GENERALIZED OBESITY, SERUM LIPIDS AND LIPOPROTEINS IN ADULT MALES IN A SEMI-URBANCOMMUNITY IN THE NORTH OF JORDAN

Omar K. ALboqai, Ph.D. , Riyad M. Saraireh Msc*, Munib A. Saif, MD**, Marwan M. Diab Msc ^.*

ABSTRACT

Objective: To describe the relationship between generalized obesity with serum lipids and lipoproteins.

Methods: Multi stage sampling technique was used to select the participants from Alsareeh area. A total of 400 apparently healthy adult males aged 30-50 years were invited to participate in the study, of whom 306 (103 non-obese, 100 overweight, 103 obese) completed the study.
A pilot tested interview questionnaire was designated in the study to collect the dietary history. Triglycerides, total cholesterol, and high density lipoprotein cholesterol were measured, whereas low density lipoprotein cholesterol and total cholesterol to high density lipoprotein cholesterol ratio were calculated. A dverse serum lipids and lipoproteins were categorized based on National Institute of Health (NIH,1998). Obesity was categorized into three groups as indicated by BMI-C WHO (1997) for generalized obesity.

Results: There was an increases in the prevalence rate of adverse serum lipids and lipoproteins as BMI level increases. Their prevalence rate among overweight and obese subjects compared with non-obese subjects were 2.1-8.9 and 4.9-10.9 times respectively.
Adjusted odds ratios of adverse serum lipids and lipoproteins after controlling for confounded factor among overweight and obese subjects compared with non-obese were 5.08-6.27 (95%CI) and 11.59 -13.8 (95%CI) respectively.

Conclusions: Amount of body fat is the major risk factors on adverse serum lipids and lipoproteins which emphasizes the need for concentrated efforts to prevent and treat obesity rather than just its associated diseases.

Keywords: Obesity, Body mass index (BMI), Serum lipids lipoproteins, Jordan

JRMS June 2006; 13(1): 39-45

Introduction

Obesity is a widespread metabolic abnormality. It is a growing epidemic worldwide including Jordan and has affected people with different ethnic and cultural backgrounds [1-3].

Obesity has long been recognized as an associated factor with a variety of adverse health consequences chiefly among which are diabetes, hypertension, adverse serum lipids and lipoproteins, increased cardiovascular events and mortality [4-7]. These patients are also more likely to present with silent disease and as a cluster of metabolic syndrome [5,8]. and as a cluster of metabolic syndrome [1,5,8]. The most commonly recognized risk factors in the metabolic syndrome are highly correlated with each other and are pre-sumed to reflect common metabolic pathways and they interact to increase risk in a synergistic fashion [1,8].

Furthermore, several reports and epidemiological studies at international and regional levels reported a positive association between obesity and adverse serum lipids and lipoproteins [9-18]. Increases in serum lipoproteins concentration showed positive association with body mass index (BMI) [9-15], waist circumference [16-18], and waist to hip ratio (WHR) [5,14,18-20]. The risk was increased among men who had been overweight in childhood or/and adolescence [20,21]. It has been

From Departmets of :
*Nutrition & General Services, Royal Medical Services College (RMSC), Marka-Jordan
** Family Medicine, Princess Aisha Medical Complex, Marka - Jordan
^ Nursing, Princess Aisha Medical Complex, Marka - Jordan
Correspondence should be addressed to Dr. Dr O. Alboqai, (RMSC)
Manuscript received April 6, 2005. Accepted August 18, 2005.

suggested that other factors, such as dietary fat from animal source, genetic susceptibility, sedentary life style and age, may also play a role in producing adverse serum lipids and lipoproteins [19,22-24]. Weight reduction could also reverse the abnormal biochemical characteristics of obesity [1,2,5]. Most Clinical studies assessing the health effect of generalized obesity rely on BMI [1,2].

Recent studies conducted in Jordan indicate high prevalence of diabetes mellitus, hypertension and dyslipidemia. Also cardiovascular diseases are the first leading cause of death in Jordan as reported by annual reports of Royal medical services [25-29]. Data are needed regarding the relationship between obesity with adverse serum lipids and lipoproteins in Jordan.

Methods

A multi stage cluster sampling design was used to recruit the studied sample. The primary sampling unit considered the division of the study area into five clusters. Systematic sample of households, (every tenth house), after a random start was selected. One resident for each BMI categories, from each household, was invited to participate; if fulfilled the study criteria. Whenever there was more than one for each BMI categories, or for any of BMI categories, one person was randomly selected. If the selected household had no volunteers, or they rejected to participate,and/ or they were not fulfilling the study criteria the selected one was from the next closest household. sample size calculation used the following formula: $N = (Z_{\alpha}.Z_{\beta})\ \sigma^2/\mu_1 - \mu_2$

The determined sample size based on the formula was 63 subjects for each group. Inclusion criteria of the study were subjects who were initially healthy, none was receiving medications such as hypertensive, hypoglycemic and anti-hyperlipidemia drugs or drugs for cardiovascular disease or had any clinical conditions known to affect carbohydrate, protein or lipid metabolism or body composition. None had acute illness, weight fluctuations more than 2 kg during the last six months prior to testing, major ailment or disease for two years, or enrolled in strenuous exercises. Subjects had to be between the age of 30-50 years, lived most of their lives in Jordan and their body mass index (BMI) within BMI categories (WHO, 1997).

Grouping and exclusion of subjects

In an initial interview, to which 350 out of 400 (87.5%) of eligible subjects responded, where as 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) had completed questionnaires, anthropometric measurements, and biochemical parameters. A total of 44 subjects were excluded on the basis of the study criteria. After obtaining the complete data on a total of 350 Jordanian adult males aged 30-50 years 44 subjects were excluded on the basis of the study criteria as shown in (Table I. IV). 306 remaining subjects (103 BMI< 25 kg/m^2, 100 BMI ≥ 25 <30 kg/m^2 and 103BMI ≥ 30 kg/m^2) were involved in the final study sample number on which the statistical analysis would be carried out.

Table I. Distribution of subjects with characteristics excluding them from the study sample.

Frank diabetes mellitus	22
Cardiovascular disease	7
Taking drugs for (CVD)	7
Underweight (BMI < 18.5 kg/m^2)	4
Hypertension	4
Total	44

The sample was categorized into approximately equal three groups, non-obese, overweight and obese as indicated by BMIs: < 25, 25-29.99 and ≥30 kg /m² respectively [1].

A modifiable and pilot tested interview questionnaire was designed in the study to collect demographic, socio-cultural, dietary data using food practice questionnaire, dietary history, current smoking, history of fluctuations (weight and disease for participants) and their parents. The questionnaire also incorporated 24-hour recall food consumption; it consisted of 42 food items. Food quality and food preparation methods and the usual frequency of food intake weekly were recorded under the researcher's supervision and advice concerning various utensil sizes: plates, spoon, and cups models were specified to help in the estimation of portion sizes.

Nutrient intakes were calculated using the food composition tables for the Middle East [30]. The percentage daily intake of energy, carbohydrates, protein and fats for individuals were calculated and classified into categories. The percentage daily dietary intake of fat calories from total calorie (%CDF): %CDF value ≤33%=%CDF-C (1), >33 - ≤34.5= %CDF-C (2), and>34.5% = %CDF-C (3).

Questionnaires were distributed through the research area. The selected subjects understood the purpose of the research and showed their willingness to cooperate as reflected by thoroughness of their answers to comprehensive questionnaire. Questionnaire was completed under the guidance of authors and was carried out in the participant's home. An informed consent was obtained from each subject.

After a questionnaire was filled and anthropometric measurements obtained for each subject by the author, each subject was instructed to fast for 12-16 hours before the blood samples were obtained and to have refrained from smoking on the morning of the tests. Blood sample was obtained from each participant to measure serum triglycerides (TRIG) and total cholesterol (TC), low-density lipoprotein cholesterol, (LDL-C), high-density lipoprotein cholesterol (HDL-C) and total cholesterol (TC) to (HDL-C) ratio (TC/HDL-C). Laboratory measurements were performed using standard automated procedures (Hitachi 911) with commercially available kits (Randox Roche Diagnostics, 2000). Increased serum triglycerides(TRIG-1) is defined as serum(TRIG) level of ≥ 200 mg/dl, whereas elevated serum TC (TC-

1)defined as serum TC level of ≥ 200 mg/dl for men aged < 40 years, ≥ 240 mg/dl for men aged ≥ 40 years. Elevated low-density lipoprotein cholesterol (LDL-C-1) is often defined as serum concentration of ≥ 160 mg/dl. Low high-density lipoprotein cholesterol (HDL-C (1)) is often defined < 35 mg/dl for men. Elevated (TC/HDL-C-1) is often defined as serum concentration of > 5 [(2)].

Anthropometrical measurements such as weight, height, they were performed using the Anthropometric Standardization Reference Manual[31].. Anthropometrical measurements and Questionnaire were completed under the guidance of author and were carried out in the participant's home. Whereas blood samples were obtained at the International Academy Rehabilitation Sport Center. Data collection was conducted during March to May 2002.

The data was entered into a computer using the SPSS (statistical Package for Social Sciences, Windows version 8x, 1997; SPSS Inc, Chicago, 1L, USA. Frequency and range checks were performed initially to detect errors in data entry. Detected errors were corrected by rechecking the original data formed.

The prevalence of adverse serum lipids and lipoproteins comparing proportions in the study sample among population subgroups based on genderalized obesity categories were analyzed using X^2 test. Analysis of variance (ANOVA) was performed to test significance for mean values of variables between genderalized obesity categories.

Logistic regression analysis was carried out to estimate risk or odds ratios (ORs) of obesity indices, selected biological and environmental factors on adverse serum lipids and lipoproteins.

Results

The mean of serum TRIG, (TC), LDL-C and (TC/HDL-C) increased with increasing BMI categories, whereas the mean of HDL-C decreased with increasing BMI categories, and showed significant mean differences among BMI-C ($P<0.05$) as shown in Table II.

Table III. shows prevalence rates and ratios of the proportion of subjects with TRIG-1,TC-1 ,HDL-C-1, LDL-C-1 and (TC/HDL-C-1) among overweight and obese subjects compared with non-obese subjects were (2.1-8.9) and (4.9-10.9) times respectively.

Tables (4-8) shows The adjusted odds ratios of adverse serum lipids and lipoproteins among overweight and obese subjects compared with non-obese were (ORs 5.08-6.27, 95%CI) and (ORs 11.59 -13.8, 95%CI) respectively. Also participants who reported presence history of parental cardiovascular disease (CVD) had significant odds ratios of increased (TRIG), low HDL-C and elevated (TC/HDL-C) ranging from (1.71-2.21, 95%CI) as shown in (Tables IV,VI,VIII). participants whose % of caloric intake from dietary fat was ≥34.5 compared with participants whose % of caloric intake from dietary fat was ≤ 33% had significant odds ratios on increased (TRIG),elevated total cholesterol , low-high-density lipoprotein cholesterol and elevated total to high-density lipoprotein cholesterol ranging from (1.89 - 3.22, 95%CI) as shown in Tables IV, V, VII, VIII.

Tables IV, VII shows that childhood obesity had significant odds ratios on elevated total cholesterol, high low-density lipoprotein cholesterol. Adolescence obesity was associated significantly with increased triglycerides as shown in Table IV.

Discussion

The present study provides the first data on the relationship of generalized obesity with serum lipids and lipoproteins. High prevalence rates of overweight and obesity in developed and developing countries is recognized as a major health problem that may be important in increasing the occurrence of other chronic diseases [(1,2)]. Elevated triglycerides , total cholesterol , high LDL-C, low HDL-C and elevated TC/HDL-C were associated with (CVD) [(2,7,15,17-19,22, 24)].

The study findigs showed in Table I. that the means of lipid and lipoprotein levels increases as BMI- C increases except the mean of HDL-C decreases as as BMI- C increases. Table II. The (PRs) of adverse serum lipids and lipoproteins among overweight and obese subjects compared with non-obese subjects were (2.1-8.9) and (4.9-10.9) times respectively.

The study findings shown in Tables (III - VII) demonstrate that generalized obesity indices were the dominant independent risk factors on adverse serum lipids and lipoproteins. The adjusted odds ratios (ORs) of adverse of serum lipids and lipoproteins after controlling for confounded factors among overweight and obese subjects compared with none-obese were (ORs 5.08-6.27, 95%CI) and (ORs 11.59 -13.8, 95%CI) respectively.whereas adolecence and childhood obesity, parental CVD, age and %CDF ≥34.5 were almost associated significantly with adverse serum lipids and lipoproteins.

The study conclusion with regards to adverse serum Lipid and lipoproteins agrees with the finding of several studies [(9-15)]. But the magnitudes of adjusted risk (OR) were less than the values in this study.

The present study strongly suggests that body mass index (BMI), as generalized obesity indicator was the strongest independent factors associated with adverse serum lipids and lipoproteins. With regards to increased serum triglycerides ,this result may be considered as indirect evidence that two basic mechanisms exist for increased triglycerides among obese, may be due to overproduction of very low-density lipoprotein (VLDL) and defective lipolysis of the triglycerides (TRIG) rich lipoprotein [(4)]. Explanation for the high level of serum cholesterol in the study population may be because of food consumption, life style and genetic factors could be of minor role, while obesity was largely responsible for our findings. whereas The results of adverse lipoprotiens indirectly support the theories which claim that adverse lipoprotiens 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 [5].

Findings shown in Tables (III-VII) indicate that subjects who reported a history of childhood and adolescence obesity in comparison with subjects who have not reported almost had a significant risk of adverse of serum lipids and lipoproteins . These results are in agreement with the results of several reports and studies [2,6,20,21]. The effect of adolescent obesity and childhood obesity on adult morbidity and mortality may be due either to direct effects of childhood and adulthood obesity or to other mechanisms, including genetic factors that entrain both body fatness and adult morbidity, the changes in body fat distribution that occur in the two periods , also the ratio of intra-abdominal adipose tissue to total body fat increases almost three folds [16,20,22]. This will represent a logical mechanism to explain the effects of adolescent obesity on adult morbidity and mortality, also may be due to long duration of obesity which may reflect the time it takes for the metabolic and other effects of obesity to have their full impact.

Investigations in this study show that parental CVD, age and %CDF ≥34.5 were associated significantly with adverse of serum lipids and lipoproteins shown in Tables (III-VII). These results are in agreement with several studies [19,22-24].

Limitations of the study:

One region from semi urban community in jordan might not apply to other region such as urban community.

Lipids and lipoproteins classifications based on(NIH,1998), its better to use the recent classifications Adult Treatment panel 111(ATP111, 2003).

Based on the study findings we conclude that adverse of serum lipids and lipoproteins are mainly due to obesity. So, obesity becomes an important health problem in Jordan, comparable to that in Europe and the United States. Health education campaigns with the purpose of changing the life style of the society, e.g. dietary habits, weight management, smoking, management of obesity in children, adolescence and adults, serum lipids and lipoproteins screening of high-risk groups particularly overweight and obese subjects. It is important that factors inducing obesity should be addressed in any coordinated strategy to tackle the problem of obesity and associated co-morbidities.

Table II. Descriptive and comparative data for serum lipids and lipoproteins of the studied sample.

Dependent Variable	BMI-C	No	Mean	ï SD	BMI-C		Mean Difference	ï Std. Error	P
Age/years	0	103	38.60	6.31	0	1	-0.23	0.91	0.969
	1	100	38.83	6.63	2	0	0.90	0.90	0.612
	2	103	39.49	6.50	2	1	0.67	0.91	0.764
TRIG	0	103	126.41	59.01	0	1	-52.61	11.40	0.000
	1	100	179.02	84.81	2	0	84.24	11.32	0.000
	2	103	210.65	95.58	2	1	31.63	11.40	0.022
TC	0	103	179.80	29.75	0	1	-33.71	5.71	0.000
	1	100	213.51	45.02	2	0	52.50	5.66	0.000
	2	103	232.29	45.36	2	1	18.78	5.71	0.005
HDL-C	0	103	47.27	9.65	0	1	3.97	1.31	0.011
	1	100	43.30	8.81	2	0	-7.13	1.30	0.000
	2	103	40.14	9.53	2	1	-3.16	1.31	0.056
LDL-C	0	103	107.09	29.39	0	1	-27.31	5.13	0.000
	1	100	134.41	39.84	2	0	42.82	5.09	0.000
	2	103	149.91	39.46	2	1	15.50	5.13	0.011
TC/HDL-C	0	103	3.96	1.08	0	1	-1.17	0.20	0.000
	1	100	5.13	1.51	2	0	2.08	0.20	0.000
	2	103	6.05	1.71	2	1	0.92	0.20	0.000

Results of one-way ANOVA and Scheffe post hoc analysi (0) Non-obese, (1) overweight, (2) obese

Table III. Prevalence rates and ratios proportions of subject with adverse lipids and lipoproteins among body mass index categories in the studied sample.

Indicators	Body mass index categories (BMI-C)							
	Non -obese		Over weight		R1	Obese		R2
	N.	%	N.	%		N.	%	
	103	33.7	100	32.6		103	33.7	
TRIG -1	5	4.9	30	30.0	6.2	52	50.5	10.4
TC-1	5	4.9	43	43.0	8.9	53	51.5	10.6
HDL-C -1	7	6.8	14	14.0	2.1	34	33.0	4.9
LDLC- 1	7	6.8	25	25.0	3.7	35	34.0	5.0
TC/HDL-C-1	6	5.8	25	25.0	4.3	49	47.6	8.2

*All (R): Calculated with each category by dividing the proportions of adverse serum lipids and lipoproteins over- weight or obese/ non-obese proportion.
*All ratios of proportions were significantly at P<0.5 based on Chi-square statistic.

Table IV. Adjusted odds ratio (ORs) of elevated serum triglycerides (TRIG -1) of obesity categories after controlling for age, weight history, CVD, and hypertension among parents current smoking, educational level, monthly income, occupation, and percentage of calorie from dietary fat(%CDF) in the study sample.

95% CI for Exp (B)				
Variable	**Exp (B)**	**Lower**	**Upper**	**P**
Body mass index categories (BMI-C)				
Parental (CVD)	2.11	1.16	3.82	0.0145
Childhood obesity	0.29	1.10	1.88	0.0 283
Adolescence obesity	1.82	0.76	4.36	0.0401
%CDF-C (3)	2.71	1.20	6.12	0.0168
Overweight	5.08	1.79	14.43	0.0023

*Non significant variables are not shown in the table.

Table V. Adjusted odds ratio (ORs) of elevated serum cholesterol (Chol-c (1)) of obesity categories after controlling for age, weight history, parental CVD ,current smoking educational level, monthly income, occupation,and percentage of calorie from dietary fat in the study sample.

95% CI for Exp. (B)				
Variable	**Exp. (B)**	**Lower**	**Upper**	**P**
Body mass index categories (BMI-C)				
Childhood obesity	2.00	0.90	4.40	0.0870
%CDF-C(3)	1.89	0.92	3.89	0.0823
Overweight	12.65	4.62	34.59	0.0000
Obese	14.03	5.03	39.10	0.0000

*Non significant variables are not shown in the table.

Table VI. Adjusted odds ratio (ORs) of low serum high-density lipoprotein cholesterol (HDL-C) of obesity categories after controlling for age, weight history, parenteral CVD, current smoking, educational level, monthly income, occupation, and percentage of calorie from dietary fat in the study sample

95% CI for Exp(B)				
Variable	**Exp(B)**	**Lower**	**Upper**	**P**
Body mass index categories (BMI-C)				
Parental (CVD)	1.82	0.98	3.37	0.0743
Overweight	2.21	0.85	5.76	0.1031
Obese	6.92	2.88	16.60	0.0000

*Non significant variables are not shown in the table.

Table VII. Adjusted odds ratios (ORs) of high serum low-density lipoprotein cholesterol (LDL-C) of obesity categories after controlling for age, weight history, and parental CVD ,current smoking, educational level, monthly income, occupation, and percentage of calorie from dietary fat in the study sample

	95% CI for Exp (B)			
Variable	**Exp (B)**	**Lower**	**Upper**	**P**
Body mass index categories (BMI-C)				
Age-C(1)	3.08	1.65	5.75	0.0004
Childhood obesity	5.70	2.08	15.66	0.0007
Adolescence obesity	0.44	0.17	1.12	0.0852
%CDF-C(2)	3.22	1.31	7.92	0.0107
Overweight	5.14	1.91	13.83	0.0012
Obese	5.52	2.01	15.17	0.0009

*Non significant variables are not shown in the table.

Table VIII. Adjusted odds ratio (ORs) of (TC/HDL-C-1) of obesity categories after controlling for age, weight history, parental CVD, smoking, physical activity, educational level, monthly income, occupation and percentage of calorie from dietary fat in the study sample.

	95% CI for Exp (B)			
Variable	**Exp (B)**	**Lower**	**Upper**	**P**
Body mass index categories (BMI-C)				
Age-C (1)	1.91	1.06	3.46	0.0325
Parental(CVD)	1.71	0.94	3.09	0.0779
Smoking	2.01	1.12	3.62	0.0191
%CDF-C (2)	2.64	1.13	6.14	0.0245
Overweight	4.40	1.63	11.88	0.0034
Obese	10.80	4.06	28.69	0.0034

*Non significant variables are not shown in the table.

References

1. **World Health Organization.** Report of a WHO consultation on obesity. Preventing and managing the Global Epidemic1997; Geneva, WHO.
2. **National Institute of Health (NIH).** Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults-the evidence report. *Obes Res* 1998; 6: 51-210.
3. **Allison DB, Saunder SE.** Obesity in North America, An overview. *Medical Clinics of North America* 2000; 84: 305-328.
4. **Sheehan, MT, Jensen MD.** Metabolic complications of obesity pathophsiologic considerations. *Medical Clinic of North America* 2000; 84: 363-384.
5. **Kissebah, AH, Krakower GR, Sonneberg GE, Hennes MMI.** Clinical manifestations of the metabolic syndrome. In: Bray GA, Bouchard C, James WPT. Eds. Handbook of Obesity. New York. Marcel Dekker, 1998; 601-633.
6. **Kopelman PG.** Obesity as a medical problem. *Nature* 2000; 404: 633-645.
7. **Baik I, Ascherio A, Rimm EB, *et al*.** Adiposity and mortality in men. *Am J Epidemiol* 2000; 158(3): 264-270.
8. **Reaven GM.** Pathophysiology of insulin resistance in human disease. *Physiological Reviews* 1995; 75: 473-485.
9. **Jung TR.** Obesity as a disease. *British Medical Bulletin* 1997; 53: 307-321.
10. **Haffner SM, Stern MP, Hazuda HP, *et al*.** The role of behavioral variables and fat patterning in explaining ethnic differences in serum lipids and lipoprotein. *Am J Epidemiol* 1986; 123: 830-839.
11. منظمة الاغذية والزراعة العالمية- المكتب الاقليمي للشرق الادنى- القاهرةز مركز البحرين للدراسات **والبحو. الوضع التغذوي والرقابة** الغذائية في مجلس التعاون لدول الخليج العربية، 1998 ، 1-61.
12. **Rimm EB, Stampfer MG, Giovannuci E, *et al*.** Body size and fat distribuation of coronary heart disease among middle-aged and older US men. *Am J Epidemiol* 1995; 141(12): 1117-1126.
13. **Denke MA, Sempos CT, Grundy SM.** Excess body weight, An underrecognized contributor to high blood cholesterol levels in white American men. *Arch Intern Med* 1994; 154: 401-410.
14. **Must A, Spadano J, Coakley EH, *et al*.** The disease burden associated with overweight and obesity. *JAMA Middle East* 2000; 10: 63-70.
15. **Hu FB, Wang B, Chen C, *et al*.** Body mass index and cardiovascular risk factors in a rural Chinese population. *Am J. Epidemiol* 2000; 51: 88-97.
16. **Walton C, Lees B, Crook D, *et al*.** Body fat distribution, rather than genderalized adiposity influence serum lipids and lipoproteins in healthy men independent of age. *Am J Med* 1995; 99: 459-464.
17. **Lemieux I, Pascot A, Couillard C, *et al*.** Hypertriglyceridemic waist. A marker of the atherogenic metabolic traid (hyperinsulinemia, hyperapolipoprotein B; small, dense LDL) in men. *Circulation* 2000; 102: 179-184.
18. **Han TS, Van Leer EM, Seidell JC, Lean ME.** Waist circumference action levels in the identification of cardiovascular risk factors, prevalence study in a random sample. *BMJ* 1995; 311: 1401-1405.

19. **Mansfield E, Mcpherson R, Koski KG.** Diet and waist to hip ratio, important predictors of lipoprotein levels in sedentary and active young men with no evidence of cardiovascular disease. *J Am Diet Assoc* 1999; 99: 1373-1379.
20. **Maffeis C.** Childhood obesity: The genetic-environmental interface. *Balliere's Clinical Endocrinology and Metabolism* 1999; 13(1): 31-46.
21. **Must A, Jacques PF, Dallai GE, *et al.*** Long term morbidity and mortality of overweight adolescents, a follow-up of the Harvard Growth Study of 1922-1935. *N Engl J Med 1992;* 327(19): 1350-1355.
22. **Fung TT, Hu FB, Yu J, *et al.*** Association between dietary patterns and serum biomarkers of obesity and cardiovascular disease risk. *Am J. Clin Nutr* 2001; 73: 61-67.
23. **Ellison RC, Myers RH, Zhang Y, *et al.*** Effects of similarities in life style habits on familial aggregation of high density lipoprotein and low density lipoprotein cholesterol. *Am J Epidemiol* 1999; 150(9): 910-918.
24. **Fung TT, Hu FB, Yu J, *et al.*** Leisure- time physical activity, television watching, and serum biomarkers of obesity ad cardiovascular disease risk. *Am Journal Epidemiol* 2000; 152(12): 1171-1178.
25. **Jaddou H, Batieha A, Ajlouni K.** Prevalence and associated factors of hypertension results from a three community based survey- Jordan. *J Human Hypertens* 1996; 10: 815-825.
26. **Ajlouni K, Jaddou H, Batieha A.** Diabetes and impaired glucose tolerance in Jordan: prevalence and associated risk factors. *Journal of Internal Medicine* 1998; 243: 1-6.
27. **Batieha A, Jaddou H, Ajlouni K.** hyperlipidemia in Jordan: A community based survey. *Saudi Med J* 1998; 18(3): 279-285.
28. **Ammari F, Batieha A, Jaddou H, *et al.*** A natural history of impaired glucose tolerance in North Jordan. *Practical Diabetes Int* 1998; 15: 139-140.
29. **Annual Statistical Report.** Royal Medical services (RMS) 2000- Jordan.
30. **Pellet PL, Shadarevian S.** Food composition tables for use in the Middle East, 2nd. American University of Beirut, Lebanon. 1970
31. **Lohman TG, Roche AF, Martorel RI.** Anthropometric Standardization Reference Manual (1st ed.) Champaign: III: Human Kinetics Books 1988; 1-5.