

Prevalence of low high-density lipoproteins (HDL) cholesterol and its related factors in adult Palestinians: a cross-sectional study

Basma Damiri^{1,*}, Bayan Dudin², Qosay Sharqiah², Hashem Khelaij², Rebhi Bsharat³, Moath Amir⁴

¹ Medicine & Health Science Faculty, Physiology, Drug and Toxicology Division, An-Najah National University, Nablus, Palestine; ²Department of Medicine, An-Najah National University, Nablus, Palestine; ³ Department of Mathematics, Faculty of Science, An-Najah National University, Nablus, Palestine; ⁴ Department of Medical Laboratory, Palestinian Ministry of Health, Thabet-Thabet Hospital, Tulkarm, Palestine

*Corresponding authors: bdamiri@najah.edu

Received: (25/9/2019), Accepted: (24/10/2020)

ABSTRACT

Non-communicable diseases including cardiovascular diseases (CVD) and diabetes have become the leading causes of mortality and morbidity among Palestinians. A low level of high-density lipoproteins (HDL) is a major modifiable risk factor for CVD. This study aimed to determine the prevalence and risk factors associated with low HDL among adult Palestinians. A specific objective was to establish the prevalence of metabolic syndrome among adult Palestinians. A cross-sectional study was conducted in the West Bank from 2018 to 2019 to achieve study objectives. A total of 1086 participants (526[48.3%] women and 560[51.7%] men) aged 18-65 years were included in this study. National Cholesterol Education Program (NCEP-ATP-III) criterion was used to define metabolic syndrome. Low HDL-cholesterol was highly prevalent among Palestinians (560[51.6%]) with no differences between men (288[51.3%]) and women (272[51.8%]) (p value=0.876). Levels of HDL ranged from 13.8 to 91.4 milligrams per deciliter (mg/dl) in men with a mean level of 40.53±10.48 mg/dl and from 21.0 to 98.6 mg/dl in women with a mean level of 50.30±12.5mg/dl. According to NCEP-ATP definition, metabolic syndrome was highly prevalent among adult Palestinians (366[33.8%]) with no differences between men (192[34.2%]) and women (174[33.2%]) (p value=0.707). The univariate analysis revealed that metabolic syndrome (Odd ratio (OR), 10.79, 95% Confidence Interval (CI) (7.78-14.9)) and all increased metabolic abnormalities including triglycerides (OR,4.284, 95%CI (3.23-5.681)), fasting blood sugar (OR, 2.145, 95%CI (1.561-2.949)), blood pressure (OR, 2.133, 95%CI (1.671-2.272)), waist circumferences (OR, 2.506, 95%CI (1.937-3.242)), and obesity (OR, 2.176, 95%CI (1.685-2.809)) were significantly associated with low HDL (p value <0.001). The univariate analysis revealed also a significant association between low HDL and being married (OR, 2.183, 95%CI (1.695-2.817)), smoking (OR, 1.704, 95%CI (1.269-2.289)), and exposure to pesticides (OR, 1.702, 95%CI (1.164-2.489)) (p value <0.001). Logistic-Regression Model identified only increased triglycerides (OR, 3.341, 95%CI (2.165-5.155), p value <0.001) and increased waist circumferences (OR, 1.841, 95%CI (1.200-2.825), p value=0.005) to be significantly associated with low HDL. Although low HDL was highly prevalent among overweight and obese (412[38.08%]), it was highly prevalent among underweight and normal weight adults (147 [13.59%]). Low HDL was highly prevalent among adult Palestinians with dyslipidemia and central obesity being the most associated abnormalities. Action should be taken to prevent the rise of preventable non-communicable diseases. If no action is taken to reduce these diseases, they will become an increasing burden for the Palestinian health system.

Keywords: Cardiovascular Disease; Metabolic Syndrome; Low HDL; Central Obesity; Dyslipidemia, Palestinians, NCEP-ATP III.

INTRODUCTION

High-density lipoproteins (HDL) are endogenous nanoparticles involved in the transport and metabolism of cholesterol, phospholipids, and triglycerides [1]. It re-

moves excess cholesterol from atherosclerotic plaques and has anti-inflammatory and anti-oxidative properties that protect the cardiovascular system [2]. Circulating HDL-cholesterol also transports endogenous pro-

teins, vitamins, hormones, and microRNA to various organs [1].

The reference range of HDL levels is 40-50 milligram per deciliter (mg/dl) in men and 50-60 mg/dl in women [3]. Low levels of HDL, or hypoalphalipoproteinemia (HA), has no clear-cut definition and includes a variety of conditions, ranging from mild to severe, in which concentrations of alpha lipoproteins or HDL are reduced [4]. An arbitrary cutoff used frequently in the old literature includes a HDL level that falls within the 10th percentile of HDL levels [4]. The US National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) redefined the HDL level that constitutes a formal CHD risk factor. The minimum desirable level was raised from 35 to 40 mg/dL for both men and women [5]. For metabolic syndrome (MetS) in which multiple mild abnormalities in lipids, waist size (abdominal circumference), blood pressure, and blood sugar increase the risk of CAD, the designated HDL cholesterol levels that contribute to the syndrome are sex-specific [6]. For men, a high-risk HDL cholesterol level is less than 40 mg/dl, but for women, the high-risk HDL cholesterol level is less than 50 mg/dl [7, 8].

Several epidemiological and genetic studies confirmed the association between elevated HDL levels and protection against atherogenesis [9, 10]. On the other hand, a low level of serum HDL cholesterol was established to be an independent risk factor for coronary artery disease (CAD) [11, 12]. It is shown to be a key component in the prediction of cardiovascular disease (CVD) [13, 14]. Moreover, there is evidence that low HDL-cholesterol is in association with metabolic syndrome and type 2 diabetes patients [15]. It has been estimated that more than 80% of all CVD mortality occurs in developing countries [16]. Moreover, emerging data from developing countries suggest that low HDL may be the most common lipid abnormality observed in these societies [17].

Many risk factors have influence on HDL levels. The etiology of HDL deficiency ranges from secondary factors to specific genetic mutations, such as Tangier disease and fish-eye disease [18, 19]. Secondary factors such as gender, age, obesity, smoking, alcohol, diet, starvation, stress, physical

activity, drugs (e.g., steroids, statins, and fibrates) or other metabolic disorders (e.g., insulin resistance and liver disease) have been shown to influence HDL levels in numerous epidemiological studies [20-28]. Diseases that affect levels of HDL include hypertriglyceridemia, uncontrolled diabetes mellitus (DM), metabolic syndrome and chronic renal failure [29-31]. Environmental influences are known to regulate HDL cholesterol levels; however, genetic factors are also known to be important [17]. Levels of HDL appear to be under a strong inherited basis, with heritability estimates of 40–60% [32-34]. Genetic variants may also contribute to inter-individual variability in HDL response to environmental factors [17]. Genes are modulated by several non-modifiable factors such as gender and age and modifiable factors, such as diet, smoking, obesity, and alcohol intake among many others.

Cardiovascular disease has become the most common leading cause of mortality among Palestinians. Few studies were conducted to describe metabolic syndrome in vulnerable groups had described the percentages of low HDL among Palestinians. Low HDL percentages had ranged from 28.6% among refugee women in the West Bank [35] to 74.7% among overweight and obese young adults [36]. Low HDL-level was the second most common component of MetS after central obesity among overweight and obese subjects [36-39]. Factors behind this low HDL in the Palestinian population were not investigated.

To the authors' best knowledge, no studies were conducted to establish the prevalence of low HDL cholesterol and to investigate its associated risk factors in Palestinians. Such studies are important in drawing protective policies. Large-scale prospective studies have indicated that each 1 mg/dl increase in HDL is associated with a two to three percent decrease in the risk of CAD in men and women, respectively [40]. This study aimed to determine the prevalence of low HDL and the risk factors associated with it among the adult population aged 18-65 years in the West Bank. A specific objective was to determine levels of low HDL as well as the association between low HDL and lifestyle and metabolic abnormalities using the National

Cholesterol Education Program (NCEP-ATP III) criterion.

METHODS

Study design, population, and sampling technique

A cross-sectional study was conducted in the north of the West Bank from 2018 to 2019. There were 296058 adults in the north of the West Bank aged >18 years (50.9% males and 49.1% females) [41]. Ten health care clinics distributed in the north of the West Bank were chosen randomly to recruit 1384 subjects. Apparently healthy subjects aged 18-65 years who attend health care clinics in the north of the West Bank were recruited during the study period. To make sure that the subjects were healthy, they were interviewed and asked if they had diseases, types of medications they take regularly, if they had any surgery in the last year, if they had health care files in the health care clinic, the reason for coming to health care clinics, and if they had certain diseases or conditions as explained in the exclusion criteria. Every third apparently healthy subject was chosen to participate in the study and was interviewed for personal, socio-demographic, and lifestyle questions. The subject was also invited to give blood samples after fasting for 12 hours. Subjects with hypo- or hyperthyroidism, Cushing syndrome, epilepsy, or who was taking regular medications other than anti-diabetic or anti-hyperlipidemia medications or refused to give a blood sample were excluded.

Blood analysis, measurement tools accuracy and precision assessment

The National Cholesterol Education Program (NCEP-ATP III) criterion was used to diagnose metabolic syndrome in this study. Briefly, NCEP-ATP III proposed that the individual must have at least three of the following cardiovascular risk factors to be diagnosed with MetS: Raised fasting blood glucose ≥ 110 milligrams per deciliter, raised blood pressure: systolic BP ≥ 130 or diastolic BP ≥ 85 millimeter of mercury (mm Hg), raised triglycerides ≥ 150 (mg/dl), low HDL cholesterol (HDL) (men < 40 milligrams per deciliter, women < 50 mg/dl, and increased waist circumference ≥ 102 centimeters in men or ≥ 88 centimeters in women. Blood

analysis was carried out in An-Najah National Hospital Laboratories. Blood samples were collected and analyzed for blood sugar, triglycerides, and HDL using "Roche Chemistry Analyzer Cobas C 50.1). Accuracy and precision of both anthropometric tools and the questionnaire were assessed. Interviews were conducted to avoid possible language and literacy issues. The diagnostic criteria, anthropometrics, and blood pressure measurements, venous blood collection and biochemical analysis, and measurement tools accuracy and precision assessment were used and published in previous works [36, 39, 42]. Current substance user for tobacco or alcohol is a substance user who has used the substance at least once during the past 30 days. Exposed to pesticides is any participant who was exposed to pesticides through his occupation such as farmers.

Data analysis

Descriptive statistics were computed to assess the personal characteristics of the participants. Univariate analysis was conducted using a Chi-square test with an odds ratio (OR) calculated for risk factors. Multivariate logistic regression was performed for variables found to be significant in univariate analysis. A P-value of < 0.05 was considered to indicate statistical significance and a confidence interval was set at 95%. Statistical Product and Service Solutions (SPSS) (version 22, IBM Corporation) was used for data entry and analysis.

Ethics

Ethical approval was obtained from Institutional Review Board "IRB" at An-Najah National University in Palestine and additional approvals were obtained from the Palestinian Ministry of Health before research conduction. The study was carried out following the ethical standards, Declarations of Helsinki. A consent form was obtained from each participant prior to participation. All participants were assured that all data will be confidential and available for the researcher only. The blood tests were free of charge.

RESULTS

General characteristics

The total number of participants in this study was 1086 (526[48.3%] women and

560[51.7%] men) with a response rate of 80.2%. Six hundred and ninety-one (63.4%) of them aged 18-40 years, 707(65.3%) were overweight and obese, 388(35.9%) were singles, 400(36.8%) were urban, 609(56.1%) were rural, 77(7.1%) were refugees, 239(22.9%) were current smokers and the duration of smoking ranged from around one year to 53 years, 7(0.7%) were current alcohol users, 520(48%) were employed, 180(16.6%) were students, 129(11.9%) were using pesticides in their work, 80(7.4%) walk every day for two hours or more, 789(72.2%) of them sleep for eight hours or more per day, and 365(33.7%) sit in front of televisions or computers more than or equal to four hours per day (Table 1).

Table (1): Socio-demographic, life-style, characteristics of respondents.

Gender	n (%)
Male	560(51.7)
Female	526(48.3)
Age groups	
18-40 years	691(63.4)
41-65 years	391(36.4)
Smoking tobacco; cigarettes or water pipes	
Current smoker	239 (22.9)
Ex-smoker	206(19.0)
Alcohol intake	
Current user	7(0.7)
Ex-user	14(1.3)
Marital status	
Single	388(35.9)
Married	681(62.9)
Divorced or widow	13(1.2)
BMI	
≥25	707(65.3)
<25	375(34.7)
Work and physical activity	
Employed	520(48.0)
Unemployed	382(35.2)
Students	180(16.6)
Occupational exposure to pesticides	129(11.9)
Sleep >8 hours/ day	789(72.2)
Walk ≥2 hours/day	80(7.4)
Regular physical activity ≥5	121(11.2)

Gender	n (%)
times/week	
Long sitting ≥4hours/day (e.g. in front of televisions or computer)	365(33.7)
Locality	
City	400(36.8)
Village	609(56.1)
Camp	77(7.1)

Prevalence of metabolic syndrome, metabolic abnormalities, and levels of HDL according to NCEP-ATPIII definition

Obesity was highly prevalent among adult Palestinians (706[65.2%]) with no significant differences between men (374[67.1%]) and women (332[63.2%]) (p value= 0.182) (Table 2). According to NCEP-ATP definition, metabolic syndrome was highly prevalent among adult Palestinians (366[33.8%]) with no significant differences between men (192[34.2%]) and women (174[33.2%]) (p value= 0.707). The most prevalent metabolic abnormalities were low HDL (560[51.6%]) with no differences between men (288[51.3%]) and women (272[51.8%]) (p value= 0.876), followed by increased blood pressure (494[45.5%]) with significant increase among men (281[50.1%]) compared to women (216[40.6%]) (p value= 0.002), and increased waist circumferences (390[35.9%]) with significant increase in women (233[42.3%]) compared to men (167[29.8%]) (p value <0.001), and increased triglycerides (349[32.2%]) with significant increase in men (220[39.3%]) compared to women (129[24.6%]) (p value <0.001). The least prevalent metabolic abnormality was increased fasting blood sugar (206[19%]) with no significant differences between men (110[19.6%]) and women (96[18.3%]) (p value= 0.578) (Table 2). Levels of HDL ranged from 13.8 to 91.4 milligrams per deciliter (mg/dl) in the men with a mean level of 40.53 (mg/dl) and ranged from 21.0 to 98.6 (mg/dl) in women with a mean level of 50.3 mg/dl (Table 2). With increasing body mass index, the prevalence of low HDL had significantly increased (p value <0.001) in both men (p value= 0.011) and women (p value <0.001). It was 0.7% among underweight, 12.8% among normal weight, 15.8% among overweight, and 22.1% among obese (Table 2).

Table (2): Prevalence of metabolic syndrome, metabolic abnormalities, and levels of HDL according to NCEP-ATPIII definition.

Metabolic syndrome and metabolic abnormalities	Men n(%)	Women n(%)	Total n(%)	P-value
Metabolic syndrome (MetS)	192(34.2)	174(33.2)	366(33.8)	0.707
Low high-density lipoproteins (HDL)	288(51.3)	272(51.8)	560(51.6)	0.876
High triglycerides	220(39.3)	129(24.6)	349(32.2)	<0.001
High blood pressure (BP)	281(50.1)	213(40.6)	494(45.5)	0.002
Increased waist circumferences (WC)	167(29.8)	233(42.3)	390(35.9)	<0.001
High fasting blood sugar (FBS)	110(19.6)	96(18.3)	206(19.0)	0.578
Body mass index (BMI) \geq 25	374(67.1)	332(63.2)	706(65.2)	0.182
Levels of HDL in mg/dl				
10-19.9	5(0.9)	0(0)	5(0.5)	
20-29.9	62(11.1)	25(4.8)	87(8)	
30-39.9	221(39.4)	75(14.3)	296(27.3)	
40-49.9	192(34.2)	176(33.5)	368(33.9)	
50-59.9	59(10.5)	130(24.8)	189(17.4)	
\geq 60	22(3.9)	119(22.7)	141(13.0)	
Mean \pm SD	40.53 \pm 10.48	50.30 \pm 12.5		
Classes of Body Mass Index				
Under-weight	2(0.4)	6(1.1)	8(0.74)	
Normal-weight	84(15.0)	55(10.5)	139(12.8)	
Over-weight	98(17.5)	74(14.1)	172(15.8)	
Obese	103(18.4)	137(26.0)	240(22.1)	

Abbreviations: mg/dl: Milligrams Per Deciliter, HDL: High-Density Lipoproteins, SD: Standard Deviation

Univariate analysis of metabolic abnormalities, lifestyle, and environmental risk factors

For metabolic abnormalities factors, the univariate analysis revealed that metabolic syndrome (Odd ratio (OR, 10.79, 95% Confidence Interval (CI) (7.78-14.9)) and all increased metabolic abnormalities including triglycerides (OR, 4.284, 95% CI (3.23-5.681)), high fasting blood sugar (OR, 2.145, 95% CI (1.561-2.949)), high blood pressure (OR, 2.133, 95% CI (1.671-2.2721)), increased waist circumferences (OR, 2.506, 95% CI (1.937-3.242)), and obesity (OR,

2.176, 95% CI (1.685-2.809)) were significantly associated with low HDL (p value <0.001) (Table3). For lifestyle and environmental factors, the univariate analysis revealed a significant association between low HDL and marital status for married (OR, 2.183, 95% CI (1.695-2.817)), current tobacco smoking (OR, 1.704, 95% CI (1.269-2.289)), occupational exposure to pesticides (OR, 1.702, 95% CI (1.164-2.489)) (p value <0.001). Gender, alcohol intake, physical activity, and sedentary lifestyle had shown no association with low HDL (Table 3).

Table (3): Univariate analysis of metabolic abnormalities factors.

Metabolic abnormalities	N (%)	OR	95%(CI)	P-value
Triglyceride				
High	260(46.6)	4.284	3.23-5.681	<0.001
Normal	298(53.4)			
Blood pressure (BP)				
High	305(54.4)	2.133	1.671-2.2721	<0.001
Normal	255(45.5)			

Metabolic abnormalities	N (%)	OR	95%(CI)	P-value
Systolic blood pressure (SBP)				
High	254(45.5)	2.314	1.792-2.988	<0.001
Normal	304(54.4)			
Diastolic blood pressure (DBP)				
High	196(35.1)	1.823	1.395-2.388	<0.001
Normal	362(64.9)			
Waist circumferences (WC)				
High	257(45.9)	2.506	1.937-3.242	<0.001
Normal	303(54.1)			
Fasting blood sugar (FBS)				
High	137(24.5)	2.145	1.561-2.949	<0.001
Normal	432(75.5)			
Body mass index (BMI)				
≥25	412(73.7)	2.176	1.685-2.809	<0.001
<25	147(26.3)			
Metabolic syndrome (MetS)				
Yes	311(55.7)	10.78	7.78-14.93	<0.001
No	247(44.3)			
Social, life- style, and environmental factors				
Gender				
Male	288(51.8)	0.981	0.773-1.245	0.876
Female	272(48.6)			
Marital status				
Single	152(27.1)	0.458	0.355-0.590	<0.001
Married, divorced, widow	408(72.9)			
Current tobacco smoking				
Yes	147(27.4)	1.704	1.269-2.289	<0.001
No	390(72.6)			
Alcohol intake				
Yes	8(1.5)	1.272	0.438-3.693	0.760
No	526(98.5)			
Exposure to occupational pesticide				
Yes	81(15.1)	1.702	1.164-2.489	0.006
No	456(84.9)			
Sleep>8 hours/day				
Yes	369(74.0)	0.805	0.605-1.071	0.136
No	139(26.0)			
Walk≥2 hours/day				
Yes	43(14.3)	1.131	0.705-1.814	0.610
No	258(85.9)			
Regular physical activity ≥5 times/week				
Yes	53(9.9)	0.707	0.483-1.035	0.074
No	484(90.1)			
Long sitting ≥4hours/day				
Yes	180(33.5)	0.878	0.680-1.132	0.315
No	357(66.5)			

Abbreviations: OR: Odds Ratio, CI: Confident Interval

Multivariate logistic regression analysis

The multivariate logistic regression model included all variables found to be significant in the univariate analysis; increased fasting blood sugar, increased serum triglycerides, increased blood pressure, increased waist circumferences, increased BMI, marital status, tobacco smoking, and exposure to occupational pesticides. Controlling for all the

above-mentioned variables, the Logistic-Regression Model identified only increased triglycerides (OR, 3.341, 95%CI (2.165-5.155), p value <0.001)) and increased waist circumferences (OR, 1.841, 95%CI (1.200-2.825), p value=0.005)) to be significantly associated with low HDL (Table 4).

Table (4): Logistic regression model of risk factors for low HDL.

Age	OR	95%(CI)	P-value
	1.001	0.981-1.020	0.959
Triglycerides High Normal	3.341 Reference group	2.165-5.155	<0.001
Blood pressure (BP) High Normal	1.153 Reference group	0.770-1.726	0.491
Waist circumferences (WC) High Normal	1.841 Reference group	1.200-2.825	0.005
Fasting blood sugar (FBS) High Normal	1.038 Reference group	0.612-1.759	0.890
Marital status Single Married, divorced, widow	0.646 Reference group	0.396-1.055	0.081
Tobacco smoking Yes No	1.0304 Reference group	0.859-1.980	0.212
Exposure to occupational pesticide Yes No	1.017 Reference group	0.606-1.707	0.948
Walk \geq 2 hours/day Yes No	1.118 Reference group	0.666-1.876	0.672
Address City Village Camps	0.496 0.404 Reference group	0.242-1.019 0.404-0.200	0.56 0.011

*reference category is participants with normal HDL

Abbreviations: OR: Odds Ratio, CI: Confident Interval

DISCUSSION

The results of this study had revealed that the prevalence of low HDL was high among adult Palestinians (51.5%) in both men and women with no gender differences. Low HDL is shown to be a key component in the prediction of cardiovascular disease risk and may trigger the pathogenesis of metabolic syndrome [13, 14]. It carries the strongest risk in predicting the development of metabolic syndrome and might directly affect glucose metabolism [15, 43, 44]. Metabolic syndrome has also been demonstrated as a common precursor to the development of diabetes mellitus (DM) and cardiovascular diseases [45]. In this study, the prevalence of metabolic syndrome was high and strongly associated with low HDL levels. Therefore, the results of this study indicate that at least half of the adult Palestinian population could be at risk of CVD. Moreover, around 85% of the par-

icipants with low HDL had MetS. A previous study was conducted in Gaza Strip-Palestine affirmed that 98% of low HDL population with type 2 diabetes are diagnosed with MetS [46]. These results outweigh the prevalence of low HDL among MetS patients worldwide [47-49].

The univariate analysis revealed that low HDL was highly associated with metabolic abnormalities, which is consistent with other studies [15, 35, 36, 39, 42, 50]. Non-communicable diseases including cardiovascular diseases and diabetes have become the leading causes of mortality and morbidity among Palestinians. They have resulted in substantial direct morbidity and mortality in occupied Palestinian territories [51]. The prevalence of diabetes was estimated to be high (15.3%) among Palestinians compared to a worldwide prevalence (6%) [52]. Cardiac disease was reported to be the number one

cause of death in the occupied Palestinian territories, accounting for 21.0% of all deaths. Hypertension was ranked eighth and accounting for about 5% of all deaths [53]. Therefore, low HDL, as well as metabolic syndrome, are increasingly becoming a challenging public health issue in Palestine [51]. The low HDL and its many consequences, including metabolic syndrome will continue to increase unless we can find a way to prevent obesity and metabolic syndrome in adults.

Although decreased HDL had increased with increasing BMI and metabolic abnormalities, it was also highly prevalent among normal-weight adults indicating that other factors could influence low HDL among Palestinians. This line of results takes on added importance because these individuals are frequently undetected and undiagnosed because of their normal BMI. This also indicates that low HDL level is becoming an increasingly prominent feature not only in MetS patients but also in general population. Therefore, screening in individuals with a normal BMI is important in preventing diabetes and cardiovascular diseases. Action should be taken to prevent and therefore, to slow the rise of metabolic abnormalities. If no action is taken to reduce these abnormalities, they will become an increasing burden for the health system.

It is known that levels of HDL are modifiable through behavioral factors including diet, physical activity, alcohol intake, smoking, and exposure to pesticides [20, 23, 54]. In terms of risk factors, the univariate analysis revealed that tobacco smoking, occupational exposure to pesticides, and being married were associated with low HDL. It has also been demonstrated previously that smoking cessation is associated with an up to 10% increase in HDL level [55]. Moreover, low HDL is more prevalent among married than single men and women [46, 49, 56]. These factors of low HDL level are attractive targets for approaches to increase HDL. It can increase through several lifestyle changes. The most important among these are smoking cessation and increased physical activity especially in those who are obese and overweight [57]. It was estimated that for every 1 kg of weight loss, serum HDL increases by

0.35 mg/dl [58, 59]. Therefore, none pharmacologic approaches that can effectively increase serum levels of HDL including weight control, specific nutritional choices, exercise, and smoking cessation should be adapted [59].

By using logistic regression analysis, only dyslipidemia and central obesity remained independent risk factors for low HDL. This agrees with previous studies which indicated that central obesity increases with age and is more frequent in women; the higher the central obesity, the lower the concentration of HDL [60]. These results also agree with other studies that revealed no association with HDL levels and blood pressure [61]. Dyslipidemias were reported to be strongly associated with low HDL, metabolic syndrome and thus CVD [62, 63]. The high prevalence and disease burden of elevated serum triglyceride and central obesity highlight the need for implementation of multi-component interventions to reduce the prevalence and disease burden of these abnormalities. More research investigating factors associated with dyslipidemia among Palestinians is recommended.

Many studies showed that those who are genetically predisposed to have low HDL levels have HDL in the range of less than 30 mg/dl [64-67]. Only 16% of the participants were with HDL levels less than 30 mg/dl and they were more likely to be men, obese and aged 40 years or more. Genetic factors were not investigated directly in this study and thus, further genetic studies for low HDL are recommended. We also recommend spending more efforts in developing targeting preventive interventions and awareness campaigns for key risk factors for non-communicable diseases. Moreover, we recommend further studies to be conducted in different areas in the West Bank. Finally, we recommend genetic studies to be conducted for dyslipidemia. In light of higher rates of overweight, obesity, central obesity, and metabolic abnormalities in adults, more research is needed for the south of the West Bank. We recommend screening adults using increased waist circumference.

Limitations

Even though the strength of this study is its large scale was 1082 participants, this

study has some limitations. Genetic factors were not investigated. Ongoing research to investigate genetic factors was established. Another possible limitation is that we were not able to assess the diet as a risk factor. Further studies are needed in this area.

Conclusion

The prevalence of low HDL and metabolic syndrome was high. Although low HDL had increased with increasing body mass index and metabolic abnormalities, it was also highly prevalent among underweight and normal-weight adults indicating that other factors could influence decreased HDL among Palestinians. This study found higher than what anticipated prevalence of low HDL in normal-weight adults. Action should be taken to prevent and therefore, to slow the rise of preventable non-communicable diseases such as obesity, diabetes, hypertension, and dyslipidemia and thus, cardiovascular diseases. If no action is taken to reduce these diseases, these diseases will become an increasing burden for the health system. The results of this study have important clinical implications for screening adults using increased waist circumference.

Competing of Interests: The authors declare that they have no competing interests.

Ethical Considerations: The study was carried out in accordance with the ethical standards, Declarations of Helsinki. Approval was obtained from Institutional Review Board "IRB" at An-Najah National University in Palestine prior to the research conduction. Approval of Palestinian Ministry of Health was taken prior to research conduction. All study participants were freely accepted to join the study and they provided a signed consent form. All were assured that all collected data will be confidential and available for the researcher only. It was explained to the participants that they had the right to withdraw from the research anytime. Blood tests were free of charge.

Authors' Contributors

B. Damiri wrote the initial draft of the manuscript. B. Damiri, B. Dudin, Q. Sharqiah, H. Khlaif, M. Amer contributed to the study design, literature the search and carried out the data collection. B. Damiri and R. Bsharat analyzed the data and prepared data tables. All authors were involved in in-

terrupting the data and had full approval of the submitted and published version and all authors approved the final manuscript.

ACKNOWLEDGMENTS

We are grateful to Mr Kahlid Abu Khatir, Dr. Qasem Daghlas, and Rabe'e Noor for their help.

REFERENCES

- 1) Kuai R, Li D, Chen YE, Moon JJ, Schwendeman A. High-Density Lipoproteins: Nature's Multifunctional Nanoparticles. *ACS Nano*. 2016; 10(3): 3015-3041.
- 2) Fisher EA, Feig JE, Hewing B, Hazen SL, Smith JD. High-density lipoprotein function, dysfunction, and reverse cholesterol transport. *Arterioscler Thromb Vasc Biol*. 2012; 32(12): 2813-2820.
- 3) Williamson MA, Snyder LM. *Wallach's Interpretation of Diagnostic Tests: Pathways to Arriving at a Clinical Diagnosis*: Lippincott Williams & Wilkins; 2014.
- 4) Singh V, Sharma R, Kumar A, Deedwania P. Low high-density lipoprotein cholesterol: current status and future strategies for management. *Vasc Health Risk Manag*. 2010; 6: 979-996.
- 5) Expert Panel on Detection E, Treatment of High Blood Cholesterol in A. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA*. 2001; 285(19): 2486-2497.
- 6) Beigh SH, Jain S. Prevalence of metabolic syndrome and gender differences. *Bioinformation*. 2012; 8(13): 613-616.
- 7) Karalis DG. Intensive lowering of low-density lipoprotein cholesterol levels for primary prevention of coronary artery disease. *Mayo Clin Proc*. 2009; 84(4): 345-352.
- 8) Azizi F, Rahmani M, Emami H, Mirmiran P, Hajipour R, Madjid M, et al. Cardiovascular risk factors in an Iranian urban population: Tehran lipid

- and glucose study (phase 1). *Soz Praventivmed.* 2002; 47(6): 408-426.
- 9) Ansell BJ, Watson KE, Fogelman AM, Navab M, Fonarow GC. High-density lipoprotein function recent advances. *J Am Coll Cardiol.* 2005; 46(10): 1792-1798.
 - 10) Soran H, Schofield JD, Durrington PN. Antioxidant properties of HDL. *Front Pharmacol.* 2015; 6: 222.
 - 11) Hadaegh F, Harati H, Ghanbarian A, Azizi F. Prevalence of coronary heart disease among Tehran adults: Tehran Lipid and Glucose Study. *East Mediterr Health J.* 2009; 15(1): 57-66.
 - 12) Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. *Am J Med.* 1977; 62(5): 707-714.
 - 13) Rader DJ, Hovingh GK. HDL and cardiovascular disease. *Lancet.* 2014; 384(9943): 618-625.
 - 14) Yusuf S, Reddy S, Ôunpoo S, Anand S. Global burden of cardiovascular diseases: part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation.* 2001; 104(22): 2746-2753.
 - 15) Marjani A. A Review on HDL-cholesterol Alterations in Metabolic Syndrome. *J Biol Sci.* 2013; 13(8): 679-684.
 - 16) Graham I, Cooney MT, Bradley D, Dudina A, Reiner Z. Dyslipidemias in the prevention of cardiovascular disease: risks and causality. *Curr Cardiol Rep.* 2012; 14(6):709-720.
 - 17) Ordovas JM. HDL genetics: candidate genes, genome wide scans and gene-environment interactions. *Cardiovasc Drugs Ther.* 2002; 16(4): 273-281.
 - 18) McIntyre N. Familial LCAT deficiency and fish-eye disease. *J Inherit Metab Dis.* 1988; 11 Suppl 1:45-56.
 - 19) Puntoni M, Sbrana F, Bigazzi F, Sampietro T. Tangier disease: epidemiology, pathophysiology, and management. *Am J Cardiovasc Drugs.* 2012; 12(5): 303-311.
 - 20) Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking intensity and lipoprotein abnormalities in active smokers. *J Clin Lipidol.* 2009; 3(6): 372-378.
 - 21) T S-D, RE K. Exercise intensity: its effect on the high-density lipoprotein profile. *Arch Phys Med Rehabil.* 1999; 80(6): 691-695.
 - 22) Hausenloy DJ, Yellon DM. Targeting residual cardiovascular risk: raising high-density lipoprotein cholesterol levels. *Heart.* 2008; 94(6): 706-714.
 - 23) Lee D-H, Steffes MW, Sjödin A, Jones RS, Needham LL, Jacobs DR, Jr. Low dose organochlorine pesticides and polychlorinated biphenyls predict obesity, dyslipidemia, and insulin resistance among people free of diabetes. *PloS one.* 2011; 6(1): e15977-e15977.
 - 24) Kanel GC, Radvan G, Peters RL. High-density lipoprotein cholesterol and liver disease. *Hepatology.* 1983;3(3):343-348.
 - 25) S R, J G. Effect of obesity on high-density lipoprotein metabolism. *Obesity (Silver Spring).* 2007; 15(12): 2875-2888.
 - 26) Hausenloy DJ, Yellon DM. Targeting residual cardiovascular risk: raising high-density lipoprotein cholesterol levels. *Heart.* 2008; 94(6): 706-714.
 - 27) Lehtonen A. Effect of beta blockers on blood lipid profile. *Am Heart J.* 1985; 109(5 Pt 2): 1192-1196.
 - 28) Hartgens F, Rietjens G, Keizer HA, Kuipers H, Wolffenbuttel BH. Effects of androgenic-anabolic steroids on apolipoproteins and lipoprotein (a). *Br J Sports Med.* 2004; 38(3): 253-259.
 - 29) Verges B. Pathophysiology of diabetic dyslipidaemia: where are we? *Diabetologia.* 2015; 58(5): 886-899.
 - 30) Reiss AB, Miyawaki N, Moon J, Kasselman LJ, Voloshyna I, D'Avino R, Jr., et al. CKD, arterial calcification, atherosclerosis and bone health: Interrelationships and controversies. *Atherosclerosis.* 2018; 278: 49-59.
 - 31) Borja MS, Hammerson B, Tang C, Savinova OV, Shearer GC, Oda MN. Apolipoprotein A-I exchange is impaired in metabolic syndrome patients asymptomatic for diabetes and

- cardiovascular disease. *PLoS One*. 2017; 12(8): e0182217.
- 32) Weissglas-Volkov D, Pajukanta P. Genetic causes of high and low serum HDL-cholesterol. *J Lipid Res*. 2010; 51(8): 2032-2057.
- 33) Qasim A, Rader DJ. Human genetics of variation in high-density lipoprotein cholesterol. *Curr Atheroscler Rep*. 2006; 8(3): 198-205.
- 34) Lusis AJ, Mar R, Pajukanta P. Genetics of atherosclerosis. *Annu Rev Genomics Hum Genet*. 2004;5:189-218.
- 35) Massad SG, Khalili M, Karmally W, Abdalla M, Khammash U, Mehari GM, et al. Metabolic Syndrome among Refugee Women from the West Bank, Palestine: A Cross-Sectional Study. *Nutrients*. 2018; 10(8).
- 36) Damiri B, Aghbar A, Alkhdour S, Arafat Y. Characterization and prevalence of metabolic syndrome among overweight and obese young Palestinian students at An-Najah National University. *Diabetes Metab Syndr*. 2018;12(3):343-348.
- 37) Abu Sham'a RA, Darwazah AK, Kufri FH, Yassin IH, Torok NI. MetS and cardiovascular risk factors among Palestinians of East Jerusalem. *East Mediterr Health J*. 2009; 15(6): 1464-1473.
- 38) Sweileh WM, Zyoud SH, Dalal SA, Ibwini S, Sawalha AF, Ali I. Prevalence of metabolic syndrome among patients with schizophrenia in Palestine. *BMC Psychiatry*. 2012; 12(1): 235.
- 39) Damiri B, Abualsoud MS, Samara AM, Salameh SK. Metabolic syndrome among overweight and obese adults in Palestinian refugee camps. *Diabetol Metab Syndr*. 2018; 10:34.
- 40) Mahdy Ali K, Wonnerth A, Huber K, Wojta J. Cardiovascular disease risk reduction by raising HDL cholesterol--current therapies and future opportunities. *Br J Pharmacol*. 2012; 167(6): 1177-1194.
- 41) PCBS PPCBos. Preliminary Results of the Population, Housing and Establishments Census, 2017. Ramallah, Palestine; 2018.
- 42) Damiri B, Abu Alhala A, Najjar L, Alqadome S. Metabolic Syndrome and its Risk Factors among Overweight and Obese Palestinian Schoolchildren using IDF and NCEP-ATP/III Definitions. *Ann Clin Lab Res*. 2018; 06(03): 8.
- 43) Castaneda G, Bhuket T, Liu B, Wong RJ. Low serum high density lipoprotein is associated with the greatest risk of metabolic syndrome among U.S. adults. *Diabetes Metab Syndr*. 2018; 12(1): 5-8.
- 44) Drew BG, Rye KA, Duffy SJ, Barter P, Kingwell BA. The emerging role of HDL in glucose metabolism. *Nat Rev Endocrinol*. 2012; 8(4): 237-245.
- 45) Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*. 2005; 112(17): 2735-2752.
- 46) El Bilbeisi AH, Hosseini S, Djafarian K. Dietary Patterns and Metabolic Syndrome among Type 2 Diabetes Patients in Gaza Strip, Palestine. *Ethiop J Health Sci*. 2017; 27(3): 227-238.
- 47) Bruckert E. Epidemiology of low HDL-cholesterol: results of studies and surveys. *European Heart Journal Supplements*. 2006; 8(suppl_F): F17-F22.
- 48) Ge P, Dong C, Ren X, Weiderpass E, Zhang C, Fan H, et al. The high prevalence of low HDL-cholesterol levels and dyslipidemia in rural populations in northwestern China. *PLoS one*. 2015; 10(12): e0144104.
- 49) Kim SM, Han JH, Park HS. Prevalence of low HDL-cholesterol levels and associated factors among Koreans. *Circ J*. 2006; 70(7): 820-826.
- 50) Parhofer KG. Interaction between Glucose and Lipid Metabolism: More than Diabetic Dyslipidemia. *Diabetes Metab J*. 2015; 39(5): 353-362.
- 51) El Bilbeisi AH, Shab-Bidar S, Jackson D, Djafarian K. The Prevalence of Metabolic Syndrome and Its Related Factors among Adults in Palestine: A Meta-Analysis. *Ethiop J Health Sci*. 2017; 27(1): 77-84.

- 52) Abu-Rmeileh NM, Husseini A, Capewell S, O'Flaherty M, project M. Preventing type 2 diabetes among Palestinians: comparing five future policy scenarios. *BMJ Open*. 2013; 3(12): e003558.
- 53) Husseini A, Abu-Rmeileh NM, Mikki N, Ramahi TM, Ghosh HA, Barghuthi N, et al. Cardiovascular diseases, diabetes mellitus, and cancer in the occupied Palestinian territory. *Lancet*. 2009; 373(9668): 1041-1049.
- 54) Mann S, Beedie C, Jimenez A. Differential effects of aerobic exercise, resistance training and combined exercise modalities on cholesterol and the lipid profile: review, synthesis and recommendations. *Sports Med*. 2014; 44(2): 211-221.
- 55) Barter PJ. The causes and consequences of low levels of high density lipoproteins in patients with diabetes. *Diabetes Metab J*. 2011; 35(2): 101-106.
- 56) Mirmiran P, Mohammadi F, Allahverdian S, Azizi F. Association of educational level and marital status with dietary intake and cardiovascular risk factors in Tehranian adults: Tehran lipid and glucose study (TLGS). *Nutrition Research*. 2002; 22(12): 1365-1375.
- 57) Kodama S, Tanaka S, Saito K, Shu M, Sone Y, Onitake F, et al. Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: a meta-analysis. *Arch Intern Med*. 2007; 167(10): 999-1008.
- 58) Singh IM, Shishehbor MH, Ansell BJ. High-density lipoprotein as a therapeutic target: a systematic review. *JAMA*. 2007; 298(7): 786-798.
- 59) Ginsberg HN. Nonpharmacologic management of low levels of high-density lipoprotein cholesterol. *Am J Cardiol*. 2000; 86(12a): 411-451.
- 60) Arimura ST, Moura BM, Pimentel GD, Silva ME, Sousa MV. Waist circumference is better associated with high density lipoprotein (HDL-c) than with body mass index (BMI) in adults with metabolic syndrome. *Nutr Hosp*. 2011; 26(6): 1328-1332.
- 61) Saidu H, Karaye KM, Okeahialam BN. Plasma lipid profile in Nigerians with high - normal blood pressure. *BMC Research Notes*. 2014; 7(1): 930.
- 62) Steinhagen-Thiessen E, Bramlage P, Losch C, Hauner H, Schunkert H, Vogt A, et al. Dyslipidemia in primary care--prevalence, recognition, treatment and control: data from the German Metabolic and Cardiovascular Risk Project (GEMCAS). *Cardiovasc Diabetol*. 2008; 7(1): 31.
- 63) Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation*. 2009; 120(16): 1640-1645.
- 64) Real JT, Martinez-Hervas S, Garcia-Garcia AB, Chaves FJ, Civera M, Ascaso JF, et al. Association of C677T polymorphism in MTHFR gene, high homocysteine and low HDL cholesterol plasma values in heterozygous familial hypercholesterolemia. *J Atheroscler Thromb*. 2009; 16(6): 815-820.
- 65) Lazo-Porrás M, Bernabe-Ortiz A, Malaga G, Gilman RH, Acuna-Villaorduna A, Cardenas-Montero D, et al. Low HDL cholesterol as a cardiovascular risk factor in rural, urban, and rural-urban migrants: PERU MIGRANT cohort study. *Atherosclerosis*. 2016; 246: 36-43.
- 66) Linsel-Nitschke P, Tall AR. HDL as a target in the treatment of atherosclerotic cardiovascular disease. *Nat Rev Drug Discov*. 2005; 4(3): 193-205.
- 67) Santos RD, Schaefer EJ, Asztalos BF, Polisecki E, Wang J, Hegele RA, et al. Characterization of high density lipoprotein particles in familial apolipoprotein A-I deficiency. *J Lipid Res*. 2008; 49(2): 349-357.