and metabolic diseases. Despite having high fat deposits, a specific group of people with metabolically healthy obesity (MHO) has been found, with preserved lipid and inflammatory profiles and normal blood pressure, unlike the metabolically unhealthy obesity (MUO) condition [12]. On the other hand, there are individuals with unhealthy normal weight (UN) who are at increased risk of disease and mortality despite their normal weight based on classifications [13]. Considering the aforementioned, while there is a widely studied correlation between obesity and health issues, especially NCDs, the actual connection may not be as straightforward.

Data derived from both animal and human experiments indicates that the association between obesity and NCDs originated mainly from disturbed levels of circulating lipids, characterized by elevation in triglyceride (TG), very low-density lipoprotein (VLDL), apolipoprotein B (Apo B), non-high-density lipoprotein cholesterol (non-HDL-C), small dense low-density lipoprotein (sdLDL), low-density lipoprotein (LDL) levels, but decreasing in HDL and Apo A1 [14, 15]. Disturbed levels of lipids lead to abnormal accumulation of them and lipotoxicity, which consequently lead to adverse effects on normal body metabolism. In addition to obesity, dyslipidemia is a complex condition that is affected by various factors such as age, gender, genetic background, lifestyle, socioeconomic status, and other chronic metabolic diseases [16, 17].

Despite considering the critical role of dyslipidemia in obesity-related diseases, it should be noticed that the pattern and prevalence of dyslipidemia is not the same in all individuals with obesity and that obesity does not automatically mean dyslipidemia and increased risk of NCDs. About 60%–70% of patients with obesity have dyslipidemia, and obesity-related dyslipidemia is commonly classified as atherogenic dyslipidemia (AD), defined by the presence of increased TG, low HDL-C, and high plasma levels of sdLDL, and is known to promote the development of atherosclerosis and increase the risk of coronary events [18, 19]. Studies revealed that visceral adipose tissue (VAT), which is mainly increased in individuals with obesity, causes atherogenic dyslipidemia and the accumulation of excess fat in the abdomen is the main cause of insulin resistance [20]. Therefore, an early diagnosis and active treatment of dyslipidemia are excellent precautions for cardiovascular diseases.

As discussed, both obesity and dyslipidemia are complex disorders influenced by multiple factors, figuring out the exact links between BMI and dyslipidemia in a specified population offers significant practical utility for risk assessment, early identification, and precise personalized strategies for managing cardiovascular risks. Therefore, the objective of this study was to examine the associations between BMI and dyslipidemia in the Prospective Epidemiological Research Studies in Iran (PERSIAN) Guilan Cohort study (PGCS) population.

2 | Material and Methods

2.1 | Participants

This analytical cross-sectional study was a part of the PGCS population, as a sub-study of the PERSIAN cohort [21], including 10,519 participants aged 35 to 70 of both sexes living in some urban and rural areas of Some'e Sara County, Guilan, Iran, from October 8, 2014 to January 20, 2017 [22].

2.2 | Data Collection

Participants were initially registered and received an 11-digit code. The questionnaire was then completed through a face-to-face interview.

2.3 | Demographic Data and Life Style Characteristics

Information including age, sex, BMI categories (underweight: < 18.50 kg/m^2, normal: 18.50–24.99 kg/m^2, overweight: 25–30 kg/m^2, obesity: >30 kg/m^2), marital status, education, employment status, place of residency, wealth status quartiles, physical activity, smoking and hookah, drugs and alcohol consumption [22].

2.4 | Laboratory Data

Five mL samples of fasting blood were collected by expert technicians and labeled for biochemistry tests including TG, total cholesterol, HDL, and LDL. Blood samples were sent to the Cohort Central Laboratory in a cold box for testing [22].

2.5 | Definition of Dyslipidemia

Dyslipidemia was defined as serum total cholesterol ≥ 200 mg/dL, TG ≥ 150 mg/dL, LDL > 130 mg/dL or more than 70 mg/dL in people with a history of diabetes, heart attack, cardiac stent, coronary artery bypass graft, or other cardiovascular diseases,

and Abnormal HDL (serum HDL < 40 mg/dL in men and < 50 mg/dL in women) [22].

2.6 | Informed Consent

This study was conducted based on the Helsinki Declaration, and consent was obtained from the participants before conducting the study.

2.7 | Ethics Committee Approval

Ethical committee approval was obtained by the Ethics Committee of Guilan University of Medical Sciences, Rasht, Iran (Ethics Code: IR. GUMS.1401.277).

2.8 | Statistical Analysis

The values of qualitative and quantitative variables are represented as "frequency (percentage)" and "mean ± standard deviation," respectively. To assess the demographic features of individuals with and without dyslipidemia, an independent *t*-test was employed for quantitative data, while a chi-square test was utilized for qualitative factors. Furthermore, both unadjusted and adjusted studies employed simple logistic regression and multiple logistic regression techniques to evaluate the association between dyslipidemia and BMI, accordingly. The findings of these analyses were reported as unadjusted odds ratio (OR) and adjusted odds ratio (aOR) together with 95% confidence intervals (95% CI). The study employed Poisson regression analysis to examine the association between the number of dyslipidemia components and body mass index (BMI). The outcomes of this approach were presented in the form of incidence rate ratio (IRR) and 95% CI. The data analysis was conducted using SPSS version 16 software with a significance level < 0.05.

3 | Result

Among the 10,519 individuals included in the study, the average age was 51.52 ± 8.90 years, and 53.55% of the participants were female. A significant proportion, 75.83%, had dyslipidemia, predominantly observed in the age group of 45–54 years (37.63%). The average BMI of the individuals studied was 28.14 ± 5.09 kg/m^2. Based on the BMI analysis, 1.34% of participants were underweight, 26.10% had normal weight, 39.91% were overweight, and 32.65% were obese. Notably, among those with dyslipidemia, 41.18% and 35.39% were overweight and obese, respectively, highlighting a significant association between weight status and dyslipidemia in the study population. A comprehensive overview of the demographic and clinical profiles of the study participants is presented in Table 1.

The prevalence and number of components of dyslipidemia in all participants and by gender is shown in Table 2. Hypercholesterolemia was observed in 40.31% of participants, hypertriglyceridemia in 43.13%, low HDL levels in 40.51%, and high LDL levels in 28.89%.

A significant relationship between BMI (both quantitative and qualitative) and dyslipidemia was observed ($p < 0.01$) (Table 3). In the unadjusted model (Model 1), there was a positive association between BMI and the prevalence of dyslipidemia. The OR for this association was 1.09 (95% CI: 1.08–1.10), indicating that for a one-unit increase in participants' BMI, the possibility of having dyslipidemia increased by 9%. The observed association between the variables remained statistically significant even after adjusting for age, gender, and other demographic variables in Models 2 and 3. The relationship between hypercholesterolemia, hypertriglyceridemia, Low HDL, high LDL and BMI with and without adjusting for demographic variables is presented in Table 4.

4 | Discussion

Both high BMI and dyslipidemia are shown to be substantial risk factors for NCDs such as cardiovascular disease, cancer, and diabetes [7]. Despite the impact of these "metabolic factors" on the risk of cardiovascular disease, they are modifiable by both lifestyle changes and medical therapies, which attracts considerable research attention [23].

Results from the present study revealed that the prevalence of dyslipidemia was 75.83%. The prevalence of dyslipidemia in developing countries is gradually increasing due to socioeconomic transitions and lifestyle transformations, for example, shifting from traditional, nutritionally balanced diets to western ones [24]. The overall prevalence of dyslipidemia reported in this study is consistent with the findings in northern and southern Ethiopia [25, 26]. However, the reported prevalence is higher than the findings reported in Africa (29.7%) [27], Eastern Ethiopia (34.8%) [28], and China (43.3%) [17]. The reason for the high prevalence in this study can be attributed to the difference in sample size, study environment, study population, socioeconomic status, and lifestyle of the study participants.

In the unadjusted model, the possibility of having dyslipidemia increased with higher BMI; approximately for a one-unit increase in participants' BMI, the risk of having dyslipidemia increased by 9%. This relationship remained significant after adjusting for age and gender, and also for all variables. This finding is consistent with the Yudin et al. study [20]. Various studies have also reported that obesity contributes to the release of large amounts of free fatty acids by lipolysis, which leads to dyslipidemia [1, 29].

The proportion of women, people who were unemployed, or those living in rural areas were more affected by dyslipidemia. These results were not consistent with the study of Erem et al. and Antal et al. [30, 31]. But the findings of many previous studies show that women have a significantly higher prevalence of dyslipidemia [26, 32, 33]. Unlike the present study, the relationship between dyslipidemia, and smoking was confirmed in different studies. Smoking may increase LDL, and TG levels but decrease HDL levels, which increases dyslipidemia [34–36].

TABLE 1 | Demographic comparison between people with and without dyslipidemia in PERSIAN Guilan cohort study.

	Total 10,519 (%)	With dyslipidemia 7977 (75.83%)	Without dyslipidemia 2542 (24.16%)	*p*-value
Age (year)				0.65
35–44	3138 (29.83)	2329 (29.20)	809 (31.82)	
45–54	3854 (36.64)	3002 (37.63)	852 (33.52)	
55–65	2730 (25.95)	2082 (26.10)	648 (25.49)	
> 65	797 (7.58)	564 (7.07)	233 (9.17)	
Mean ± SD	51.52 ± 8.90	51.50 ± 8.76	51.56 ± 9.33	0.80
Sex				< 0.01
Male	4886 (46.45)	3424 (42.92)	1462 (57.51)	
Female	5633 (53.55)	4553 (57.08)	1080 (42.49)	
Marital status				0.09
Single	305 (2.90)	232 (2.91)	73 (2.87)	
Married	9527 (90.57)	7196 (90.21)	2330 (91.66)	
Widow	566 (5.38)	453 (5.68)	113 (4.44)	
Divorce	122 (1.16)	96 (1.20)	26 (1.02)	
Education				
Illiterate	1783 (16.95)	1317 (16.51)	421 (16.56)	0.19
1–5 years of schooling	3312 (31.49)	2475 (31.03)	837 (32.93)	
6–12 years of schooling	4831 (45.93)	3692 (46.28)	1139 (44.81)	
University/college	638 (6.06)	493 (6.18)	145 (5.70)	
Employment status				< 0.01
Unemployed	4781 (45.45)	3805 (47.70)	976 (38.39)	
Employed	5738 (54.55)	4172 (52.30)	1566 (61.60)	
Place of residency				< 0.01
Urban	4612 (43.84)	3408 (42.72)	1204 (47.36)	
Rural	5907 (56.15)	4569 (57.28)	1338 (52.64)	
Wealth status quartiles				< 0.01
1st	2630 (25.00)	1935 (24.25)	695 (27.34)	
2nd	2630 (25.00)	1950 (24.44)	680 (26.75)	
3rd	2630 (25.00)	2021 (25.33)	609 (23.96)	
4th	2629 (24.99)	2071 (25.96)	558 (21.95)	
BMI categories, kg/m^2				< 0.01
Underweight	141 (1.34)	69 (0.86)	72 (2.83)	
Normal	2745 (26.10)	1800 (22.56)	945 (37.17)	
Overweight	4198 (39.91)	3285 (41.18)	913 (35.92)	
Obese	3435 (32.65)	2823 (35.39)	612 (24.07)	
Mean ± SD	28.14 ± 5.09	28.62 ± 4.98	26.66 ± 5.13	< 0.01
Physical activity				< 0.01
1st	2630 (25.00)	2115 (26.51)	515 (20.26)	
2nd	2630 (25.00)	2079 (26.06)	551 (21.68)	
3rd	2630 (25.00)	1967 (24.66)	663 (26.08)	
4th	2629 (24.99)	1816 (22.76)	813 (31.98)	
Ever cigarette smoker	2584 (24.56)	1796 (22.51)	788 (31.00)	< 0.01
Ever hookah smoker	1515 (14.40)	1055 (13.22)	460 (18.10)	< 0.01

(Continues)

TABLE 1 | (Continued)

	Total 10,519 (%)	With dyslipidemia 7977 (75.83%)	Without dyslipidemia 2542 (24.16%)	*p*-value
Ever drug user	726 (6.90)	534 (6.69)	192 (7.55)	0.14
Ever alcohol user	1395 (13.26)	943 (11.82)	452 (17.78)	< 0.01

Abbreviations: BMI, body mass index; SD, standard deviation.
Significance level: *p*-value < 0.05.

TABLE 2 | Prevalence and number of components of dyslipidemia in all participants and by gender in the PERSIAN Guilan cohort study.

	Male *n* = 4886 (%)	Female *n* = 5633 (%)	Total *n* = 10,519 (%)
Component of dyslipidemia			
Hypercholesterolemia	1900 (38.88)	2340 (41.54)	4240 (40.31)
Hypertriglyceridemia	2226 (45.56)	2311 (41.03)	4537 (43.13)
Low HDL	1293 (26.46)	2968 (52.69)	4261 (40.51)
High LDL	1371 (28.06)	1678 (29.79)	3039 (28.89)
Number of components of dyslipidemia			
0	1462 (29.92)	1080 (19.17)	2542 (24.17)
1	1060 (21.69)	1515 (26.90)	2575 (24.48)
2	1482 (30.33)	1745 (30.98)	3227 (30.68)
3	762 (15.60)	880 (15.62)	1642 (15.61)
4	120 (2.46)	413 (7.33)	533 (5.07)
Dyslipidemia	3424 (70.08)	4553 (80.83)	7977 (75.83)

TABLE 3 | The relationship between dyslipidemia and BMI in the participants of the PERSIAN Guilan cohort study.

	Prevalence of dyslipidemia		Model 1		Model 2		Model 3	
	n/*N*	%	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
BMI (quantitative)			1.09 (1.08–1.10)	< 0.01	1.07 (1.06–1.08)	< 0.01	1.07 (1.06–1.08)	< 0.01
BMI (qualitative)								
Normal	1800/2745	65.57	1		1		1	
Underweight	69/141	48.94	0.50 (0.36–0.71)	< 0.01	0.52 (0.37–0.73)	< 0.01	0.56 (0.40–0.79)	< 0.01
Overweight	3285/4198	78.25	1.89 (1.70–2.10)	< 0.01	1.77 (1.58–1.97)	< 0.01	1.70 (1.52–1.90)	< 0.01
Obese	2823/3435	82.18	2.42 (2.15–2.72)	< 0.01	2.03 (1.79–2.31)	< 0.01	1.94 (1.71–2.21)	< 0.01

Note: Model 1: Unadjusted model; Model 2: Adjusted for age and sex; Model 3: Adjusted for age and sex, marital status, education, employment status, place of residency, wealth status quartiles, BMI, physical activity, smoking and hookah, drugs and alcohol consumption.
Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.
Significance level: *p*-value < 0.05.

Also, there was a relationship between low physical activity and dyslipidemia that matched the study by Haile et al. [37], Li et al. [38], and Al zaheb et al. [39]. This can be explained by prolonged sitting, which means fewer calories are burned and may lead to calories being stored as lipids [40].

The results of this study regarding the relationship between the components of dyslipidemia and BMI showed that for a one-unit increase in participants' BMI, the risk of having hypercholesterolemia and hypertriglyceridemia increased by 1% and 4%, respectively. This relationship remained significant after adjusting for age and gender variables. LDL has the same feature, but it was not statistically significant. The relationship between HDL and BMI showed that by one-unit elevation of BMI, the probability of having low HDL increased by 9%. A decrease in HDL level and an increase in triglyceride level, as abnormalities of lipid metabolism, are usually seen in individuals with obesity [41, 42]. Accumulation of serum TG is caused by increased production of hepatic VLDL and decreased clearance of triglyceride-rich lipoproteins. Such an increase in production occurs due to impaired insulin signaling, which improves lipolysis and the conversion of TG to free fatty acids (FFA) in adipocytes, transporting via the blood to the liver and muscles [40]. In addition, HDL metabolism is strongly associated with obesity due to the increase in the number of chylomicrons and VLDL remnants along with impaired lipolysis. Lipolysis of triglyceride-rich HDL appears due to hepatic lipase, which leads to the production of sHDL with a decrease in the affinity of Apo A-I, which leads to a decrease in HDL levels and a decrease in circulating HDL particles, thereby impairing

TABLE 4 | The relationship between hypercholesterolemia, hypertriglyceridemia, low HDL, high LDL and BMI in the participants of the PERSIAN Guilan cohort study.

	Prevalence of dyslipidemia component		Model 1		Model 2		Model 3	
	n/*N*	%	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
Hypercholesterolemia								
BMI (quantitative)			1.01 (1.00–1.02)	< 0.01	1.01 (1.00–1.02)	< 0.01	1.01 (1.00–1.02)	< 0.01
BMI (qualitative)								
Normal	1045/2745	38.07	1		1		1	
Underweight	31/141	21.99	0.46 (0.31–0.69)	< 0.01	0.46 (0.31–0.70)	< 0.01	0.49 (0.33–0.74)	< 0.01
Overweight	1747/4198	41.62	1.16 (1.05–1.27)	< 0.01	1.15 (1.04–1.27)	< 0.01	1.15 (1.04–1.27)	< 0.01
Obese	1417/3435	41.25	1.14 (1.03–1.27)	0.01	1.12 (1.00–1.25)	0.04	1.12 (1.00–1.25)	0.04
Hypertriglyceridemia								
BMI (quantitative)			1.04 (1.04–1.05)	< 0.01	1.06 (1.05–1.07)	< 0.01	1.06 (1.05–1.07)	< 0.01
BMI (qualitative)								
Normal	939/2745	34.21	1		1		1	
Underweight	31/141	21.99	0.54 (0.36–0.81)	< 0.01	0.52 (0.35–0.78)	< 0.01	0.55 (0.36–0.82)	< 0.01
Overweight	1928/4198	45.93	1.63 (1.48–1.80)	< 0.01	1.77 (1.60–1.96)	< 0.01	1.72 (1.55–1.90)	< 0.01
Obese	1639/3435	47.71	1.76 (1.58–1.95)	< 0.01	2.12 (1.90–2.37)	< 0.01	2.03 (1.81–2.27)	< 0.01
Low HDL								
BMI (quantitative)			1.09 (1.08–1.09)	< 0.01	1.05 (1.04–1.06)	< 0.01	1.04 (1.04–1.05)	< 0.01
BMI (qualitative)								
Normal	752/2745	27.40	1		1		1	
Underweight	31/141	21.99	0.75 (0.50–1.12)	0.16	0.81 (0.53–1.23)	0.32	0.82 (0.54–1.24)	0.34
Overweight	1727/4198	41.14	1.85 (1.67–2.06)	< 0.01	1.56 (1.40–1.73)	< 0.01	1.51 (1.36–1.69)	< 0.01
Obese	1751/3435	50.98	2.76 (2.48–3.07)	< 0.01	1.80 (1.60–2.02)	< 0.01	1.71 (1.52–1.93)	< 0.01
High LDL								
BMI (quantitative)			1.01 (1.00–1.02)	0.05	1.01 (1.00–1.02)	0.12	1.01 (1.00–1.02)	0.17
BMI (qualitative)								
Normal	768/2745	27.98	1		1		1	
Underweight	25/141	17.73	0.55 (0.36–0.86)	< 0.01	0.56 (0.36–0.87)	0.01	0.60 (0.38–0.93)	0.02
Overweight	1251/4198	29.80	1.09 (0.98–1.22)	0.10	1.09 (0.98–1.21)	0.12	1.07 (0.96–1.20)	0.20
Obese	1005/3435	29.26	1.06 (0.95–1.19)	0.27	1.05 (0.93–1.18)	0.47	1.04 (0.92–1.17)	0.54

Note: Model 1: Unadjusted model; Model 2: Adjusted for age and sex; Model 3: Adjusted for age and sex, marital status, education, employment status, place of residency, wealth status quartiles, BMI, physical activity, smoking and hookah, drugs and alcohol consumption.
Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.
Significance level *p*-value: < 0.05.

cholesterol transport [43]. Consistent with the statistically non-significant correlation between LDL and BMI in the current examined population, other studies have also reported modest differences in LDL-C levels between individuals with and without obesity. However, this result is quite controversial and requires further investigation. This misleading may be explained by considering the role of obesity in reducing the size of LDL particles, resulting in the formation of atherogenic, denser, and smaller particles (sdLDL). The increased atherogenicity of these particles may be attributed to their enhanced ability to pass through the artery intima, stronger binding for intra-arterial proteoglycans, and greater susceptibility to oxidation. Consequently, this leads to increased absorption by local macrophages and the promotion of a pro-inflammatory state. Given their association with cardiovascular disorders, the clinical and detailed examination of LDL particles in the lipid profile of patients should be prioritized to prevent the mismanagement of dyslipidemia [19, 40, 44].

The risk of dyslipidemia was shown to be higher in the high BMI group based on the results of this study. Therefore, weight loss should be recommended for individuals with a high BMI. Additionally, less physical activity was observed among people with dyslipidemia. Thus, community-based education in physical activity and sports is considered of great importance. Routine assessment of BMI should be conducted in primary care

clinics for adults and children to facilitate the early diagnosis, evaluation, and treatment of obesity and related disorders.

The strength of this study was the examination of 10,519 people who were referred to the cohort of Some'e Sara County, Guilan, Iran. On the other hand, considering the referral center, the presence of trained nurses, general practitioners, specialists, and the central laboratory in the center, it can be claimed that the results of the study have great validity. In this study, only one anthropometric index was examined and other indices such as the waist–hip ratio (WHR), and waist circumference (WC) were not examined. Therefore, it is suggested that future studies investigate the relationship between other anthropometric indicators and dyslipidemia.

5 | Conclusions

Given the high prevalence of dyslipidemia and obesity in our studied population and their significant association, it is imperative to give more consideration to this concept in public health decision making. Greater focus should be given on accessing and modifying the components of dyslipidemia, particularly LDL particles, as a potentially significant research target to prevent the mismanagement of dyslipidemia in individuals with obesity. The outcomes of this study not only supported the existing evidence on the connection between overweight/obesity and lipid profile but also offer valuable insights for public health professionals in this region. Therefore, weight loss should be recommended, suitable anti-lipid agents and regional-adapted lipid profile assessment should be applied, and community-based education would be helpful to increase public and professionals' knowledge.

Author contributions

F.M.G., J.S., and F.J. participated in the research design. S.Y. and F.S. participated in writing the first draft. F.J., S.M., F.S., M.N., and M.B. participated in the performance of the research and analytic tools. S.M. participated in data analysis. All authors reviewed and confirmed the final manuscript.

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Disclosure

The current study was extracted from an internal medicine residency thesis.

Ethics Statement

This study was approved by the ethics committees of the Guilan University of Medical Sciences [IR.GUMS.REC.1401.277]. It was carried out according to the guidelines rendered in the Declaration of Helsinki. Informed consent was obtained from all participants.

Consent

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The datasets used during the current study are available on PERSIAN Guilan cohort study (PGCS) center, Guilan University of Medical Sciences, Guilan, Iran. The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

References

1. T. Zhang, J. Chen, X. Tang, Q. Luo, D. Xu, and B. Yu, "Interaction Between Adipocytes and High-Density Lipoprotein: New Insights Into the Mechanism of Obesity-Induced Dyslipidemia and Atherosclerosis," *Lipids in Health and Disease* 18 (2019): 1–11, https://doi.org/10.1186/s12944-019-1170-9.

2. G. Collaborators, J. Ärnlöv, P. Zheng, et al., "Global Burden of 87 Risk Factors in 204 Countries and Territories, 1990–2019: A Systematic Analysis for the Global Burden of Disease Study 2019," *Lancet* 396, no. 10258 (2020): 1223–1249, https://doi.org/10.1016/s0140-6736(20)30752-2.

3. M. H. Eftekhari, S. Nikookar, H. Tabatabaei, and M. R. Zahedani, "GW27-e0096 Overweight and Obesity and Their Associated Factors in Adult Women Referring to Medical and Health Centers in Shiraz, Iran," *Journal of the American College of Cardiology* 68, no. 16S (2016): C182–C183, https://doi.org/10.1016/j.jacc.2016.07.689.

4. N. S. Farrag, L. J. Cheskin, and M. K. Farag, "A Systematic Review of Childhood Obesity in the Middle East and North Africa (MENA) Region: Prevalence and Risk Factors Meta-Analysis," *Advances in pediatric research* 4 (2017).

5. F. Pourfarzi, A. Sadjadi, H. Poustchi, and F. Amani, "Prevalence of Overweight and Obesity in Iranian Population: A Population-Based Study in Northwestern of Iran," *Journal of Public Health Research* 11, no. 1 (2022): 475, https://doi.org/10.4081/jphr.2021.2475.

6. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.

7. T. M. Powell-Wiley, P. Poirier, L. E. Burke, et al., "Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association," *Circulation* 143, no. 21 (2021): e984–e1010, https://doi.org/10.1161/cir.0000000000000973.

8. I. Kyrou, H. S. Randeva, C. Tsigos, G. Kaltsas, and M. O. Weickert, "Clinical Problems Caused by Obesity," in *Endotext* [Internet], eds. K. R. Feingold, B. Anawalt, M. R. Blackman, et al. (South Dartmouth (MA): MDText.com, Inc., 2018).

9. https://apps.who.int/iris/bitstream/handle/10665/353747/9789289057738-eng.pdf.

10. A. M. Jastreboff, C. M. Kotz, S. Kahan, A. S. Kelly, and S. B. Heymsfield, "Obesity as a Disease: The Obesity Society 2018 Position Statement," *Obesity* 27, no. 1 (2019): 7–9, https://doi.org/10.1002/oby.22378.

11. P. Gordon-Larsen and S. B. Heymsfield, "Obesity as a Disease, Not a Behavior," *Circulation* 137, no. 15 (2018): 1543–1545, https://doi.org/10.1161/circulationaha.118.032780.

12. E. Navarro, A. N. Funtikova, M. Fíto, and H. Schröder, "Can Metabolically Healthy Obesity Be Explained by Diet, Genetics, and Inflammation?," *Molecular Nutrition & Food Research* 59, no. 1 (2015): 75–93, https://doi.org/10.1002/mnfr.201400521.

13. F. Badoud, M. Perreault, M. A. Zulyniak, and D. M. Mutch, "Molecular Insights Into the Role of White Adipose Tissue in Metabolically Unhealthy Normal Weight and Metabolically Healthy Obese Individuals," *FASEB Journal* 29, no. 3 (2015): 748–758, https://doi.org/10.1096/fj.14-263913.

14. H. E. Bays, P. P. Toth, P. M. Kris-Etherton, et al., "Obesity, Adiposity, and Dyslipidemia: A Consensus Statement From the National Lipid Association," *Journal of clinical lipidology* 7, no. 4 (2013): 304–383, https://doi.org/10.1016/j.jacl.2013.04.001.

15. C. Xiao, S. Dash, C. Morgantini, R. A. Hegele, and G. F. Lewis, "Pharmacological Targeting of the Atherogenic Dyslipidemia Complex: The Next Frontier in CVD Prevention Beyond Lowering LDL Cholesterol," *Diabetes* 65, no. 7 (2016): 1767–1778, https://doi.org/10.2337/db16-0046.

16. A. Pirillo, M. Casula, E. Olmastroni, G. D. Norata, and A. L. Catapano, "Global Epidemiology of Dyslipidaemias," *Nature Reviews Cardiology* 18, no. 10 (2021): 689–700, https://doi.org/10.1038/s41569-021-00541-4.

17. S. Opoku, Y. Gan, W. Fu, et al., "Prevalence and Risk Factors for Dyslipidemia Among Adults in Rural and Urban China: Findings From the China National Stroke Screening and Prevention Project (CNSSPP)," *BMC Public Health* 19 (2019): 1–15, https://doi.org/10.1186/s12889-019-7827-5.

18. B. Nussbaumerova and H. Rosolova, "Obesity and Dyslipidemia," *Current Atherosclerosis Reports* 25, no. 12 (2023): 947–955, https://doi.org/10.1007/s11883-023-01167-2.

19. J. Vekic, A. Stefanovic, and A. Zeljkovic, "Obesity and Dyslipidemia: A Review of Current Evidence," *Current Obesity Reports* 12, no. 3 (2023): 207–222, https://doi.org/10.1007/s13679-023-00518-z.

20. R. Yudin, A. M. Aman, H. Rasyid, S. Bakri, H. Sanusi, and A. A. Zainuddin, "Risk of Dyslipidemia in Obese Young Adult Subjects as Measured by Various Obesity Indices," *Journal of Endocrinology and Metabolism* 12, no. 3 (2022): 102–106, https://doi.org/10.14740/jem819.

21. H. Poustchi, S. Eghtesad, F. Kamangar, et al., "Prospective Epidemiological Research Studies in Iran (The Persian Cohort Study): Rationale, Objectives, and Design," *American Journal of Epidemiology* 187, no. 4 (2018): 647–655, https://doi.org/10.1093/aje/kwx314.

22. F. Mansour-Ghanaei, F. Joukar, M. R. Naghipour, et al., "The Persian Guilan Cohort Study (PGCS)," *Archives of Iranian Medicine* 22, no. 1 (2019).

23. W. H. Organization, *WHO European Regional Obesity Report 2022* (Regional Office for Europe: World Health Organization, 2022).

24. M. S. Ahmmed, S. D. Shuvo, D. K. Paul, et al., "Prevalence of Dyslipidemia and Associated Risk Factors Among Newly Diagnosed Type-2 Diabetes Mellitus (T2DM) Patients in Kushtia, Bangladesh," *PLOS Global Public Health* 1, no. 12 (2021): e0000003, https://doi.org/10.1371/journal.pgph.0000003.

25. G. Gebreegziabiher, T. Belachew, K. Mehari, and D. Tamiru, "Prevalence of Dyslipidemia and Associated Risk Factors Among Adult Residents of Mekelle City, Northern Ethiopia," *PLoS One* 16, no. 2 (2021): e0243103, https://doi.org/10.1371/journal.pone.0243103.

26. S. Bekele, T. Yohannes, and A. E. Mohammed, "Dyslipidemia and Associated Factors Among Diabetic Patients Attending Durame General Hospital in Southern Nations, Nationalities, and People's Region," *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy* 10 (2017): 265–271, https://doi.org/10.2147/dmso.s135064.

27. J. J. Noubiap, J. J. Bigna, J. R. Nansseu, et al., "Prevalence of Dyslipidaemia Among Adults in Africa: A Systematic Review and Meta-Analysis," *Lancet Global Health* 6, no. 9 (2018): e998–e1007, https://doi.org/10.1016/s2214-109x(18)30275-4.

28. B. Sufa, G. Abebe, and W. Cheneke, "Dyslipidemia and Associated Factors Among Women Using Hormonal Contraceptives in Harar Town, Eastern Ethiopia," *BMC Research Notes* 12 (2019): 1–7, https://doi.org/10.1186/s13104-019-4148-9.

29. U. J. Jung and M.-S. Choi, "Obesity and its Metabolic Complications: The Role of Adipokines and the Relationship Between Obesity, Inflammation, Insulin Resistance, Dyslipidemia and Nonalcoholic Fatty Liver Disease," *International Journal of Molecular Sciences* 15, no. 4 (2014): 6184–6223, https://doi.org/10.3390/ijms15046184.

30. C. Erem, A. Hacihasanoglu, O. Deger, M. Kocak, and M. Topbas, "Prevalence of Dyslipidemia and Associated Risk Factors Among Turkish Adults: Trabzon Lipid Study," *Endocrine* 34, no. 1-3 (2008): 36–51, https://doi.org/10.1007/s12020-008-9100-z.

31. M. Antal, K. Nagy, A. Regöly-Mérei, L. Bíró, C. Szabó, and B. Rabin, "Assessment of Cardiovascular Risk Factors Among Hungarian University Students in Budapest," *Annals of Nutrition and Metabolism* 50, no. 2 (2006): 103–107, https://doi.org/10.1159/000090497.

32. T. H. Agete and N. G. Eshetu, "Factors Associated With Atherogenic Dyslipidemia Among Hypertensive Patients at Southern Ethiopia," *International Journal of Medicine and Medical Sciences* 10, no. 7 (2018): 86–93, https://doi.org/10.5897/ijmms2018.1357.

33. S. Darroudi, M. Saberi-Karimian, M. Tayefi, et al., "Prevalence of Combined and Noncombined Dyslipidemia in an Iranian Population," *Journal of Clinical Laboratory Analysis* 32, no. 8 (2018): e22579, https://doi.org/10.1002/jcla.22579.

34. M. Moradinazar, Y. Pasdar, F. Najafi, et al., "Association Between Dyslipidemia and Blood Lipids Concentration With Smoking Habits in the Kurdish Population of Iran," *BMC Public Health* 20, no. 1 (2020): 1–10, https://doi.org/10.1186/s12889-020-08809-z.

35. A. Bartelt, O. Bruns, R. Reimer, et al., "Brown Adipose Tissue Activity Controls Triglyceride Clearance," *Nature Medicine* 17, no. 2 (2011): 200–205, https://doi.org/10.1038/nm.2297.

36. N. J. Woudberg, J. H. Goedecke, D. Blackhurst, et al., "Association Between Ethnicity and Obesity With High-Density Lipoprotein (HDL) Function and Subclass Distribution," *Lipids in Health and Disease* 15 (2016): 1–11, https://doi.org/10.1186/s12944-016-0257-9.

37. K. Haile and A. Timerga, "Dyslipidemia and its Associated Risk Factors Among Adult Type-2 Diabetic Patients at Jimma University Medical Center, Jimma, Southwest Ethiopia," *Diabetes, Metabolic Syndrome and Obesity* 13 (2020): 4589–4597, https://doi.org/10.2147/dmso.s283171.

38. Y. Li, L. Zhao, D. Yu, and G. Ding, "The Prevalence and Risk Factors of Dyslipidemia in Different Diabetic Progression Stages Among Middle-Aged and Elderly Populations in China," *PLoS One* 13, no. 10 (2018): e0205709, https://doi.org/10.1371/journal.pone.0205709.

39. R. A. Alzaheb and A. H. Altemani, "Prevalence and Associated Factors of Dyslipidemia Among Adults With Type 2 Diabetes Mellitus in Saudi Arabia," *Diabetes, Metabolic Syndrome and Obesity* 13 (2020): 4033–4040, https://doi.org/10.2147/dmso.s246068.

40. Feingold K. R. Obesity and Dyslipidemia. 2015.

41. J. Steinberger, C. Moorehead, V. Katch, and A. P. Rocchini, "Relationship Between Insulin Resistance and Abnormal Lipid Profile in Obese Adolescents," *Journal of Pediatrics* 126, no. 5 (1995): 690–695, https://doi.org/10.1016/s0022-3476(95)70394-2.

42. Z. Yang, X. Ding, J. Liu, et al., "Associations Between Anthropometric Parameters and Lipid Profiles in Chinese Individuals With Age ≥ 40 Years and BMI< 28kg/m^2," *PLoS One* 12, no. 6 (2017): e0178343, https://doi.org/10.1371/journal.pone.0178343.

43. B. Klop, J. W. F. Elte, and C. M. Castro, "Dyslipidemia in Obesity: Mechanisms and Potential Targets," *Nutrients* 5, no. 4 (2013): 1218–1240, https://doi.org/10.3390/nu5041218.

44. H. E. Bays, C. Kirkpatrick, K. C. Maki, et al., "Obesity, Dyslipidemia, and Cardiovascular Disease: A Joint Expert Review From the Obesity Medicine Association and the National Lipid Association 2024," *Obesity Pillars* 10 (2024): 100108, https://doi.org/10.1016/j.obpill.2024.100108.