Article published online: 2022-07-07

Original Article

High Prevalence of Dyslipidemia Irrespective of Obesity in the Cape Coast Metropolis of Ghana

Samuel Acquah, Benjamin Ackon Eghan[1], Johnson Nyarko Boampong[2]

Department of Medical Biochemistry, School of Medical Sciences, [2]Department of Biomedical Science, School of Allied Health Sciences, College of Health and Allied Sciences, University of Cape Coast, Cape Coast, [1]Department of Medicine, School of Medical Sciences, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana

Abstract

Objective: To investigate the relationship between obesity, insulin resistance, and lipid profile in type 2 diabetes patients and nondiabetic controls in the Cape Coast Metropolis of Ghana. **Patients and Methods:** Levels of fasting blood glucose (FBG), glycosylated hemoglobin, lipid profile, insulin resistance, and β-cell function were measured in 115 diabetes patients and 115 age-matched nondiabetic controls. In addition, body weight, height, waist circumference (WC), hip circumference, and blood pressure were measured. Body mass index and waist-to-hip ratio were calculated. **Results:** Apart from diabetes patients with normal weight who exhibited higher ($P < 0.05$) FBG but lower systolic blood pressure than their overweight/obese counterparts, levels of all the other metabolites were comparable between the two weight groups in both diabetics and nondiabetic controls. Diabetic patients with systolic hypertension had higher ($P < 0.05$) low-density lipoprotein (LDL) and WC but nondiabetic hypertensives had lower ($P < 0.05$) FBG than their respective normotensives. In people with diabetes, dyslipidemia of total cholesterol (CHOL), LDL, and triglyceride were more prevalent in overweight/obese and systolic hypertensives. In controls, prevalence dyslipidemia of total and LDL CHOLs was higher in normal weight and hypertensives than their respective overweight/obese and normotensive counterparts. **Conclusion:** Nondiabetic respondents with normal weight may be at higher risk of cardiovascular disease through dyslipidemia than their overweight/obese counterpart. This metabolic paradox requires further investigations in the Ghanaian population.

Keywords: Dyslipidemia, hypertension, metabolic paradox, obesity, overweight, type 2 diabetes mellitus

Introduction

The global rise in the incidence of type 2 diabetes mellitus (T2DM) is believed to be driven in part by increased prevalence of obesity. The excess fat in the body serves as a major inflammatory source linking obesity to several chronic noncommunicable diseases such as T2DM. The International Diabetes Federation has predicted developing countries to experience a higher incidence of T2DM than developed economies.[1] This prediction hinges on expected increased spate of obesity due to urbanization, aging, sedentary lifestyles, and adoption of westernized diet. Excess fat accumulation in different populations can be assessed by various anthropometric indices of obesity such as waist circumference (WC), hip circumference, waist-to-hip ratio (WHR), and the body mass index (BMI). Although each of these techniques has been successfully employed to associate obesity with various health conditions, none of them is ideal in identifying all the different forms of pathologic fat accumulation observed in any given populace. The BMI, for instance, provides an indication of the global weight of an individual without regard to fat distribution resulting in overestimation of risk for highly muscular individuals like sportsmen and women. WHR and WC which are thought to be superior to BMI in predicting obesity-associated health conditions[2] fail to account for fat composition properly.[3] Indeed, various epidemiological studies[3-6] have failed to confirm the reported superiority of central obesity indices such as WHR and WC[2,7] over BMI in predicting obesity-related health conditions. Despite the lack of consensus on the best index for predicting obesity-associated

Address for correspondence: Dr. Samuel Acquah, Department of Medical Biochemistry, School of Medical Sciences, College of Health and Allied Sciences, University of Cape Coast, Cape Coast, Ghana.
E-mail: s.acquah@uccsms.edu.gh

Access this article online

Quick Response Code:

Website: www.ijmbs.org

DOI: 10.4103/ijmbs.ijmbs_4_17

How to cite this article: Acquah S, Eghan BA, Boampong JN. High prevalence of dyslipidemia irrespective of obesity in the cape coast metropolis of Ghana. Ibnosina J Med Biomed Sci 2017;9:103-10.

health conditions, BMI remains the preferred index due to its simplicity, reduced measurement errors, comparability across populations, and strong correlation with indices of central obesity.[2,8] Irrespective of the index used, increased fat accumulation is associated with several adverse health outcomes. Development of most chronic noncommunicable diseases known to be mediated by fat accumulation begins as a gradual process through certain identifiable and measurable indices that can be used to assess the risk or extent of development of that chronic disease. In the case of T2DM, several indices such as insulin resistance, impaired beta cell function, and abnormal levels of circulating lipids which are known to be mediated by obesity have been implicated in different populations. However, the extent to which these indices are mediated by obesity in the development of T2DM and its associated complications differ from one population to the other. Little is known about the extent to which the above-named indices are linked to obesity and the development of T2DM in Ghana. Therefore, this study was designed to investigate the link between overweight/obesity and indices of insulin resistance, beta cell function, circulating lipid levels, and blood pressure in type 2 diabetics and nondiabetic controls in the Cape Coast Metropolis of the Central Region of Ghana.

Patients and Methods

Setting and design

Selection of participants for the study, blood pressure, and anthropometric measurements as well as the laboratory determination of fasting blood glucose (FBG), glycosylated hemoglobin (HbA1c), and lipid profile were carried out at the Cape Coast Teaching Hospital (CCTH). However, measurement of serum insulin level was done at the laboratory of the School of Medical Sciences, University of Cape Coast. CCTH is the only teaching hospital in the Central Region and serves as the tertiary hospital to which the various health facilities in the region refer cases. CCTH is located in Cape Coast, the capital of Central Region and has a well-structured diabetic clinic with patients from various parts of the region. According to data from the 2010 Population and Housing Census released by the Ghana Statistical Service, the region had a population of 2,201,863 with females constituting 52.3%.[9] On Cape Coast, the estimated population of 169,894 was made up of 51.3% females. With a regional intercensal growth rate of 3.1%, the estimated population of Cape Coast in 2015 was 197,912. Cape Coast has a relatively high concentration of educational institutions in the region and in fact, hosts some top-notch Senior High Schools in the country. The principal occupations of inhabitants of Cape Coast include farming and trading in the informal sector with a relatively small proportion of the workforce in the formal sector. People with diabetes were recruited from diabetes patients attending the CCTH diabetic clinic. People with diabetes who enrolled in the study were nonsmokers and nonusers of insulin therapy or lipid-lowering drugs. Nondiabetic control respondents who were age matched with their diabetic counterpart were selected from the general inhabitants of the Cape Coast Metropolis and were subjected to the same inclusion and exclusion criteria as patients. The study was approved by the Committee on Human Research, Publications and Ethics of the Kwame Nkrumah University of Science and Technology, School of Medical Sciences and Komfo Anokye Teaching Hospital, Kumasi. An informed written consent was obtained from all study participants.

Methods

Details of participants' selection, inclusion and exclusion criteria, blood sample collection, and laboratory measurements of biochemical indices have been published elsewhere.[10,11] Anthropometric indices were measured by WHO expert panel report of 2008.[12] Levels of FBG, HbA1c, and lipid profile were determined by standard methods.[10,11,13] Enzyme-linked immunosorbent assay was used to determine serum insulin level. Homeostatic models assessment of insulin resistance (HOMAIR) and homeostatic model assessment of beta cell secretion (HOMAB) developed by Matthews *et al.* were used for estimation of insulin resistance and beta cell function, respectively.[14] All measurements were done after a 12-h overnight fast. In all, 230 respondents consisting of 115 people with diabetes and an equal number of control respondents were enrolled in the study. However, for the current report, only 223 (96.96%) respondents consisting of 113 diabetics and 110 nondiabetic controls had complete data for FBG, HbA1c, systolic blood pressure (SBP), diastolic blood pressure (DBP), serum insulin level, HOMAIR, HOMAB, and lipid profile, for inclusion. In each study group, females constituted more than 70% of respondents. Hypertension was diagnosed by the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure as SBP ≥140 mmHg or DBP ≥90 mmHg. Dyslipidemia was defined according to Warnick *et al*.[15] as follows: total serum cholesterol (CHOL) >5.2 mmol/L; low-density lipoprotein (LDL) >2.6 mmol/L; HDL <1.03 mmol/L; and triglycerides (TRGs) >1.7 mmol/L. Overweight/obesity was defined as BMI >25 kg/m^2. Already diagnosed hypertensives enrolled in the study were on amlodipine or nifedipine with the people with diabetes using metformin as the hypoglycemic agent.

Data analysis and statistics

Data analysis was done by the SPSS software version 17 (SPSS Inc., Chicago, USA). Data are presented as mean ± standard deviation. In each study group, respondents were divided into two: normal and overweight/obese according to BMI classification to obtain a total of four categories. Mean levels of the various measured parameters were compared across categories by one-way ANOVA with Tukey's *post hoc* honest significant difference (HSD) test. Subsequently, respondents were further categorized into normotensive and hypertensive groups based on systolic or DBP values. For these categorizations, mean levels of the various indices in each study group were compared by two-tailed independent sample *t*-test. Prevalence dyslipidemia was compared between groups by Chi-square test. Bivariate correlation, simple and stepwise

linear regression analyses were carried out. In all analyses, $P < 0.05$ was considered statistically significant.

Each study group was subdivided based on BMI, SBP, or DBP for comparison of indices. With respect to BMI, respondents were initially categorized into three weight groups: normal weight (BMI ≤25), overweight (BMI = 25–30), and obese (BMI >30) weight groups, for comparison of measured indices. Subsequently, respondents were recategorized into two weight groups: normal-weight and overweight/obese groups, because the overweight and obese respondents did not differ significantly ($P > 0.05$) in mean levels of any of the measured indices (data not shown) in both the person with diabetes and the nondiabetic control respondents.

Results

The mean levels of the measured parameters according to BMI are shown in Table 1. Apart from the mean levels of CHOL, TRG, serum insulin, and DBP that were comparable ($P > 0.05$) across groups, all the other measured parameters differed significantly ($P < 0.05$) between normal-weight and overweight/obese respondents. Diabetic patients with normal weight exhibited the highest mean levels of FBG, HbA1c, and HOMAIR but

Table 1: Blood pressure and biochemical indices of diabetic and nondiabetic respondents according to body mass index classification

Parameter	Diabetic patients		Nondiabetic controls		*F*	*P*
	BMI ≤25 (*n*=53)	BMI >25 (*n*=60)	BMI ≤25 (*n*=55)	BMI >25 (*n*=55)		
Age (years)	56.9±9.3	56.9±8.3	54.2±10.3	51.6±10.4	3.064	0.030*
FBG (mmol/L)	8.5±4.5	6.8±2.6	4.5±0.8	4.7±0.7	23.8	<0.001*
HbA1c (mmol/L)	5.0±1.6	4.5±1.6	4.3±1.8	3.97±0.41	4.144	0.007*
LDL (mmol/L)	3.3±0.9	3.2±1.1	3.8±1.1	3.2±0.9	2.672	0.049*
HDL (mmol/L)	1.1±0.4	1.1±0.5	1.3±0.1	1.3±0.1	4.893	0.003*
Total cholesterol (mmol/L)	5.4±1.2	5.3±1.3	5.6±1.1	5.0±0.9	1.608	0.189
TRG (mmol/L)	1.2±0.7	1.3±0.6	1.1±0.4	1.2±0.6	1.664	0.177
Insulin	5.1±1.7	5.8±1.6	4.6±1.4	5.9±1.9	2.188	0.092
HOMAIR	1.73±2.38	1.58±1.81	1.06±1.76	1.10±1.85	4.999	0.003*
HOMAB	36.19±2.03	46.32±2.73	121.62±2.26	114.03±2.14	16.127	<0.001*
Systolic (mmHg)	130±20	143±22	133±26	130±24	2.302	0.002*
Diastolic (mmHg)	81±14	82±10	83±16	80±16	1.455	0.127
Hip (cm)	103±10	105±10	104±12	105±11	1.342	0.096
WC (cm)	95±26	97±24	96±29	97±43	1.659	0.18
WHR	0.93±0.05	0.92±0.06	0.92±0.07	0.92.41±0.09	1.596	0.21
Tukey HSD	**Diabetic individuals**		***P***	**Nondiabetic controls**		***P***
	BMI <25	BMI >25		BMI <25	BMI >25	
Age (years)	56.9±9.3	56.9±8.3	0.995	54.2±10.3	51.6±10.4	0.2
FBG (mmol/L)	8.5±4.5	6.8±2.6	0.020*	4.5±0.8	4.7±0.7	0.993
HbA1c	4.5±1.6	5.0±1.6	0.707	4.3±1.8	4.0±0.4	0.504
LDL	3.3±0.9	3.2±1.1	0.983	3.8±1.1	3.2±0.9	0.092
HDL	1.14±0.41	1.07±0.47	0.708	1.27±0.12	1.3±0.1	0.999
HOMAIR	1.73±2.38	1.58±1.81	0.956	1.06±1.76	1.10±1.85	0.992
HOMAB	36.19±2.03	46.32±2.73	0.764	121.62±2.26	114.03±2.14	0.988
Systolic	130±20	143±22	0.038*	133±26	130±24	0.602
Tukey HSD	**BMI ≤25**		***P***	**BMI >25**		***P***
	DM	ND		DM	ND	
Age (years)	56.9±9.29	54.23±10.30	0.491	56.89±8.29	51.57±10.37	0.016*
FBG (mmol/L)	8.46±4.46	4.50±0.77	<0.001*	6.79±2.64	4.66±0.73	<0.001*
HbA1c	4.54±1.64	4.34±1.84	0.974	4.95±1.57	3.97±0.41	0.004*
LDL	3.33±0.88	3.78±1.12	0.375	3.23±1.13	3.23±0.92	0.999
HDL	1.14±0.41	1.27±0.12	0.884	1.07±0.47	1.27±0.11	0.002
HOMAIR	1.73±2.38	1.06±1.76	0.048*	1.58±1.81	1.10±1.85	0.030*
HOMAB	36.19±2.03	121.62±2.26	<0.001*	46.32±2.73	114.03±2.14	<0.001*
Systolic	130±20	133±26	0.578	143±22	130±24	0.025*

Figures represent mean±SD. *Significant *P* value, insulin is measured in μIU/mL. BMI: Body mass index, WHR: Waist-to-hip ratio, WC: Waist circumference, LDL: Low-density lipoprotein cholesterol, HDL: High-density lipoprotein cholesterol, TRG: Triglyceride, FBG: Fasting blood glucose, HbA1c: Glycosylated hemoglobin, HOMAIR: Homeostatic model assessment of insulin resistance, HOMAB: Homeostatic model assessment of beta cell secretion, SD: Standard deviation, DM: Diabetes mellitus, HSD: Honest significant difference, ND: Nondiabetic

the lowest level of HOMAB [$P < 0.05$; Table 1]. With respect to SBP, people with diabetes who were overweight/obese exhibited the highest mean level. However, Tukey *post hoc* HSD comparison of means revealed that respondents in the diabetic group with normal weight had significantly [$P < 0.05$; Table 1] higher mean level of FBG but lower SBP than their overweight/obese counterpart. Mean levels of all the other parameters did not differ [$P > 0.05$; Table 1] between the groups. In the nondiabetic study group, no significant [$P > 0.05$; Table 1] difference was observed between normal-weight and overweight/obese individuals in any of the measured parameters. In a bivariate Pearson correlational test in controls with normal weight, HOMAIR correlated negatively with WC ($R = -0.441$; $P = 0.007$) but positively with SBP (R = +0.375; $P = 0.024$). HbA1c correlated positively with TRG (R = +0.293; $P = 0.041$). A similar analysis in the overweight/obese nondiabetic controls revealed HOMAIR correlating negatively with WC ($R = -0.516$; $P < 0.001$) and SBP ($R = -0.401$; $P = 0.005$). LDL associated positively with WC (R = +0.295; $P = 0.04$) while HDL exhibited a negative correlation with hip circumference (R = −0.353; $P = 0.016$). WC associated positively with SBP (R = +0.488; $P < 0.001$). In a subsequent multiple stepwise regression analysis with HOMAIR as the dependent variable, none of the variables emerged as an independent predictor of HOMAIR in either of the weight-based study groups.

In the individuals with diabetes with normal weight, FBG correlated negatively with WC ($R = -0.474$; $P = 0.041$) as HbA1c positively correlated with HOMAIR (R = +0.602; $P = 0.014$) but negatively with LDL (R = −0.6; $P = 0.007$) and DBP (R = −0.456; $P = 0.049$). In their overweight/obese counterpart, positive correlation was observed between FBG and insulin ($R = +0.394$; $P = 0.07$) and HOMAB (R = +0.289; $P = 0.036$). Furthermore, insulin correlated positively with HOMAB (R = +0.428; $P = 0.003$). A stepwise multiple regression analysis with HOMAIR as the dependent variable showed that in the diabetics with normal-weight category, HbA1c was the only significant ($R^2 = 0.763$, adjusted $R^2 = 0.736$; $P < 0.001$) independent predictor of HOMAIR with the model explaining 73.6% of the observed variation. No significant predictor of HOMAIR was found in the diabetics who were overweight/obese.

In Figure 1, the prevalence dyslipidemia observed in respondents of the current study according to BMI categorization is presented. Interestingly, dyslipidemia of TRG was lower, but those of LDL and CHOL were higher ($P < 0.001$) in nondiabetic controls with normal weight than their overweight/obese counterpart. In the diabetic group, overweight/obese diabetics exhibited higher prevalence of the various forms of dyslipidemia than their counterpart with normal weight.

In systolic normotensive nondiabetic controls, FBG was higher [$P = 0.004$; Table 2] than their hypertensive counterpart. However, in the diabetics, levels of LDL and WC were higher in systolic hypertensives [$P < 0.05$; Table 2]. In a similar analysis according to DBP, nondiabetic hypertensives exhibited

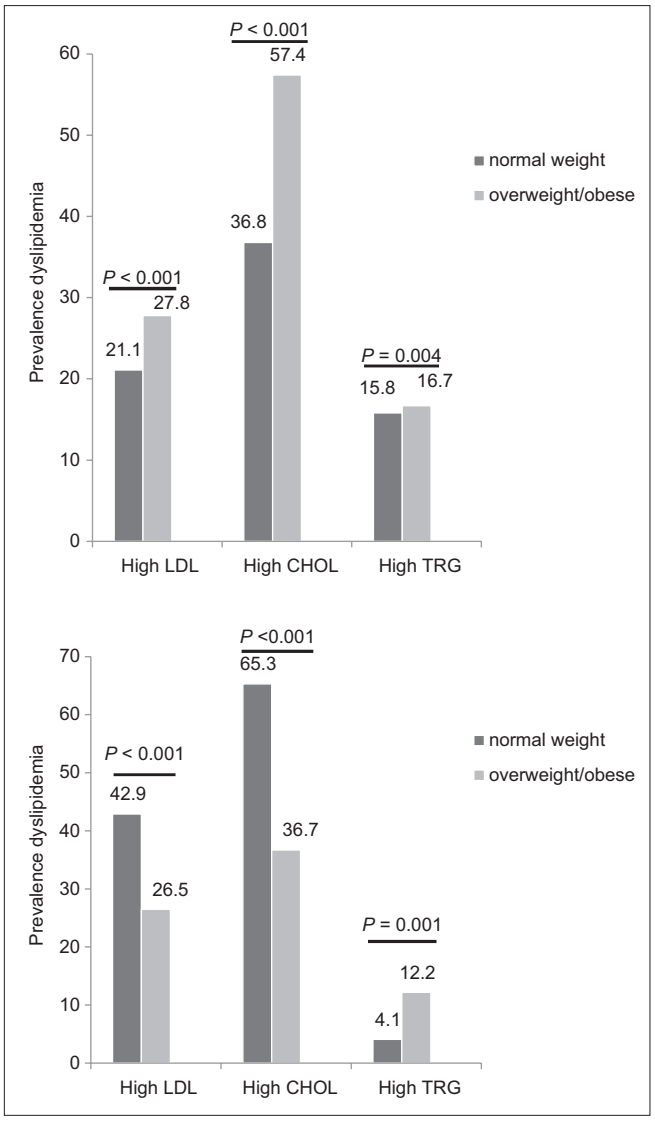

Figure 1: Prevalence dyslipidemia in diabetic respondents above and in nondiabetic respondents below.

lower [$P < 0.05$; Table 3] mean BMI and hip circumference than their diastolic normotensive counterpart.

Pearson correlational test revealed that in the systolic hypertensive diabetics, HOMAIR associated positively with FBG ($R = +0.487$; $P = 0.03$) and HOMAB ($R = +0.468$; $P = 0.032$). In addition, WC correlated negatively with LDL ($R = -0.567$; $P = 0.003$) and CHOL ($R = -0.501$; $P = 0.009$) but age correlated positively with TRG ($R = +0.438$; $P = 0.033$). Above all, HbA1c exhibited a negative correlation with HDL ($R = -0.438$; $P = 0.025$). In their normotensive counterpart, HOMAIR rather correlated positively with DBP ($R = +0.348$; $P = 0.024$) and duration of the diabetic condition ($R = +0.321$; $P = 0.038$). HbA1c correlated positively with TRG and FBG ($R = +0.31$; $P = 0.027$) but negatively with HDL ($R = -0.32$; $P = 0.022$). Insulin associated positively with LDL ($R = +0.34$; $P = 0.026$) and HOMAB ($R = +0.52$; $P = 0.001$). Above all, CHOL exhibited a negative correlation with DBP but

Table 2: Mean values of the various parameters based on systolic blood pressure classification

Parameter	Nondiabetic		*P*	Diabetic		*P*
	≤140 (*n*=72)	>140 (*n*=38)		≤140 (*n*=60)	>140 (*n*=53)	
Age (years)	52.15±9.86	55.56±10.48	0.12	55.34±10.14	58.69±7.38	0.104
Duration (years)	-	-	-	7.05±5.25	6.35±4.52	0.528
FBG (mmol/L)	4.75±0.78	4.28±0.63	0.004*	7.30±3.23	7.08±2.97	0.734
HbA1c (mmol/L)	4.45±2.23	4.03±0.53	0.136	5.49±2.32	4.85±1.39	0.139
LDL (mmol/L)	3.45±1.09	3.66±0.96	0.35	2.96±0.79	3.55±1.05	0.003*
HDL (mmol/L)	1.26±0.11	1.26±0.13	0.885	1.25±0.16	1.22±0.11	0.308
Total cholesterol (mmol/L)	5.25±1.09	5.42±0.86	0.44	5.39±1.47	5.50±1.06	0.692
TRG (mmol/L)	1.11±0.53	1.10±0.33	0.854	1.27±0.65	1.23±0.58	0.79
Insulin	5.68±1.86	4.59±1.25	0.077	5.54±1.61	5.49±1.59	0.93
HOMAIR	1.08±1.84	1.08±1.76	0.982	1.54±2.08	1.64±1.80	0.709
HOMAB	114.95±2.30	120.87±1.93	0.785	43.36±2.20	44.47±2.96	0.909
BMI (kg/m2)	26.5±5.8	25.2±40.1	0.241	28.9±6.4	30.4±6.7	0.328
Hip (cm)	104.79±10.51	101.35±10.63	0.594	100.66±12.30	96.51±13.42	0.71
WC (cm)	100.60±9.22	98.36±20.48	0.144	93.42±49.43	85.90±39.31	0.006*
WHR	0.96±0.07	0.97±0.08	0.64	0.92.81±0.06	0.89±0.09	0.389

Figures represent mean±SD. *Significant *P* value, insulin is measured in µIU/mL. BMI: Body mass index, WHR: Waist-to-hip ratio, WC: Waist circumference, LDL: Low-density lipoprotein cholesterol, HDL: High-density lipoprotein cholesterol, TRG: Triglyceride, FBG: Fasting blood glucose, HbA1c: Glycosylated hemoglobin, HOMAIR: Homeostatic model assessment of insulin resistance, HOMAB: Homeostatic model assessment of beta cell secretion, SD: Standard deviation

Table 3: Mean values of the various parameters based on diastolic blood pressure classification

Parameter	Nondiabetic		*P*	Diabetic		*P*
	≤90 (*n*=76)	>90 (*n*=34)		≤90 (*n*=70)	>90 (*n*=43)	
Age (years)	53.03±10.63	53.89±8.98	0.706	57.98±9.07	55±8.65	0.169
Duration (years)	-	-	-	6.75±4.63	6.58±5.42	0.884
FBG (mmol/L)	4.67±0.80	4.41±0.63	0.119	7.32±3.09	6.92±3.26	0.591
HbA1c (mmol/L)	4.06±0.53	4.43±2.39	0.21	5.38±2.16	4.84±1.59	0.255
LDL (mmol/L)	3.54±1.08	3.46±0.98	0.751	3.11±0.88	3.42±1.09	0.157
HDL (mmol/L)	1.26±0.11	1.27±0.14	0.841	1.24±0.15	1.23±0.12	0.804
Total cholesterol (mmol/L)	5.30±1.07	5.32±0.95	0.953	5.45±1.40	5.37±1.09	0.796
TRG (mmol/L)	1.09±0.50	1.15±0.39	0.575	1.25±0.65	1.25±0.56	0.976
Insulin	5.39±1.72	5.01±1.64	0.57	5.77±1.68	4.95±1.68	0.192
HOMAIR	1.08±1.84	1.10±1.77	0.928	1.56±1.96	1.66±1.92	0.714
HOMAB	116.36±2.28	118.28±1.93	0.932	46.19±2.26	39±3.26	0.483
BMI (kg/m2)	26.77±5.76	24.38±3.64	0.045*	29.43±6.21	30.17±7.29	0.328
Hip (cm)	105.1±10.5	101.2±10.4	0.044*	105.6±13.6	105.7±12.4	0.981
WC (cm)	100.9±28.8	94.1±20.8	0.42	96.1±49.0	94.1±43.1	0.697
WHR	0.96±0.08	0.93±0.06	0.116	0.91±0.07	0.89±0.09	0.308

Figures represent mean±SD. *Significant *P* value, insulin is measured in µIU/mL. BMI: Body mass index, WHR: Waist-to-hip ratio, WC: Waist circumference, LDL: Low-density lipoprotein cholesterol, HDL: High-density lipoprotein cholesterol, TRG: Triglyceride, FBG: Fasting blood glucose, HbA1c: Glycosylated hemoglobin, HOMAIR: Homeostatic model assessment of insulin resistance, HOMAB: Homeostatic model assessment of beta cell secretion, SD: Standard deviation

HOMAB showed a positive association with hip circumference ($R = +0.386$; $P = 0.022$) and WHR ($R = +0.309$; $P = 0.046$).

In the controls, HOMAB correlated positively with HOMAIR ($R = +0.552$; $P = 0.005$) but negatively with WC ($R = -0.503$; $P = 0.012$) in the systolic hypertensives. Furthermore, HOMAIR correlated negatively with WC ($R = -0.827$; $P < 0.001$). Meanwhile, age positively correlated with WHR ($R = +0.438$; $P = 0.047$) and LDL associated negatively with BMI ($R = -0.445$; $P = 0.018$) and HbA1c ($R = -0.401$; $P = 0.035$). BMI correlated negatively with CHOL ($R = -0.422$; $P = 0.025$). Finally, HDL correlated negatively with insulin ($R = -0.422$; $P = 0.036$). Similar analysis in the systolic normotensive nondiabetic controls showed a negative correlation between WC and HOMAIR ($R = -0.426$; $P = 0.001$) and between LDL and hip circumference ($R = -0.255$; $P = 0.042$).

Further categorization based on DBP revealed that in the nondiabetic normotensive respondents, HOMAIR negatively correlated with SBP ($R = -0.322$; $P = 0.006$) and WC ($R = -0.425$; $P < 0.001$). Furthermore, SBP associated positively with WC ($R = +0.339$; $P = 0.003$), CHOL ($R = +0.238$; $P = 0.029$), and LDL ($R = +0.278$; $P = 0.01$). CHOL negatively correlated with BMI ($R = -0.267$; $P = 0.015$). In their hypertensive counterpart, HOMAIR negatively correlated with WC ($R = -0.875$; $P < 0.001$) but positively with SBP ($R = +0.71$; $P = 0.014$), HDL ($R = +0.64$; $P = 0.034$), and HOMAB ($R = +0.755$; $P = 0.007$). WC correlated negatively with HDL ($R = -0.578$; $P = 0.039$) and HOMAB ($R = -0.799$; $P = 0.003$) but HOMAB positively correlated with SBP ($R = +0.754$; $P = 0.007$). FBG on the other hand exhibited a negative correlation with BMI ($R = -0.568$; $P = 0.043$) and age ($R = -0.579$; $P = 0.038$).

In the diabetic group, diabetic diastolic normotensives showed a direct correlation between HOMAIR and duration ($R = +0.292$; $P = 0.034$). Furthermore, SBP correlated positively with LDL ($R = +0.336$; $P = 0.006$) and hip circumference ($R = +0.345$; $P = 0.011$). HDL positively correlated with BMI ($R = +0.261$; $P = 0.039$) and insulin ($R = +0.281$; $P = 0.034$) as HOMAB associated with hip circumference ($R = +0.378$; $P = 0.007$) and insulin ($R = +0.479$; $P < 0.001$). With respect to the diabetic diastolic hypertensives, HOMAIR positively correlated with FBG ($R = +0.744$; $P = 0.021$) and HOMAB (+0.721; $P = 0.044$). A negative correlation was observed between WC and SBP ($R = -0.721$; $P = 0.044$). Age correlated positively with HbA1c ($R = +0.729$; $P = 0.026$) and WHR ($R = +0.748$; $P = 0.033$). Above all, insulin correlated positively with BMI ($R = +0.759$; $P = 0.048$) as did HbA1c and TRG ($R = +0.669$; $P = 0.049$). However, multiple linear regression analyses failed to demonstrate any of the measured variables as a significant ($P > 0.05$) predictor of HOMAIR as far as DBP or SBP classification of respondents was concerned. The prevalence of various forms of dyslipidemia differed significantly between normotensive and hypertensive respondents [$P < 0.05$; Figures 2 and 3] of the current study.

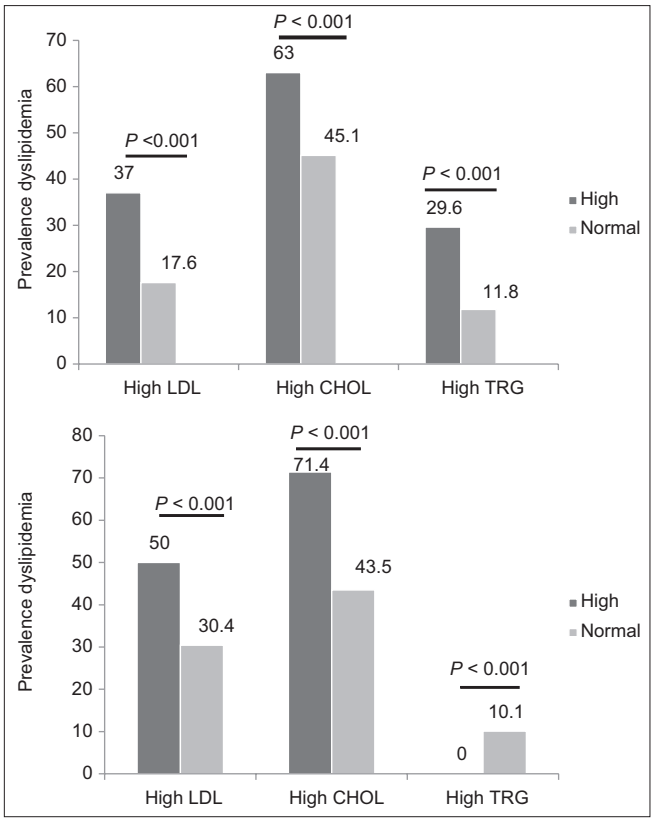

Figure 2: Prevalence dyslipidemia in diabetic respondents above and in nondiabetic respondents below according to systolic blood pressure.

Discussion

The observed higher FBG in diabetics with normal weight than their overweight/obese counterpart contradicts previous studies[16,17] that demonstrated raised FBG levels with increased BMI in overweight/obese respondents and points to improved glucose homeostasis in our overweight/obese respondents in support of Arnlöv *et al*.[18] The contradiction could be ascribed to variation in study design, race, ethnicity, and sample size between the current study and the earlier ones.[16,17] Thus, our overweight/obese diabetic respondents appear to respond better to the given treatment regimen than their normal-weight counterpart in accordance with the concept of obesity paradox.[19] The obesity paradox which postulates that some individuals who are overweight/obese appear to have better prognosis than their normal-weight counterpart with similar cardiovascular disease risk or health condition has been reported in various obesity-associated cardiovascular diseases and risk factors.[19,20]

Indeed, the positive association exhibited by insulin with FBG and HOMAB in the overweight/obese but not the normal-weight diabetic respondents gives further credence to the improved metabolic status of our overweight/obese diabetic respondents.

In the controls, the metabolic profile of overweight/obese and normal-weight respondents was comparable with respect to the level of a number of the measured parameters. However, the higher prevalence of dyslipidemia of total and LDL CHOLs in normal weight than overweight/obese respondents points to a likely higher risk of dyslipidemia-related cardiovascular disease in normal-weight control respondents of the current study and portrays the overweight/obese respondents as metabolically healthier. This observation which corroborates Voulgari *et al*.[21] has been ascribed to differences in body fat content whereby normal-weight individuals with high body fat exhibit higher prevalence of dyslipidemia, metabolic syndrome, and other cardiovascular risk factors.[22] The finding is further buttressed by the negative correlation observed between HOMAIR and WC in normal-weight control respondents in the current study, suggesting that, in this group, reduction of WC would rather result in increased HOMAIR instead of a decrease as widely acknowledged.[23,24] This observation, which requires further and larger studies for confirmation, particularly, in the Ghanaian setting, is quite significant because it may shift the paradigm of current educational campaigns that target overweight/obese individuals as the only group at risk of dyslipidemia-related cardiovascular disease.

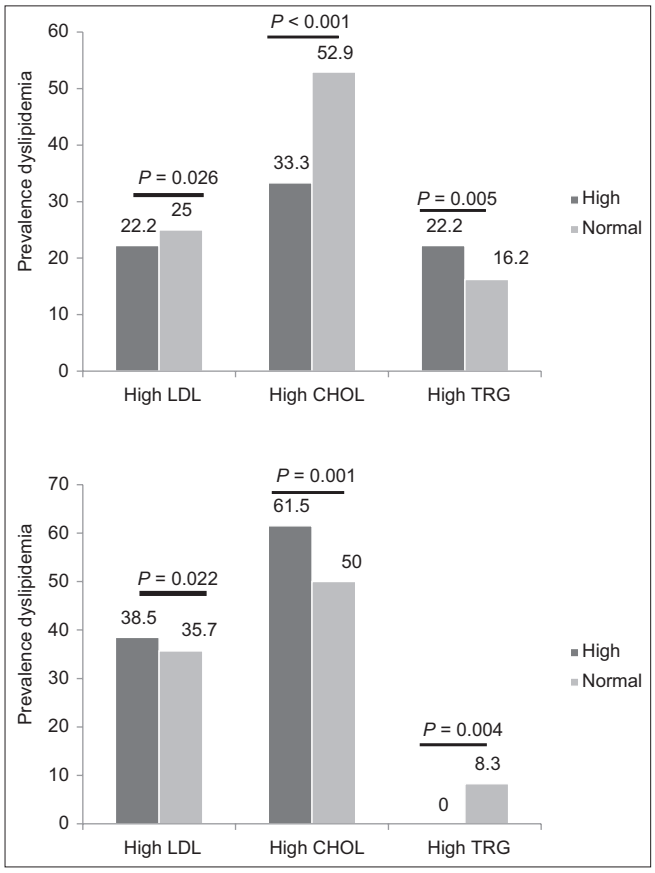

Figure 3: Prevalence dyslipidemia in diabetic respondents above and in nondiabetic respondents below according to diastolic blood pressure.

The observed trend of higher SBP in overweight/obese diabetics compared with their normal-weight counterpart appears to support earlier reports[18,25] and confirms the well-known role of excess adiposity in blood pressure dysregulation. Dyslipidemia predates and drives the development of hypertension through endothelial damage.[26,27] In the current study, mean levels of the various parameters were comparable between normotensives and hypertensives with the exception of higher LDL and WC in hypertensive diabetics but lower FBG, BMI, and hip circumference in nondiabetic hypertensives compared to their respective normotensive counterparts. Whereas the observation for the diabetic hypertensives appears to support the already acknowledged role of visceral adiposity and LDL in the development of hypertension,[26,27] that of the nondiabetic respondents seems to suggest that global fat accumulation *per se* or circulating glucose has little or no effect on the development of hypertension. Rather, it is the abdominal fat accumulation that seems to have the potential to drive increased blood pressure levels. This finding which appears to validate earlier reports[2,7] that questioned the reliability of BMI over WC or WHR in detecting obesity-related health conditions requires further investigation in the Ghanaian populace, particularly, for confirmation, so as to devise appropriate remedial measures to avert its negative health consequences. Meanwhile, the reciprocal association exhibited by SBP and HOMAIR in nondiabetic diastolic normotensives compared with their hypertensive counterpart in the present study suggests that other cellular conditions greatly influence how HOMAIR associates with SBP. As observed, in diastolic hypertensives, HOMAIR drives SBP and vice versa. This observation supports a number of earlier reports that showed improved insulin sensitivity, glucose uptake, and protection of β-cells against hyperglycemia-induced apoptosis by nifedipine,[28,29] a major antihypertensive drug used by hypertensive respondents in the current study. To this end, the comparable level of HOMAIR and a number of measured indices between hypertensives and normotensives or between respondents with normal-weight and their overweight/obese counterpart observed in the current study could be partly ascribed to treatment effect.

However, the negative correlation between HOMAIR and WC observed in control respondents irrespective of hypertensive status contradicts several reports that associated insulin resistance with visceral adiposity.[30,31] Visceral fat is more active metabolically[32] and hydrolyzes readily to nonesterified fatty acids to release inflammatory cytokines.[33,34] These features are thought to link visceral adiposity, measured as WC, to insulin resistance such that increased WC should result in an increased HOMAIR instead of a decrease that our data seem to portray. This paradox requires further and larger study to better understand it in the Ghanaian context to pave way for appropriate remedial measures to be developed for adoption in the light of varied degrees of dyslipidemia in both hypertensive and normotensive respondents of the current study.

Conclusion

Dyslipidemia of total and LDL CHOLs was more prevalent in normal-weight and normotensive nondiabetic controls than their respective overweight/obese and hypertensive counterparts. Further studies are required to better understand the paradox in the Ghanaian context. Public education on dyslipidemia and hypertension should target both overweight/obese and normal-weight individuals.

Acknowledgment

The authors are grateful to the study participants and staff and management of CCTH for use of their facility. Also, they are deeply appreciative of the staff and management of University of Cape Coast, School of Medical Sciences, notably, those responsible for the multipurpose laboratory for their countless support.

Disclosures

The authors are responsible for the conduct of the study, data collection, and analysis. They drafted and revised the manuscript and approved its final version.

Financial support and sponsorship

The study was funded by a grant from University of Cape Coast.

Conflicts of interest

There are no conflicts of interest.

Compliance with ethical principles

Approval for the study was granted the university's IRB and informed consent was obtained from all participants prior to the study.

References

1. International Diabetes Federation. IDF Diabetes Atlas. 7th ed. Brussels, Belgium: International Diabetes Federation; 2015.
2. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. Am J Clin Nutr 2004;79:379-84.
3. Vazquez G, Duval S, Jacobs DR Jr., Silventoinen K. Comparison of body mass index, waist circumference, and waist/hip ratio in predicting incident diabetes: A meta-analysis. Epidemiol Rev 2007;29:115-28.
4. Hartemink N, Boshuizen HC, Nagelkerke NJ, Jacobs MA, van Houwelingen HC. Combining risk estimates from observational studies with different exposure cutpoints: A meta-analysis on body mass index and diabetes type 2. Am J Epidemiol 2006;163:1042-52.
5. Decoda Study Group, Nyamdorj R, Qiao Q, Lam TH, Tuomilehto J, Ho SY, *et al.* BMI compared with central obesity indicators in relation to diabetes and hypertension in Asians. Obesity (Silver Spring) 2008;16:1622-35.
6. Zhou Z, Hu D, Chen J. Association between obesity indices and blood pressure or hypertension: Which index is the best? Public Health Nutr 2009;12:1061-71.
7. Katzmarzyk PT, Heymsfield SB, Bouchard C. Clinical utility of visceral adipose tissue for the identification of cardiometabolic risk in white and African American adults. Am J Clin Nutr 2013;97:480-6.
8. Panoulas VF, Ahmad N, Fazal AA, Kassamali RH, Nightingale P, Kitas GD, *et al.* The inter-operator variability in measuring waist circumference and its potential impact on the diagnosis of the metabolic syndrome. Postgrad Med J 2008;84:344-7.
9. Ghana Statistical Service. 2010 Population and Housing Census, Summary Results of Final Report. Accra: Sakoa Press Limited; 2012. p. 15.
10. Acquah S, Boampong JN, Eghan Jnr BA, Eriksson M. Evidence of insulin resistance in adult uncomplicated malaria: Result of a two-year prospective study. Malar Res Treat 2014;2014:136148.
11. Acquah S, Eghan Jnr BA, Bawa S, Boampong JN. Differential response in lipid levels of type 2 diabetics and non-diabetic controls to falciparum malaria. Asian J Med Sci 2015;6:71-6.
12. World Health Organization. Waist Circumference and Waist-Hip Ratio: Report of a WHO Expert Consultation 2008. Geneva, Switzerland: World Health Organization; 2008.
13. Acquah S, Boampong JN, Adusu J, Achampong EK, Setorglo J, Obiri-Yeboah D. Lipid and lipoprotein levels in type 2 diabetes patients attending the Central Regional Hospital in the Cape Coast metropolis of Ghana. Int J Health Res 2011;4:75-82.
14. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: Insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-9.
15. Warnick GR, Myers GL, Cooper GR, Rifai N. Impact of the third cholesterol report from the adult treatment panel of the national cholesterol education program on the clinical laboratory. Clin Chem 2002;48:11-7.
16. Ning F, Pang ZC, Dong YH, Gao WG, Nan HR, Wang SJ, *et al.* Risk factors associated with the dramatic increase in the prevalence of diabetes in the adult Chinese population in Qingdao, China. Diabet Med 2009;26:855-63.
17. Shai I, Jiang R, Manson JE, Stampfer MJ, Willett WC, Colditz GA, *et al.* Ethnicity, obesity, and risk of type 2 diabetes in women: A 20-year follow-up study. Diabetes Care 2006;29:1585-90.
18. Arnlöv J, Ingelsson E, Sundström J, Lind L. Impact of body mass index and the metabolic syndrome on the risk of cardiovascular disease and death in middle-aged men. Circulation 2010;121:230-6.
19. Lavie CJ, Alpert MA, Arena R, Mehra MR, Milani RV, Ventura HO. Impact of obesity and the obesity paradox on prevalence and prognosis in heart failure. JACC Heart Fail 2013;1:93-102.
20. De Schutter A, Lavie CJ, Milani RV. The impact of obesity on risk factors and prevalence and prognosis of coronary heart disease-the obesity paradox. Prog Cardiovasc Dis 2014;56:401-8.
21. Voulgari C, Tentolouris N, Dilaveris P, Tousoulis D, Katsilambros N, Stefanadis C. Increased heart failure risk in normal-weight people with metabolic syndrome compared with metabolically healthy obese individuals. J Am Coll Cardiol 2011;58:1343-50.
22. Romero-Corral A, Somers VK, Sierra-Johnson J, Korenfeld Y, Boarin S, Korinek J, *et al.* Normal weight obesity: A risk factor for cardiometabolic dysregulation and cardiovascular mortality. Eur Heart J 2010;31:737-46.
23. Park K, Lee DH, Erickson DJ, Himes JH, Shikany JM, Jacobs DR Jr. Association of long-term change in waist circumference with insulin resistance. Obesity (Silver Spring) 2010;18:370-6.
24. Park YM, Kwon HS, Lim SY, Lee JH, Yoon KH, Son HY, *et al.* Optimal waist circumference cutoff value reflecting insulin resistance as a diagnostic criterion of metabolic syndrome in a nondiabetic Korean population aged 40 years and over: The Chungju Metabolic Disease Cohort (CMC) study. Yonsei Med J 2010;51:511-8.
25. Russo C, Jin Z, Homma S, Rundek T, Elkind MS, Sacco RL, *et al.* Effect of obesity and overweight on left ventricular diastolic function: A community-based study in an elderly cohort. J Am Coll Cardiol 2011;57:1368-74.
26. Osuji CU, Omejua EG, Onwubuya EI, Ahaneku GI. Serum lipid profile of newly diagnosed hypertensive patients in nnewi, South-East Nigeria. Int J Hypertens 2012;2012:710486.
27. Laaksonen DE, Niskanen L, Nyyssönen K, Lakka TA, Laukkanen JA, Salonen JT. Dyslipidaemia as a predictor of hypertension in middle-aged men. Eur Heart J 2008;29:2561-8.
28. Hassan KA, Mudawi MM, Sulaiman MI. Pharmacodynamics drug interactions of metformin with aspirin and nifedipine. Asian J Pharm Res Health Care 2016;8:4-10.
29. Wang Y, Gao L, Li Y, Chen H, Sun Z. Nifedipine protects INS-1 ß-cell from high glucose-induced ER stress and apoptosis. Int J Mol Sci 2011;12:7569-80.
30. Hanley AJ, Wagenknecht LE, Norris JM, Bryer-Ash M, Chen YI, Anderson AM, *et al.* Insulin resistance, beta cell dysfunction and visceral adiposity as predictors of incident diabetes: The Insulin Resistance Atherosclerosis Study (IRAS) Family study. Diabetologia 2009;52:2079-86.
31. Ledoux S, Coupaye M, Essig M, Msika S, Roy C, Queguiner I, *et al.* Traditional anthropometric parameters still predict metabolic disorders in women with severe obesity. Obesity (Silver Spring) 2010;18:1026-32.
32. Yang YK, Chen M, Clements RH, Abrams GA, Aprahamian CJ, Harmon CM. Human mesenteric adipose tissue plays unique role versus subcutaneous and omental fat in obesity related diabetes. Cell Physiol Biochem 2008;22:531-8.
33. Gastaldelli A, Cusi K, Pettiti M, Hardies J, Miyazaki Y, Berria R, *et al.* Relationship between hepatic/visceral fat and hepatic insulin resistance in nondiabetic and type 2 diabetic subjects. Gastroenterology 2007;133:496-506.
34. Hyatt TC, Phadke RP, Hunter GR, Bush NC, Muñoz AJ, Gower BA. Insulin sensitivity in African-American and white women: Association with inflammation. Obesity (Silver Spring) 2009;17:276-82.

Reviewers:	Editors:
Ashraf Elghul (Abu Dhabi, UAE)	Salem Beshyah (Abu Dhabi, UAE)
Khadija Hafidh (Dubai, UAE)	Elmahdi Elkhammas (Ohio, USA)