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CENTRAL ADIPOSITY RATHER THAN OVERALL OBESITY INFLUENCES CARDIO-METABOLIC RISK FACTORS AMONG ADULT MALES IN NORTHERN JORDAN

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SUMMARY

*The aim of the study was to examine the effect of normal and high waist circumference on the risk of coronary heart disease within the same body mass index categories. A cross-sectional study using the multistage cluster sampling technique was used to recruit study participants. A total of 948 apparently healthy adult Jordanian males aged 30-50 years were recruited. Dietary history and smoking habits were recorded. Body weight, height, and waist circumference were measured and body mass index was calculated as an indicator of overall obesity, whereas waist circumference was used to classify central adiposity. Blood pressure and blood samples were obtained to determine metabolic abnormalities and to estimate the risk of coronary heart disease. The mean waist circumference and body mass index of the study subjects were 89.9 cm and 27.2 kg/m^2, respectively. The prevalence of metabolic variable abnormalities and moderate or high risk of coronary heart disease were significantly (*P*<0.05) higher in the high waist circumference category as compared with normal waist circumference category within the three body mass index categories among Jordanian adult men. The results suggested that central adiposity rather than overall obesity contributed to the increase in the prevalence of metabolic abnormalities and risk of coronary heart disease.*

INTRODUCTION

During the last decade, coronary heart disease (CHD) was considered the leading cause of morbidity and mortality among adults in developed and developing countries (1-6). Nowadays, the incidence of CHD is decreasing in some western countries such as the USA, Western Europe, and Australia due to primary prevention through modification of CHD risk factors, whereas the incidence of CHD is steeply increasing in central and eastern Europe, and in developing countries (5-10), leading to substantial disability and being the main reason for the rising cost of health care, which is beyond the capacity of even the best health care systems. Understanding the CHD pathogenesis is critical to primary prevention at the population level, as well as to appropriate management of high risk individuals (i.e. secondary prevention). The American

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Heart Association (AHA) and American College of Cardiology have reported (11,12) that the traditional risk factors for CHD have included hypertension, total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), triglycerides (TG), cigarette smoking, diabetes, and advancing age (13,14). Overall obesity reflected by body mass index (BMI) and central adiposity reflected by waist circumference (WC) are designated by the AHA as major risk factors associated with an increased risk of CHD (15,16). The link between obesity and CHD is in part functional with the metabolic abnormalities (traditional CHD risk factors) imposed on CHD (17-22).

Obesity co-morbidities are more likely to present with silent disease and as a cluster of metabolic syndrome interact to increase the risk in a synergistic fashion, particularly among middle aged men (16,19,23,24). Obesity has developed dramatically and rapidly in Jordan in the last three decades. The prevalence of obesity (BMI ≥30) among Jordanian adult males in semi-urban communities was 32.7% (25). On the other hand, the prevalence of metabolic syndrome among Jordanian obese males in semi-urban communities was found to be 32.0% (26). CHD was the most important and leading factor of death in Jordan, whereas the economic cost of health care for Jordanians amounts to approximately one billion dollars (4). In Jordan, the need for more effective preventive strategies against CHD has become urgent and cannot be postponed.

The Adult Treatment Panel III (ATP III) guidelines foster a new perspective in the dual concept of global CHD risk assessment and primary prevention (16). The applicability of the concept of global CHD risk and obesity and metabolic syndrome associated with increasing levels of BMI and WC is not well understood among Jordanian adult males. This study using the methods described by ATP III, BMI-categories (BMI-C), and WC values established by WHO 1997 (27) was designed to examine the hypothesis on the significant (P<0.05) increase in the prevalence of metabolic abnormalities and to assess the CHD global risk using the Framingham Point Scoring System (FPSS) (2,28), with increasing WC values within the three BMI-C among Jordanian adult men.

METHODS

Selection and recruitment of study subjects

The participants were apparently healthy adult Jordanian males aged 30-50 years, from a semi-urban community with a homogeneous socioeconomic and cultural background residing in Al sarieh and Sal areas in Northern Jordan. The cross-sectional study used the multistage cluster sampling design to assess the prevalence of metabolic abnormalities and CHD global risk among obesity types and their classes. Subjects were considered eligible for inclusion in the study if they were apparently healthy, not receiving medications such as hypertensive, hypoglycemic and antihyperlipidemic drugs or drugs for cardiovascular disease, and were free from any clinical condition known to affect carbohydrate, protein or lipid metabolism or body composition. None of the study subjects had any acute illness, weight fluctuations of more than 5 kg during the last two months prior to testing, major ailment or disease for two years, or exposure to strenuous exercises. Recruited subjects should have been within BMI-C, normal weight, overweight and class I obese, and all participants with BMI ≥35.0 kg/m^2 were excluded from data analysis (WHO, 1997) (27).

In an initial interview, 1068 out of 1210 (88.3%) eligible subjects responded, of which 11.7% refused to participate in the study for personal reasons. All of the 1086 subjects (318 BMI <25 kg/m^2, 320 BMI >25<30 kg/m^2 and 405 BMI ≥30 kg/m^2) completed the study procedures. A total of 120 subjects were excluded on the basis of the study criteria, as they had frank diabetes mellitus (n=40), cardiovascular disease (CVD) (n=11), BMI <18.5 or ≥35 kg/m^2 (n=55) and hypertension (n=14). The remaining 948 subjects were included in statistical analyses.

The primary sampling unit considered the division of each study area into five clusters. Systematic sampling of households (every tenth house) was done after

random start had been selected. One resident for each BMI category (BMI-C) from each household was invited to participate if meeting the study criteria. Whenever there were more than one individual for each BMI-C, or for any BMI-C, one person was randomly selected. If the selected household had no volunteers and/or they were not fulfilling the study criteria, the next household was used.

Data collection and Framingham risk calculation

A modifiable and pilot pre-tested questionnaire was designed to collect information on demographic characteristics, smoking habits, history of weight fluctuations, and disease in study participants. Questionnaires were distributed through the study areas. The selected subjects understood the purpose of the research and showed their willingness to cooperate as reflected by thoroughness of their answers to the comprehensive questionnaire. The questionnaire was completed under the guidance of researchers and was carried out in the participant's home. An informed consent was obtained from each subject.

After the questionnaire was filled out, anthropometric measurements of body height, body weight and waist circumference were obtained for each participant by using the Anthropometric Standardization Reference Manual (28). Height was measured while the subject was barefoot and wearing minimal clothing to facilitate correct positioning of the body using an equidistant non-stretchable measuring tape and was recorded to the nearest 0.1 cm. Body weight was measured using an electronic scale (Seca, Germany). The subjects were weighed standing on their feet, barefooted and wearing light clothes and it was recorded to the nearest 0.1 kg. Waist circumference (WC) was measured using a non-elastic measuring tape at the narrowest level between the lowest rib and the iliac crest at the end of normal expiration and was recorded to the nearest 0.1 cm. BMI was calculated by dividing the weight in kilograms (kg) by height in square meters (m^2). Blood pressure was measured using standardized sphygmomanometers with a 12-12.5 cm cuff to cover two-thirds of the upper arm. The investigators performed the procedure while the subject was in sitting position with the arm at the level of the heart and after 15-min rest. The cuff was deflated at a rate of 2-3 mm Hg *per* second. Systolic blood pressure (SBP) was taken upon hearing the first sound, whereas diastolic blood pressure (DBP) was taken upon complete disappearance of Korotkoff sounds (phase V). Each subject was instructed to fast for 12-16 hours before blood sampling and to have refrained from smoking on the morning of testing. Blood sample was obtained from each participant to measure serum triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol, (LDL-C) and high-density lipoprotein cholesterol (HDL-C). Laboratory measurements were performed using standard automated procedures (Hitachi 911 auto-analyzer, Roche, Germany) with commercially available kits (Randox Roche Diagnostics, 2000). Blood samples were obtained at the International Academy Rehabilitation Sport Center. Data were collected during the period from March 2001 to May 2002.

Overall obesity was categorized according to BMI into three groups: normal, overweight and obese as indicated by BMI-C: <25, 25-29.99 and ≥30 kg /m^2, respectively (24). Central adiposity was categorized into two classes according to WC: normal (<94 cm) and high (>94 cm), following the International Diabetes Federation (IDF) criteria for WC (29). Categorization of biochemical parameters was based on the Adult Treatment Panel III (ATP III) criteria (30) taking into consideration borderline and above cutoffs as abnormal, with the exception of HDL-C where borderline and lower cutoffs were classified as abnormal.

Estimation of CHD risk for the next 10 years for each participant was calculated based on the Framingham risk scoring scheme (1), which takes into account the following risk factors: age, TC, HDL-C, SBP, and smoking habits; it was done in two steps: the first step was to calculate the number of points for each risk factor and then counting the total risk factor score for each participant. The second step was to classify the subjects into three categories: 10- year risk for CHD of >20% defined as high risk; 10%-<20% defined as

moderate risk; and <10% defined as low risk (2,12,30), and to define CHD risk by BMI and WC categories according to the obesity guidelines.

The data obtained were entered in the computer using SPSS (Statistical Package for Social Sciences, Windows version 9, 1997; SPSS, Inc., Chicago, IL, USA). Frequency and range checks were performed initially to detect errors in data entry. Detected errors were corrected by rechecking the original data forms. Analysis of variance (ANOVA) was used to test for any significant differences among means of lipid profile, SBP and BMI-C, whereas the probability χ^2-test examined the distribution of the prevalence of the estimated CHD risk in the next 10 years among BMI-C. The rate ratio was used to measure the degree of association between BMI-C and both lipid profile measurements and SBP. Statistical significance was set at $P<0.05$.

RESULTS

The mean age ± SD of study subjects was 39.0±6.36 years, mean SBP was 125.72 mm Hg, and mean DBP 82.3 mm Hg. Mean BMI was 27.2 kg/m^2 and mean WC 89.9 cm. The percentage of current smokers was 44.1%.

Differences between the means of SBP and DBP, fasting blood glucose, TC, LDL-C, HDL-C, and TG were statistically significant ($P<0.05$) among WC-C within the same BMI-C; the means of metabolic variables were significantly higher ($P<0.05$) among high WC groups compared with low WC groups, with the exception of HDL-C where it was lower (Tables 1 and 2).

The prevalence of metabolic abnormalities among obese subjects with high WC ranged from 31.1% to 74.8%, and in obese subjects with normal WC from 9.5% to 46.0%. In overweight subjects with high WC, it ranged from 21.6% to 68.6% and in overweight subjects with normal WC from 7.9% to 43.5%. In addition, in normal weight subjects with high WC it was from 2.3% to 22.2% and in normal weight subjects with normal WC from 0.7% to 13.5%. The prevalence of each metabolic abnormality was significantly ($P<0.05$) higher in the high WC group as compared with normal WC group within the same BMI-C.

The prevalence of estimated low CHD risk among obese subjects with high WC was significantly lower than in obese subjects with normal WC (63.3% *vs*. 68.2%; $P<0.05$). The normal weight subjects with high WC had a lower prevalence of low CHD risk as

Table 1. Mean age and metabolic variables in normal and high waist circumference groups according to body mass index categories (mean ± standard deviation)

	BMI categories					
	Normal weight		Overweight		Obese	
	Normal WC	High WC	Normal WC	High WC	Normal WC	High WC
Variable	(n =282)	(n =27)	(n =124)	(n =185)	(n =36)	(n =267)
Age (years)	38.5±6.3	42.6±4.8*	37.6±6.4	39.6±6.7	30.2±3.2	39.6±6.5*
Systolic BP (mm Hg)	120.4 ±7.5	122.1±17.3*	124.7±9.8	129.2±9.3*	126.8±11.1	133.9±13.9*
Diastolic BP (mm Hg)	77.1±6.9	79.9 ±9.5*	80.1±11.0	85.7±9.1*	91.2±8.8	93.8±9.5*
Fasting blood glucose (mg/dL)	98.9±7.7	104.0 ±12.9*	103.9±12.1	108.2±12.8*	107.0±9.1	112.5±11.2*
Total cholesterol (mg/dL)	181.7±29.9	212.3±29.8*	212.7±45.3	222.1±41.6*	219.3±35.4	228.0±39.4*
LDL-C (mg/dL)	107.7±33.1	138.7±33.9*	133.4±39.2	140.0±38.1*	147.2±39.0	150.1±39.5*
HDL-C (mg/dL)	46.3±12.3	37.6±9.6*	43.9±11.1	39.2 ±10.3*	43.0±12.9	37.9±10.1*
Triglycerides (mg/dL)	122±59.5	133.1±87.3*	160.6±75.6	180.6±85.6*	185.9±75.7	211.2±95.3*

BMI = body mass index; WC = waist circumference; BP = blood pressure; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; *significant difference in means between WC categories according to BMI classification ($P \leq 0.05$).
Normal WC was defined as WC <94 cm and high WC as WC ≥94 cm; body mass index classification (BMI categories): normal weight (BMI 18.5-24.9 kg/m2); overweight (BMI 25-29.9 kg/m^2); and obese (BMI >30 kg/m^2).

Table 2. **Comparison of prevalence of metabolic variable abnormalities in study sample with normal *vs.* high waist circumference values within different body mass index categories§**

	BMI categories					
	Normal weight		Overweight		Obese	
	Normal WC	High WC	Normal WC	High WC	Normal WC	High WC
Variable	(n =282)	(n =27)	(n =124)	(n =185)	(n =282)	(n =27)
Elevated Systolic BP (mm Hg)	0.7	2.3*	7.9	27.9*	9.5	47.6*
Elevated Diastolic BP (mm Hg)	1.4	3.2*	9.7	44.9*	17.9	74.8*
Impaired Glucose Tolerance (mg/dL)	2.1	11.1*	21.8	42.7*	25.3	47.2*
Elevated Total Cholesterol (mg/dL)	11.7	18.5*	19.4	25.9*	19.0	31.1*
Elevated LDL-C (mg/dL)	11.3	22.2*	33.3	54.1*	38.1	61.2*
Low HDL-C (mg/dL)	13.5	18.5*	32.3	49.2*	34.9	55.9*
Elevated Triglycerides (mg/dL)	12.1	14.8*	43.5	68.6*	46.0	69.7*

BMI = body mass index; WC = waist circumference; BP = blood pressure; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; § data given in this table represent abnormal measures and are presented as percent; *significant difference in means between WC categories according to BMI classification (P≤0.05).
Normal WC was defined as WC <94 cm and high WC as WC ≥94 cm; body mass index classification (BMI categories): normal weight (BMI 18.5-24.9 kg/m^2); overweight (BMI 25-29.9 kg/m^2); and obese (BMI >30 kg/m^2); elevated systolic BP ≥130 mm Hg; elevated diastolic BP ≥85 mm Hg; impaired glucose tolerance indicated by fasting plasma glucose of 110-126 mg/dL; elevated total cholesterol ≥200 mg/dL; elevated LDL-C ≥130 mg/dL; low HDL-C ≤40 mg/dL; and elevated triglycerides ≥150 mg/dL.

Table 3. **Prevalence of estimated coronary heart disease risk for the next 10 years in study sample with normal *vs.* high waist circumference values within different body mass index categories§**

	BMI categories					
	Normal weight		Overweight		Obese	
	Normal WC	High WC	Normal WC	High WC	Normal WC	High WC
CHD Risk Categories	(n =282)	(n =27)	(n =124)	(n =185)	(n =282)	(n =27)
Low (< 10%)	95.0	87.6	89.9	70.3	68.2	63.3
Moderate (10 - < 20%)	3.9	8.5	7.3	20.0	22.2	25.5
High (≥ 20%)	1.1	3.9	2.8	9.7	9.6	11.2

BMI = body mass index; WC = waist circumference; CHD = coronary heart disease; § data presented as percent; normal WC was defined as WC <94 cm and high WC as WC ≥94 cm; body mass index classification (BMI categories): normal weight (BMI 18.5-24.9 kg/m^2); overweight (BMI 25-29.9 kg/m^2); and obese (BMI >30 kg/m^2).

compared with normal weight subjects with normal WC (87.6% *vs*. 95.0%; $P<0.05$). In addition, the prevalence of estimated high CHD risk increased significantly ($P<0.05$) in subjects with high WC all across BMI-C and reached highest value in obese subjects with high WC (11.2%) (Table 3).

DISCUSSION

In this cross-sectional study, we found that central adiposity as reflected by higher WC increased the prevalence of metabolic abnormalities and CHD risk among semi-urban Jordanian adult males and it could be a better predictor of the risk of CHD than BMI. The results of this study indicated the risk of metabolic abnormalities and CHD to be significantly greater in normal-weight, overweight, and obese adult males with high WC values as compared with normal-weight, overweight, and obese adult males with normal WC values. The highest prevalence of metabolic abnormalities, ranging from 31.1% to 74.8%, and of high CHD risk category (11.2%) was found among obese subjects with high WC values. Despite the fact that the study participants were apparently healthy, the findings indicated that obesity and metabolic abnormalities such as elevated SBP and DBP, fasting blood glucose, TC, LDL-C, TG and low HDL-C were more likely to be silently present and interact to increase the risk in a synergistic fashion. Thus, metabolic abnormalities were present as a

cluster (metabolic syndrome) in middle aged obese men, particularly those with central adiposity. This finding confirms the reports from other studies (20-22,31-33). Our study has emphasized that central adiposity provides predictive power for metabolic complications beyond that provided by BMI. On the other hand, there is little doubt that the increasing prevalence of overweight/obesity is mainly responsible for the rising prevalence of the metabolic abnormalities in northern Jordan. These results are in agreement with numerous studies and reports (1,13,19,20,23,24,31,33-36). It is well known that obesity causes insulin resistance, whereas insulin resistance seemingly exacerbates the adverse effects of obesity (19,32,33). Also, the relation between obesity and metabolic risk factors is based on the discovery of multiple products released from adipocytes in abnormal amounts in the presence of obesity, particularly central adiposity, which is more bioactive than overall obesity (20). This bioactivity includes increased production of nonesterified fatty acids, inflammatory cytokines, leptin, prothrombotic factors and decreased production of adiponectin, a putative adipokine, plasminogen activator inhibitor (PAI)-1, C-reactive protein (CRP), fibrinogen, and resistin. Each of these products has been implicated in the causation of one or another of the metabolic risk factors (31,32,37-42).

Our study results may indicate the presence of two subtypes of obesity among Jordanian adult males that have been reported in the scientific literature (43). One subset of individuals have been termed the metabolically healthy but obese (MHO), a condition that is more prevalent among overall obesity as compared with central adiposity. Another subset, termed the metabolically obese but normal weight (MONW) are more prevalent among central obese as compared with overall obese participants. The study results showed that MHO individuals may be well represented and could account for as much as 20% of the obese middle aged male adults in northern Jordan. These findings are in agreement with several studies (24,43-47).

Obesity and CHD risk

The link between obesity and CHD is in part functional with the metabolic abnormalities imposed on CHD, as also stated in a number of recent reports (17,18,20,22,31,49,50). The obesity-induced metabolic syndrome is a multidimensional risk factor for CHD. Persons with the metabolic syndrome have at least a 2-fold risk of CHD compared with those without it (19,51-54).

The study showed that the prevalence of estimated low risk of CHD category for the next 10 years decreased significantly (P=0.000) in high WC class compared to normal WC class within the same BMI-C, contrary to the estimated moderate and high CHD risk categories. These findings are in agreement with other studies, which showed that overweight, moderate and high central adiposity were associated with increased CHD morbidity (3,20-22,52-59). Weight gains of 5-8 kg increased the risk of CHD by 25% (60). In British men, the incidence of CHD increased at BMI above 22 and an increase of 1 BMI unit was associated with 10% increase in the rate of coronary events (61). Similar relationships between increasing BMI and CHD were demonstrated in Finish, Swedish, Japanese, Australian and US populations (5,20,60,62). On the other hand, Canoy *et al*. (21) report that reducing weight by 1 kg could be translated to reducing CHD risk by 2% in both men and women, and alternatively reducing WC by 5 cm could lower the risk by 11% in men and 15% in women.

Waist circumference and body mass index classifications

Study results underlined the importance of incorporating BMI and WC evaluation into the routine clinical practice and provided substantial evidence that the WHO 1997 cutoff points for WC help identify those at an increased health risk within the various BMI categories (27). However, the classification system employed in the present study uses a categorized approach (normal WC <94 cm, low risk *vs*. high WC >94 cm, high risk) to establish association between WC and risk of metabolic abnormalities and

CHD. The findings of this study suggested the WC cutoffs to be lower than the WHO 1997 cutoffs because the study participants with WC >94 cm had a high prevalence of metabolic abnormalities. This is generally in agreement with the reports from several studies conducted in Asian populations (13,63,64). Our results suggest that lower cutoffs for BMI and WC are needed in the identification of Jordanian males at a high risk of CHD.

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