5 (22.7%) males, their ages ranged from 16-52 years, (mean 27.86%±10.74). Their plasma MADDS and DDS ratios were from 0.01-0.28 µg/ml (mean 0.11±0.088). While the frequency of rapid acetylators in control whose MADDS and DDS ratios were >0.30 were 8 (26.7%) of 30. They included 4 (50.0%) females and 4 (50%) males. Their ages ranged from 16-29 years, (mean 22.00±4.87). Their plasma MADDS and DDS ratios were from 0.36-1.63 µg/ml (mean 0.86±0.57). There were no non-acetylators in healthy controls (**Table 1**). The slow acetylators in the DLE patients, as a group, was not significantly different from the slow acetylators in the control group ($p>0.05$).

In this study, patients with DLE were shown to be either slow acetylators or non-acetylators, that is the plasma concentration of MADDS was undetected in plasma. In this respect, the results of this study differs from the 2 previous reports from the European countries which concluded that the frequency of slow acetylators was not different between DLE patients and normal control group.[4,5] In a parallel study, the incidence of slow acetylators in patients with spontaneous SLE was reported not different from that found in the control group.[3] Taken together, the 2 studies about the acetylator phenotype in SLE and DLE support the held view that the 2 diseases do not represent a spectrum but can be considered to be overlapping diseases with different etiologies.[1] An interesting finding in this study was that some patients were non-acetylator. In a previous report about half of the patients with Behcets' disease were found to be non-acetylators.[6] This finding can not be explained by a technical error in the method used since non-acetylator in this study as well as the previous were found in the patients and not in the control group. On both occasions patient and control samples were run together. The significance of this finding, needs further investigation to determine the genotype of non-acetylators, in order to understand this phenomena.

In conclusion, in a population of slow acetylators, it appears that slow or very slow acetylators phenotype can be considered as genetic trait predisposing to the development of DLE. This finding as well as the occurrence of non-acetylators needs further investigation to determine the genotype in a lager sample.

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Risk factors of coronary heart disease among Jordanians

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Coronary heart disease (CHD) is the first leading cause of death in developed countries. According to Ministries of health statistics, it was also found to have a significant prevalence in Jordan and the rest of the Arabic countries.[1] Prevention is of primary importance, and proper prevention requires correction of risk factors in persons at high risk. This will significantly lower the mortality rate, which reduces the economic burden resulting from stroke on patients' families and health service organizations. Numerous surveys and epidemiological studies revealed the major risk of coronary heart disease (CHD), which included diabetes, low-density lipoprotein-cholesterol (LDL-C), hypertension (HTN), smoking, inflammatory diseases and others, which could be managed effectively.[2] However, the relative significance of these factors remains controversial among many publications.[3] Nevertheless, it was well established that some environmental, genetic and ethnic factors played a significant role in these controversial conclusions.

We have conducted this study, which retrospectively involved a total of 201 patients who underwent catheterization at the Islamic and Ibn-Al-Hytham Hospitals, Amman, Jordan, in the period between 1995 and 2000. Experienced

cardiologists were consulted to determine inclusion criteria of patients. A special data form was designed. Thereafter, baseline demographic criteria (including height, weight, and body mass index), blood pressure and family history were collected with a common protocol details of which are described in another publication.[4] Monitoring parameters during follow-up included lipid profile (LDL-C, triglyceride and high-density lipoprotein-cholesterol [HDL-C]), as well as fasting blood sugar. Measurements of each lipid profile item were further stratified into various risk categories that have been defined by the National Cholesterol Education Program (NCEP) Expert Panel for detection, evaluation and treatment of high blood cholesterol in adults. To determine the significant predictors of CHD in this population, 2 multiple logistic regression models were fitted for lipid and nonlipid variables separately. In the first model, the independent effects of different age groups, gender, diabetes, diabetic types, hypertension, congestive heart failure, previous history of myocardial infarction (MI), smoking status, family history, and documented diagnosis of hyperlipidemia were tested. However, all lipid parameters were included for evaluation in the second model. **Table 1** and **Table 2** displays the adjusted odds ratios (OR) of CHD with their 95% confidence intervals, which were obtained for the nonlipid (Model l) and lipid variables (Model 2). As shown, male gender was the most significant nonlipid variable with an estimated risk for CHD of 16.7 times higher than female gender. In particular, males of age greater than 45 years had 92 times higher risk than males of age less than 45 years. In addition, other variables including hypertension, family history of any type (family history of hypertension, diabetes, or more than one type), diabetics type II, and history of previous MI were also significantly associated with CHD. An interesting finding is the impact of smoking cessation on modifying the cardiovascular morbidity with an OR=0.058. While the variables; diabetes mellitus, congestive heart failure (CHF) and hyperlipidemia were dropped out of the final model when forward and backward stepwise regression methods were applied due to their insignificance. In the second analytical model, LDL ≥220 (mg/dl) level and total cholesterol ≥200 (mg/dl) level were the only lipid parameters, which remained in the backward regression model with OR=3.75 and 10 times. Although HDL level >35 mg/dl was inversely associated with CHD (OR=0.38), the relationship did not reach the statistical significance (*p*=0.09). However, the LDL and HDL ratio had a positive trend for prediction of CHD, which has started at the "gray zone" level (ratio between 4 and 5). Patients with LDL is to HDL ratio of >5 were 20

Table 1 - Adjusted odds ratio of prevalence of coronary heart disease by nonlipid variables.

Variables (mg/dl)	Odds ratio (95% confidence interval)		*p* values
Gender	16.7	(3.4 - 82.17)	0.0005
Male	0.02	(0.007 - 0.08)	0.00
Female			
Age (years)			0.0045
30-39	0.0349	(0.0008 - 1.6)	0.08
40-49 versus >60	92	(8 - 1.57)	0.0003
Male >45	1.99	(0.9 - 261.6)	0.11
Diabetes mellitus	16.05		0.05
Diabetic type II	2.68		0.02
Hypertension	0.54		0.27
Congestive heart failure	0.38		0.015
Family history	59.9	(1.7 - 2082)	0.023
HTN			0.009
Diabetes mellitus			0.02
More than one	1.7	(0.02 - 3.1)	0.2
Hyperlipidemia	1.12		0.78
Current smokers	0.058	(0.0023 - 1.5)	0.09
Ex-smokers	28.8	(2.4 - 343.5)	0.007
History of myocardial			

HTN - hypertension disease

Table 2 - Adjusted odds ratio of prevalence of coronary heart disease by various lipid parameters.

Variables	Odds ratio (95% confidence interval)		p values
Low-density lipoprotein			
Very high risk (190-219)	3.75	(0.4 - 35.1)	0.037
Extremely high ≥220	753748	(0 - 1.87)	0.04
Total cholesterol			
Borderline-high risk (200-239)	64.4 10	(2 - 2.44)	0.018
High risk (≥240)		(0.7 - 138.8)	0.08
High-density lipoprotein			
Desirable >35	0.38	(0.125 - 1.19)	0.09
LDL:HDL ratio			
Gray zone "4-5			0.07
High risk >5	20.8	(2 - 1017)	0.12
Triglyceride			
Borderline-high risk (200-239)	0.72	(0.04 - 12)	0.3
High risk ≥240	0.11	(0.005 - 2.5)	0.8

times more likely to develop CHD than patients with ratio of <5. For triglyceride levels, none of their risk categories were independently associated with CHD incidence. The total cholesterol and HDL ratio was also of minor significance compared to other lipid parameters and therefore it was removed with the triglyceride variable from the final model.

Overall, this cross-sectional survey has revealed the high prevalence rate of CHD among the Jordanian society referred to cardiac catheterization units at 2 major medical centers in Amman in which medical services are provided for a broad range of public and private sectors. Although the prevalence of dyslipidemia of various types showed greater positive trends in patients with CHD (N=145) than non-diseased patients (N=56). The overall distributions of various risk categories of total cholesterol, triglyceride, LDL-C, HDL-C, triglyceride/HDL-C, and LDL-C/HDL-C were not significantly different between the 2 groups, probably due to the smaller number of non-diseased patients.

Nevertheless, it was concluded that hyperlipidemia of any type was not the only crucial determinant of vascular stenosis in all admitted cases of CHD. Therefore, it was recommended that further large-scale, prospective surveys should be carried out to define other "nonclassical" variables, which were recently in question as predictors of CHD, such as infectious agents, markers of inflammation, homocysteine and Lp(a).[5] However, conflict go on regarding their exact role in pathogenesis of atherosclerosis.

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