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Estimated risk of coronary heart disease in obese adult males in Northern Jordan

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ABSTRACT

Objectives: To examine the relationship between obesity, lipid profile and blood pressure, and to quantify the risk of coronary heart disease (CHD) for the next 10 years, using the Framingham risk scoring scheme among Jordanian adult males.

Methods: We conducted this study in Al-Sarieh, Jordan during the period March to May 2001. A total of 306 apparently healthy adult males, aged 30-50 years completed all the study procedures. We selected the participants using a multi-stage cluster sampling design. Dietary history and smoking habits were obtained using a pre-tested questionnaire and interview. Blood samples were obtained and examined for lipid profiles. We measured the blood pressures, as well as the weight and height to calculate the body mass index (BMI). The sample was categorized into 3 groups using the World Health Organization classifications for BMI. The risk of CHD was calculated using a scoring scale according to Framingham scheme. Analyses of data were carried out using the Chi-square test, and the Analysis of Variance.

Results: The mean age of the subjects was 39 years with a mean BMI of 28.2 kg/m^2. The percentage of current smokers was 44.1%. The mean of serum total cholesterol, triglycerides, low density lipoprotein cholesterol and systolic blood pressure, increased significantly with increasing BMI categories, whereas the mean of high density lipoprotein cholesterol decreased with increasing BMI categories. Prevalence of medium and high risk of CHD significantly increased as BMI categories increases.

Conclusion: The prevalence of estimated CHD risk for the next 10 years in moderate and high CHD categories increases as the BMI categories increases among Jordanian adult men in Al-Sarieh area.

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Coronary heart diseases (CHD) continue to be a leading cause of morbidity and mortality among adults worldwide including Jordan.[1-4] The past decade has witnessed major strides in the prevention of CHD through modification of its risk factors. The major and independent 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.[5,6] Obesity and physical inactivity are designated by the American Heart Association (AHA) as the major risk factors associated with the increase risk for CHD.[7,8] In addition, several studies and reports in western population indicated that the adverse effects of obesity on the coronary heart system are partly functional

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with the metabolic abnormalities imposed on CHD.[9-12] The AHA and the American College of Cardiology have published joint recommendations by targeting modifiable independent CHD risk factors for medical intervention in patients with CHD (namely, secondary prevention). They also emphasized the importance of preventive measures in healthy individuals with a high risk of CHD (namely, primary prevention). Thus, effective primary prevention requires an assessment of risk to categorize patients for selection of appropriate interventions.[13] Several studies and methods for CHD risk prediction had been suggested.[1,2,5,6,14-20] These methods enable the calculation of an individual's coronary multivariable risk score as a function of their values for selected established CHD risk factors for the next 10 years. The best accepted methods that evaluate algorithms or scores are those derived from the United State-Framingham study[1] and German-PROCAM study.[2]

Cardiovascular diseases are the first leading cause of death in Jordan.[4] Recent studies conducted in Jordan indicated high prevalence of obesity, diabetes mellitus, hypertension and hyperlipidemia.[21-25] This study examines, the relationship between obesity, systolic blood pressure (SBP) and lipid profile, to estimate CHD risk for the next 10 years using Framingham risk scoring scheme for the study population.

Methods. A cross-sectional study was conducted at Al-Sarieh, Northern Jordan during the period March to May 2001. Multi-stage cluster sampling was used to recruit the participants. The primary sampling unit considered the division of the study area into 5 clusters. The systematic sample of households (every tenth house), after a random start was selected. One resident for each body mass index category (BMI-C), from each household, was invited to participate; if the study criteria was fulfilled. Whenever there was more than one for each BMI-C, or for any of BMI-C, one person was randomly selected. If the selected household had no volunteers, and they were not fulfilling the study criteria, the next household was used. Inclusion criteria of the study population included subjects who were apparently healthy, not receiving medications such as hypertensive, hypoglycemic, and anti-hyperlipidemia drugs or drugs for cardiovascular disease (CVD) or had any clinical conditions known to affect carbohydrate, protein or lipid metabolism or body composition. None of the patients had acute illness, weight fluctuations more than 2 kg during the last 6 months prior to testing, major ailment or disease for 2 years, or enrolled in strenuous exercises. The subjects aged 30-50 years were within the BMI-C (WHO, 1997).[26]

In an initial interview, 350 out of 400 (87.5%) of the eligible subjects responded, of which 12.5% refused to participate in the study for personal reasons. All of the 350 subjects (108 BMI <25 kg/m^2, 110 BMI ≥25 <30 kg/m^2, and 132 BMI ≥30 kg/m^2) completed the study procedures. A total of 44 subjects were excluded on the basis of the study criteria; frank diabetes mellitus (22), CVD (7), taking drugs for CVD (7), underweight BMI <18.5 kg/m^2 (4), and hypertension (4). The remaining 306 subjects were included in statistical analyses. A modifiable and pilot pre-tested questionnaire was designed to collect information regarding the demographic characteristics, smoking habits, history of weight fluctuations, and disease of the participants. Questionnaires were distributed through the study area. 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. A questionnaire was completed under the guidance of the researchers and was carried out in the participant's home. An informed consent was obtained from each subject. After a questionnaire was filled, anthropometric measurements were obtained for each participant by using the Anthropometric Standardization Reference manual.[27] All subjects were weighed on an equilibrated portable balance scale (Seca, Germany). The weight was taken without shoes and while wearing only the indoor clothing or very light clothes. Weight was read to the nearest 100 g. Standing height was measured without shoes using a tape measure fixed to the wall. The blood pressure was measured using standardized sphygmomanometers with a 12-12.5 cm cuff to cover two-thirds of the upper arm. Normal SBP (<130 mm Hg) was based on Adult Treatment Panel III (ATP III) criteria. Body mass index was calculated by dividing the weight in kilograms (kg) by height in meters squared (m^2). Each subject was instructed to fast for 12-16 hours before the blood samples were obtained and to refrain from smoking on the morning of the tests. The blood sample was obtained from each participant to measure the serum TG, TC, LDL-C, and HDL-C. Laboratory measurements were performed using standard automated procedures (Hitachi 911) with commercially available kits (Randox Roche Diagnostics, 2000). The blood samples were obtained at the International Academy Rehabilitation Sport Center, Irbid, Jordan.

The samples were categorized according to there BMI into 3 groups, normal, overweight and obese as indicated by BMI-C: <25, 25-29.99 and ≥30 kg/m^2.[27] Categorization of biochemical parameters was based on ATP III criteria.[8] Estimation of CHD risk for the next 10 years for each participant was based

Table 1 - Distribution of the mean values of serum lipid profile and systolic blood pressure according to body mass index categories.

Dependent variables	BMI-C	Mean ± SD	BMI - C	Mean difference	± STD error	*P*-value
Triglycerides	0	126.4 (59)	0 versus 1	-52.6	11.4	0.000
	1	179 (84.8)	2 versus 0	84.2	11.3	0.000
	2	210.7 (95.6)	2 versus 1	31.6	11.4	0.022
Total cholesterol	0	179.8 (29.8)	0 versus 1	-33.7	5.7	0.000
	1	213.5 (45)	2 versus 0	52.5	5.7	0.000
	2	232.3 (45.4)	2 versus 1	18.8	5.7	0.005
High density lipoprotein cholesterol	0	47.3 (9.7)	0 versus 1	4	1.3	0.011
	1	43.3 (8.8)	2 versus 0	-7.1	1.3	0.000
	2	40.1 (9.5)	2 versus 1	-3.2	1.3	0.056
Low density lipoprotein cholesterol	0	107.1 (29.4)	0 versus 1	-27.3	5.1	0.000
	1	134.4 (39.8)	2 versus 0	42.8	5.1	0.000
	2	149.9 (39.5)	2 versus 1	15.5	5.1	0.011
Systolic blood pressure	0	120.4 (7.5)	0 versus 1	-3.2	1.5	0.101
	1	123.6 (9.9)	2 versus 0	12.7	1.5	0.000
	2	133.1 (13.3)	2 versus 1	9.6	1.5	0.000

BMI-C - body mass index categories, Results of one-way ANOVA and Scheffe post hoc analysis - Category 0 - Normal BMI (n=103), Category 1 - overweight BMI (n=100), Category 2 obese BMI (n=103), STD - standard error

on Framingham risk scoring scheme,[1] which account the following risk factors; age, TC, HDL-C, SBP, and smoking habits using 2 steps: the first step was to calculate the number of points for each risk factor and then counting the total risk factors score for each participant. The second step was to classify the subjects into 3 categories: 1) Those with 10 years risk for CHD of ≥20% defined as high risk, 2) 10 -<20% defined as moderate risk, and 3) Those with <10% defined as low risk.[2,7,8,13]

The data was entered into a computer using the Statistical Package for Social Sciences version 9. Frequency and range checks were performed initially to detect errors in the data entry. Detected errors were corrected by rechecking the original data forms. Analysis of variance was used to test for any significant differences among means of lipid profile, SBP, and among BMI-C. Whereas the likelihood Chi square test examined the distribution of the prevalence of the estimated CHD risk in the next 10 years among BMI-C. Rate ratio was used to measure the degree of the association between BMI-C and both lipid profile measurements and SBP. The *p*-value of ≤0.05 was considered statistically significant.

Results. The mean age ± SD of subjects was 38.97 ± 6.46 years, the mean SBP was 125.72 mm Hg, and the mean BMI was 28.16 kg/m^2. The mean of TG was 171.95 mg/dl, TC 208.48 mg/dl, HDL-C 43.57, and LDL-C 130.42 mg/dl. The percentage of current smokers was 44.1%. The means of serum TG, TC, LDL-C, and SBP increased with increasing BMI-C, whereas the mean of HDL-C decreased with increasing BMI-C (**Table 1**). The differences between the means of serum TG, TC, HDL-C, LDL-C, and SBP, were statistically significant (p<0.05) among the BMI-C (**Table 1**). The prevalence of estimated low risk of CHD category for the next 10 years decreased significantly (p=0.000) as BMI-C increase, contrary to the estimated moderate and high CHD categories (**Table 2**).

Table 3 summarizes the prevalence rates and rate ratios of the subjects in relation with TG, TC, HDL-C, LDL-C and BMI-C. Overweight subjects had rate ratios of adverse lipid profile of 1.72-14.24 times and obese of 2.63-17.11 times, compared with normal body weight. Whereas, overweight subjects compared with normal body weight had rate ratios of elevated SBP of 12.9 and obese 43.8 times.

Discussion. This study provides data on the relationship of BMI CHD risk and estimation of CHD risk for the next 10 years among Jordanian adult males aged 30-50 years in Al-Sarieh area for the first time. The study findings indicate that the mean levels of the lipid profile increased significantly (p=0.000) as BMI- C increases, whereas the mean level of HDL-C

Table 2 - Prevalence of estimated coronary heart disease risk for the next 10 years among body mass index categories in the studied sample.

Coronary heart disease risk categories	Body mass index categories (%)				
	Normal	Overweight	Obese	Total	*P*-value
Low (<10%)	92 (89.3)	78 (78)	67 (65)	237 (77.4)	0.000
Moderate (10 - <20%)	10 (9.7)	15 (15)	26 (25.2)	51 (16.7)	0.000
High (≥20%)	1 (1)	7 (7)	10 (9.7)	18 (5.9)	0.000
Total	**103 (33.7)**	**100 (32.6)**	**103 (33.7)**	**306 (100)**	

Table 3 - Prevalence and rate ratios of subjects of serum lipid profile and systolic blood pressure categories among body mass index categories.

BMI-C	LC (mg/dl)	Normal (%)	Overweight (%)	RR*	Obese (%)	RR†	Total (%)	*P*-value
Normal TG	<150	71.8	41	0.6	31.1	0.4	48	0.000
Border line-high TG	150 - 199	23.3	29	1.3	18.4	0.8	23.5	0.000
High TG	≥200	4.9	30	6.1	50.5	10.3	28.4	0.000
Desirable TC	<200	88.3	45	0.5	27.2	0.3	53.6	0.000
Border line-high TC	200 - 239	7.8	31	4	37.9	4.9	25.5	0.000
High TC	≥240	3.9	24	6.2	35	9	20.9	0.000
Low HDL-C	<40	21.4	37	1.7	56.3	2.6	38.2	0.000
Normal HDL-C	40 - 59.9	68	59	0.9	39.8	0.6	55.6	0.000
High HDL-C	≥60	10.7	4	0.4	3.9	0.4	6.2	0.000
Optimal LDL-C	<130	82.5	54	0.7	38.8	0.5	58.5	0.000
Border line-high LDL-C	130 - 159	10.7	21	2	27.2	2.5	19.6	0.000
High LDL-C	≥160 - 189	5.8	13	2.2	20.4	3.5	13.1	0.000
Very high LDL-C	≥190	1	12	12	13.6	13.6	8.8	0.000
Normal SBP	<130 mm Hg	94.2	76	0.8	40.8	0.4	70.3	0.000
Moderate SBP	130 - <140	4.8	14	2.9	19.4	4	12.7	0.000
High SBP	≥140	1	10	10	39.8	39.8	17	0.000

BMI-C - body mass index categories, LC - lipid profile categories, TG - triglycerides, TC - total cholesterol, HDL-C - high density lipoprotein cholesterol, LDL-C - low density lipoprotein cholesterol, SBP - systolic blood pressure, RR - rate ratios, *overweight/normal, †obese/normal

decreases significantly (p=0.000) as BMI- C increases. Also, the study indicates that prevalence rates of adverse serum lipid profile increase significantly (p=0.000) as BMI-C increases. These findings are in agreement with the findings of other studies.[18,28-31] The explanation of adverse serum lipids in the study population could be attributed mainly due to obesity whereas food consumption, lifestyle and genetic factors could play a minor role. Furthermore, these results of adverse serum lipid profile may indirectly support the theories, which claim that the adverse serum lipid profile among obese subjects may be due to improper coordination of lipoproteins, receptors, modifying enzymes, substrate availability, and other factors involved in lipoprotein synthesis, secretion, modeling, and clearance.[32,33]

The study demonstrated a significant (p=0.000) association between BMI with elevated SBP. This finding is in agreement with other studies carried out in eastern and western populations.[25,34,35] This result indirectly supports the pathophysiology underlying the development of hypertension among obese subjects, which includes sodium retention and associated increases vascular resistance, blood volume and cardiac output. The relationship between elevated SBP and CVD abnormalities are believed to be related to a combination of increased sodium retention, increased sympathetic nervous system activity alterations of rennin-angiotensin system and insulin resistance.[36-38] The study showed that the estimated high and medium CHD risk increases as BMI-C increases, whereas low CHD risk decreases as BMI-C increases. These findings are in agreement with other studies, which had shown that overweight and obesity are associated with increased morbidity from CHD.[3,15,18,29-31] Weight gains of 5-8 kg increased the CHD risk by 25%.[39] In British men, CHD incidence increased at BMIs above 22 and an increase of one BMI unit was associated with 10% increase in the rate of coronary events.[40] Similar relationships between increasing BMI and CHD have been shown in Finish, Swedish, Japanese, and US populations.[39,41,42]

We conclude that generalized obesity is a major predictor of lipids profile abnormalities and elevated SBP. The prevalence of estimated CHD risk for the next 10 years in moderate and high CHD categories increases as BMI-C increases among Jordanian adult men in Al-Sarieh area. Therefore, the study recommends that concentrated efforts should be devoted to the prevention and treatment of obesity rather than just to treat its associated co-morbidities only.

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