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Demographic and Blood Lipid Profiles in Correlation with Heart Attacks among Mediterraneans

Khaled Qabaha[1]*, Wael Abu Hassan[1], Haneen Mansour[1], Saisathya Thanigachalam[2] and Saleh Naser[2]

[1]*Arab American University, Jenin, Palestine*
[2]*Burnett School of Biomedical Sciences, College of Medicine, University of Central Florida, Orlando, Florida, USA*

Abstract

Aim: To evaluate the role of some demographic and plasma lipid variables in heart attack occurrence among Palestinians. Demographic variables under study include age, sex, smoking, sporting (walking, running, basketball, etc.) hypertension and diabetes mellitus. Cholesterol, triglyceride, LDL and HDL are the blood lipid variables under study.

Materials and Methods: Descriptive analytical and correlation design were set as a frame for data collection. Data were collected from 186 subjects, representing both healthy and unhealthy heart groups. Ninety six were free from heart attacks while 89 were diagnosed as heart attack patients. Whole blood samples were collected in EDTA tubes. Plasma samples were used to measure total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides using the commercial Human Liquicolor Kits. Demographic information's about subjects were collected from either subjects themselves or their attendant relatives in their respective hospitals.

Results: The association between demographic and lipid profiles with occurrence of heart attack was demonstrated as statistically significant. Sporting and HDL factors negatively correlated with heart attack incidences. Moreover, statistical differences between the study groups were demonstrated in all study variables, except age and sporting.

Conclusion: Irrespective of geographical or cultural factors, most of the studied demographic as well blood lipid profiles were significantly contributing to the occurrence of cardio problems as risk factors.

Keywords: Demographic; Blood lipids; Myocardial infarction; Cardiovascular

Introduction

Cardiovascular diseases, mainly myocardial infarction (MI) , are the main cause of death worldwide. It is expected to rise to 25 million deaths in 2020 [1-3]. MI is a type of coagulative necrosis in which blood supply to part of the cardiac tissue is partially or totally blocked. Blood lipid profiles such as total cholesterol (T Chol), low density lipoprotein (LDL), very low density lipoprotein (VLDL), high density lipoprotein (HDL), and triglyceride (TG) are demonstrated as significant factors that influence the occurrence of myocardial infarction [4].

High-density lipoprotein-Cholesterol (HDL-C) also called as the good cholesterol. It is the smallest lipoprotein, which transports lipid (cholesterol and triglycerides) that are deposited on the walls of the arteries back to the liver for excretion. It is done through two pathways:- direct and indirect. The most common pathway is the indirect pathway which involves cholesterol ester transfer protein (CETP), through which the cholesterol concentration in blood is decreased [5].

Low density lipoprotein- Cholesterol (LDL-C) also called as the bad cholesterol. LDL comprises of major portion of cholesterol. It includes triglyceride precursor for its particles, which enter the circulation as LDL, deliver cholesterol to tissues, and keep circulating fatty acids soluble in the aqueous environment. LDL can slowly build up in the inner walls of the arteries such as the coronary arteries, forming an intimal plaque, a thick, hard deposition known as atherosclerosis along with other substrates [5,6].

Studies have concluded that any increase in HDL level was associated with a decrease in heart attacks, while an increase in LDL level was associated with an increase in heart attacks [7]. Maruyama et al have found that low HDL cholesterol, high total cholesterol, high LDL cholesterol and high triglycerides have a positive relation in increasing the risk of myocardial infarction [4,8,]. There is no evidence so far that the specific increase of serum HDL cholesterol results in less cardiovascular diseases [9]. Besides some studies demonstrated that HDL cholesterol is a stronger risk factor for coronary artery disease than LDL cholesterol [10].

Many risk factors were found to be significantly associated with Myocardial Infarction including gender (MI occurs in men more than women), age (MI occurs most frequently in persons older than 45 years), family history, diabetes mellitus (DM), smoking, alcohol use, hyperlipidemia, physical activity and hypertension [1,11,12].

Atherosclerosis leads to malfunction of blood vessels in various anatomical locations but specifically the coronary arteries that lead to heart attack. The patients with heart attack usually suffer from sudden chest pain, shortness of breath and sweating due to obstruction of a major coronary artery ending up with partial ischemic necrosis of the heart muscle or sudden death [13].

High total and low HDL-cholesterol and high LDL-cholesterol are well known risk factors among western people [4], but, to the best of the authors' knowledge, no study has been done to investigate such relationship among Palestenians who have different life style and diet as well. The aim of this cross-sectional Palestinian population-based study was to investigate the role of high total and low HDL-cholesterol and high LDL-cholesterol as well as some demographic variables in heart attack occurrence among Palestinians. Demographic variables under

***Corresponding author:** Khaled I Qabaha, Arab American University, Jenin, Palestine, Tel: 05-99- 325358; E-mail: khaledqabaha@yahoo.com

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study include age, sex, smoking, sporting, hypertension and diabetes mellitus.

Materials and Methods

The descriptive analytical as well as the correlation design was used as a frame for data collection.

Blood samples were collected in Ethylenediaminetetraacetic acid (EDTA) tubes from one hundred and eighty five subjects (M=114/F=72). Ninety six of them were clinically diagnosed as normal subjects without history of heart attacks, while eighty nine were suffering from heart attacks and were treated in government hospitals of Jenin, Alwatani hospital- Nablus, and Ramallah government Hospital in West Bank, Palestine.

An Institutional Review Board (IBR) of the Ministry of Health in Palestine and its related directories was taken in consideration, where patients as human subjects were assured the right to participate in the study or leave the time they want. Also welfare of participants and confidentiality was addressed and all subjects have signed an informed consent to participate in the study.

Serum Samples were collected from all subjects after 10 hours of fasting to measure the concentrations of total Cholesterol, Triglycerides, Low Density Lipoprotein – Cholesterol (LDL-C) and LDL using a spectrophotometer (humalyzer junior), and analyzed using the commercial Human Liquicolor Kit.

Total cholesterol was measured enzymatically using CHOD-PAP-Method in which cholesterol is formed by hydrolysis of cholesterol esters and the formed cholesterol is oxidized with cholesterol oxides to form hydrogen peroxide which forms the indicator quinoneimine with 4-aminophenazone in the presence of phenol and peroxidase death [13].

Triglycerides were measured enzymatically using a GPO-PAP-Method in which lipases hydrolyze triglycerides to obtain glycerol which is oxidized by glycerol kinase and glycerol peroxidase to form hydrogen peroxide that forms the indicator quinoneimine with 4-aminoantipyrine and 4-chlorophenol in the presence of peroxidase [14]. LDL-C was measured according to Friedewald equation in samples with Triglyceride concentration less than 400 mg/dl [15].

HDL-cholesterol was measured using cholesterol liquicolor test kit in which the other types of cholesterol are precipitated by phosphotungstic acid and magnesium chloride. HDL-C was then measured in the supernatant fluid [13].

For the purpose of statistical analysis of the collected data, descriptive as well inferential statistics were utilized. Frequencies, means, and standard deviations were obtained and accordingly t-test and F ratios were calculated. Bivariate and partial correlations were also determined.

Results and Discussions

As shown in Tables 1 and 2, preliminary statistical analyses were

	Time of Heart Attack Occ	N	Mean	Std Deviation	Std Error Mean
Age	0	96	47.79	10.926	1.115
	1	89	56.83	12.277	1.301
Sex	0	96	1.32	0.47	0.048
	1	89	1.45	0.5	0.053
Smoker	0	96	0.32	0.47	0.048
	1	89	0.44	0.499	0.053
Sport	0	96	0.1	0.307	0.031
	1	89	0.07	0.252	0.027
Hypertension	0	96	0.17	0.375	0.038
	1	89	0.56	0.499	0.053
DM	0	96	0.07	0.261	0.027
	1	89	0.44	0.499	0.053
Cholesterol	0	96	190.73	31.181	3.182
	1	89	200.98	41.393	4.388
TG	0	96	122.53	71.741	7.322
	1	89	142.72	88.715	9.404
LDL	0	96	134.9	28.048	2.863
	1	89	146.09	38.696	4.102
HDL	0	96	34.76	9.8	1
	1	89	29.15	10.387	1.101

Table 1: Distribution of healthy/unhealthy heart subjects with reference to their demographic and blood lipid profiles.

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Age	Equal variances assumed	2.362	.126	-5.298-	183	.000	-9.040-	1.706	-12.406-	-5.673-
	Equal variances not assumed			-5.275-	176.520	.000	-9.040-	1.714	-12.422-	-5.658-
Sex	Equal variances assumed	9.813	.002	-1.774-	183	.078	-.127-	.071	-.267-	.014
	Equal variances not assumed			-1.769-	179.572	.079	-.127-	.072	-.268-	.015
Smoker	Equal variances assumed	8.640	.004	-1.618-	183	.107	-.115-	.071	-.256-	.025
	Equal variances not assumed			-1.614-	179.694	.108	-.115-	.071	-.256-	.026
Sport	Equal variances assumed	3.197	.075	.886	183	.377	.037	.041	-.045-	.119
	Equal variances not assumed			.892	180.415	.373	.037	.041	-.045-	.118
Hypertension	Equal variances assumed	62.161	.000	-6.119-	183	.000	-.395-	.065	-.523-	-.268-
	Equal variances not assumed			-6.054-	162.820	.000	-.395-	.065	-.524-	-.266-
D.M	Equal variances assumed	212.670	.000	-6.302-	183	.000	-.365-	.058	-.480-	-.251-
	Equal variances not assumed			-6.166-	130.632	.000	-.365-	.059	-.482-	-.248-

Table 2: Distribution of Levene's and t-tests for testing equality of variance and means between studied groups with reference to age, sex, smoking, hypertension and diabetes mellitus variables.

performed to test for potential differences between the healthy control (n=96) and unhealthy (n=89) groups. We find out that there were statistically significant differences when comparing the means of healthy controls with the unhealthy groups, especially, in the case of age, hypertension, and Diabetes Mellitus (DM), where t (n=183) found to be as equal to -5.298/ p = .000; -6.119/ p = 000; -6.302/ p = .000 respectively. Such results are consistent with the findings of Patel et al. [16].

In regard of attributing results to smoking and sporting (walking, running, basketball, etc.) the authors found no statistical significance among the two studied groups (α=0.05). The finding is contradictory with Varki et al. [13]. They reported that smoking and sedentary lifestyles were among the risk factors of atherosclerosis, a common cause of heart attack. The reason behind such contradiction could be due to widespread smoking habit among Palestinians, especially those who were among the clinically normal subjects in this research. Referring to sporting, it is a well known fact for the layman that sporting is not culturally present practice among Palestinians.

To check the level of variances among groups, Leven's test for equality of variances (denoted as F) was utilized. We find out that the set hypothesis which assume the absence of any "statistical significant differences" among groups as rejected in the case of sex, smoking, hypertension and diabetes mellitus (F=9.813/ p=0.002; 8.640/ p=0.004; 62.161/ p=0.000; and 212.670/ p=0.000 respectively). Such results go along with the findings of Wei et al, who concluded that diabetes mellitus, hypertension and smoking were important predictors in cardiovascular deaths [17]. Regarding sex, the authors have found that heart attacks were more among men than in women which were consistent with the findings of Canto et al., who have reported that women on average were about a decade older than men at their initial heart attacks [18].

The above hypothesis was accepted in the case of age and sporting (F=2.363/ p=0.126; and 3.197/ p=0.075; respectively), contradicting the findings of Shaper et al, they found a strong inverse relation between the risk of heart attack and physical activity without pre-existing ischemic heart disease [19].

Pooling all the data together irrespective of healthy and unhealthy heart subjects, bivariate correlations cross variables were calculated for much better understanding, as well to know the extent of influence those variables contribute to the occurrence of heart attacks (Table 3).

		Age	Sex	Smoker	Sport	Hypertension	D.M	Time of Heart Attack Occ.,	Cholesterol	TG	LDL	HDL
Age	Pearson Correlation	1	0.13	0.104	-0.076-	0.282**	0.338**	0.365**	0.170*	-0.017-	0.163*	-0.026-
	Sig. (2-tailed)		0.077	0.16	0.301	0	0	0	0.021	0.819	0.026	0.73
	N	185	185	185	185	185	185	185	185	185	185	185
Sex	Pearson Correlation	0.13	1	-0.387-**	-0.085-	0.224**	0.266**	0.13	0.061	0.068	-0.029-	0.214**
	Sig. (2-tailed)	0.077		0	0.252	0.002	0	0.078	0.407	0.357	0.7	0.003
	N	185	185	185	185	185	185	185	185	185	185	185
Smoker	Pearson Correlation	0.104	-0.387-**	1	-0.002-	-0.046-	-0.088-	0.119	0.085	-0.011-	0.159*	-0.160-*
	Sig. (2-tailed)	0.16	0		0.977	0.535	0.235	0.107	0.252	0.885	0.031	0.03
	N	185	185	185	185	185	185	185	185	185	185	185
Sport	Pearson Correlation	-0.076-	-0.085-	-0.002-	1	-0.149-*	-0.088-	-0.065-	-0.157-*	0.041	-0.121-	-0.122-
	Sig. (2-tailed)	0.301	0.252	0.977		0.043	0.233	0.377	0.033	0.583	0.102	0.099
	N	185	185	185	185	185	185	185	185	185	185	185
Hypertension	Pearson Correlation	0.282**	0.224**	-0.046-	-0.149-*	1	0.511**	0.412**	0.115	0.127	0.12	-0.087-
	Sig. (2-tailed)	0	0.002	0.535	0.043		0	0	0.119	0.084	0.103	0.241
	N	185	185	185	185	185	185	185	185	185	185	185
D.M	Pearson Correlation	0.338**	0.266**	-0.088-	-0.088-	0.511**	1	0.422**	0.078	0.104	0.083	-0.108-
	Sig. (2-tailed)	0	0	0.235	0.233	0		0	0.289	0.158	0.263	0.144
	N	185	185	185	185	185	185	185	185	185	185	185
Time of Heart Attack Occ.,	Pearson Correlation	0.365**	0.13	0.119	-0.065-	0.412**	0.422**	1	0.14	0.125	0.165*	-0.269-**
	Sig. (2-tailed)	0	0.078	0.107	0.377	0	0		0.058	0.09	0.025	0
	N	185	185	185	185	185	185	185	185	185	185	185
Cholesterol	Pearson Correlation	0.170*	0.061	0.085	-0.157-*	0.115	0.078	0.14	1	0.300**	0.930**	0.129
	Sig. (2-tailed)	0.021	0.407	0.252	0.033	0.119	0.289	0.058		0	0	0.08
	N	185	185	185	185	185	185	185	185	185	185	185
TG	Pearson Correlation	-0.017-	0.068	-0.011-	0.041	0.127	0.104	0.125	0.300**	1	0.185*	-0.259-**
	Sig. (2-tailed)	0.819	0.357	0.885	0.583	0.084	0.158	0.09	0		0.012	0
	N	185	185	185	185	185	185	185	185	185	185	185
LDL	Pearson Correlation	0.163*	-0.029-	0.159*	-0.121-	0.12	0.083	0.165*	0.930**	0.185*	1	-0.052-
	Sig. (2-tailed)	0.026	0.7	0.031	0.102	0.103	0.263	0.025	0	0.012		0.485
	N	185	185	185	185	185	185	185	185	185	185	185
HDL	Pearson Correlation	-0.026-	0.214**	-0.160-*	-0.122-	-0.087-	-0.108-	-0.269-**	0.129	-0.259-**	-0.052-	1
	Sig. (2-tailed)	0.73	0.003	0.03	0.099	0.241	0.144	0	0.08	0	0.485	
	N	185	185	185	185	185	185	185	185	185	185	185

Table 3: Matrix of bivariate correlations of investigated demographic and blood lipid profiles.

As shown in Table 3 (considering 0.01 and 0.05 as levels of significance), it is very clear that age, hypertension, DM and LDL has significant contribution to the occurrence of heart attack. Correlation coefficient findings (r=0.365; 0.412; 0.422; and 0.165 respectively) reveal higher contribution of these variables to heart attack. More interestingly, sporting and HDL respectively were found to be negatively correlated with the same (r= -0.065,-0.269). Such a result is consistent with Robin P.F. Dullaart and Patel et al. reports although, sporting contradicts the findings [20].

To further understand the findings of the study, matrix of correlation was calculated that clarified that there are other variables which significantly inter-correlate with age, hypertension, DM and LDL (Table 3); an outcome which significantly indicates their contribution to those variables which were already found to be correlated with occurrence of myocardial infarction.

To determine the contribution of such variables to the occurrence of heart attacks, and the size of that contribution, we carried out partial correlation analysis. The age variable was found to be significantly correlated with the occurrence of heart attacks. Other variables including sex, smoking, sporting, hypertension and DM. were found to contribute significantly to age as well as to the occurrence of heart attacks (Table 4). The calculated partial correlation value was found to be as ($r_{12.5}$=0.211). Such a value, as being found less than the determined correlation size between age and heart attack occurrence (i.e., 0.365), statistically indicates the contribution of the tested control variables to both age and heart attack.

Similar findings were reported by Wald et al. [21] who found that age was a strong determinant of cardiovascular diseases and also includes age-related risks. High blood pressure (BP) (>140/90 mm Hg) and diabetes mellitus are strong predictors of cardiovascular diseases, such results are consistent with De Marco et al findings [22].

The authors have found that smoking was a major risk factor for heart attacks as shown by Table 3. Such finding is consistent with Leif Erhardt 2009 and Haffey reports, who also found that cessation of smoking reduces the mortality from coronary heart disease [23,24].

Table 5 summarizes data which suggest cholesterol, TG, LDL, and HDL were found as contributing to age and occurrence of heart attacks. Such results are consistent with Boullart et al., where they have reported that high levels of LDL and low levels of HDL are main attributers to cardiovascular diseases. Also, Boullart et al. have supported the above findings in regard of TG and total cholesterol and their contribution to cardiovascular disease [25].

Over all, on a bivariate statistical basis as well partial correlation basis, a matrix scatter plotting for all studied variables set by the occurrence of heart attacks as a dependant variable was carried out to draw a clear-cut pictorial picture for all correlations. Figures 1-3 show those correlations.

Conclusion

From the findings of our research, we may conclude that most of the investigated demographic variables, statistically do contribute to the occurrence of heart attacks among the studied diseased subjects. It becomes that much clear that aged people, hypertensive, and DM patients are significantly suffer from being as victims to heart attacks and the frequency of MI occurrence and reoccurence .

Also, within the same range of variables, including sex, smoking, and sporting, all found to be in partial correlation with growing age and the probability of heart attack occurrence. When it comes to blood lipids and related profiles, it was found that they are playing their role within age and the ultimatum inevitable occurrence of MI.

Limitation of Study

The present research work as based on cross-sectional procedures, where the «time effect» is ignored. A quasi-experimental simple cohort design if was carried out could help more to control history effects; but we find it that much difficult to apply such a research procedure for the sensitivity of patients and their critical health situations, particularly in the diseased group.

Control Variables			Age	Time of Heart Attack Occ.,
Sex & Smoker & Sport & Hypertension & DM	Age	Correlation	1.000	0.211
		Significance (2-tailed)	.	0.004
		df	0	178
	Time of Heart Attack Occ.,	Correlation	0.211	1.000
		Significance (2-tailed)	0.004	.
		df	178	0

Table 4: Partial correlation with reference to sex, smoking, sporting, hypertension and diabetic mellitus variables.

Control Variables			Age	Time of Heart Attack Occ.,
Cholesterol & TG & LDL & HDL	Age	Correlation	1.000	0.350
		Significance (2-tailed)	.	0.000
		df	0	179
	Time of Heart Attack Occ.,	Correlation	0.350	1.000
		Significance (2-tailed)	0.000	.
		df	179	0

Table 5: Partial correlation with reference to cholesterol, TG, LDL and HDL.

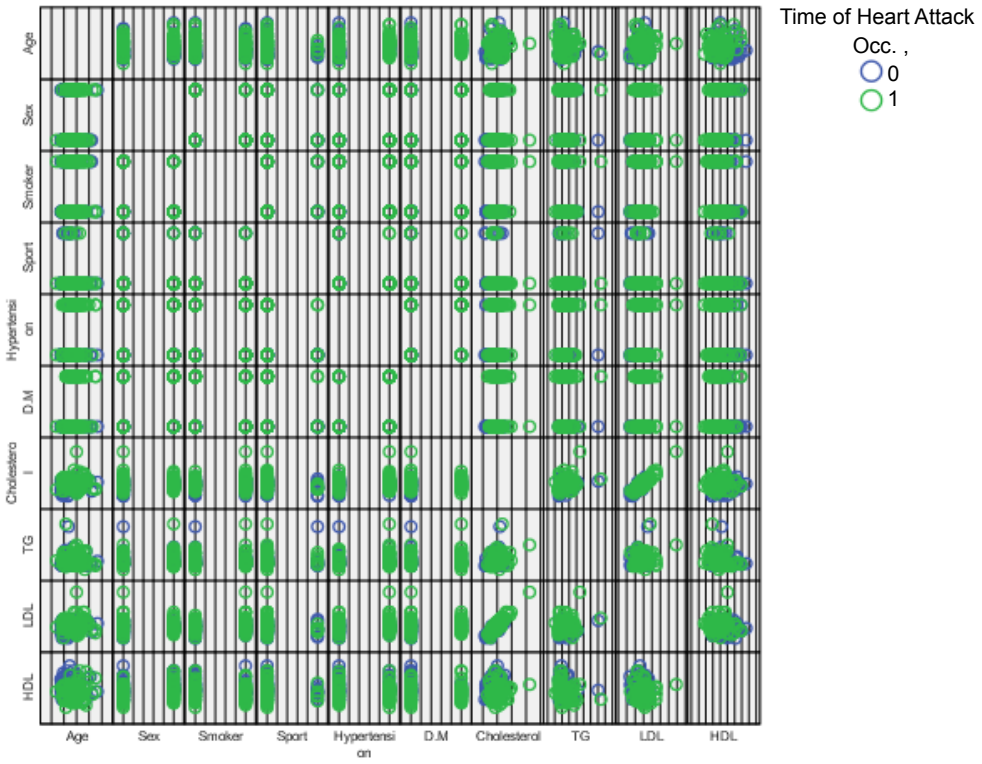

Figure 1: The matrix scatter of all study variables in correlation with healthy-unhealthy heart subjects.

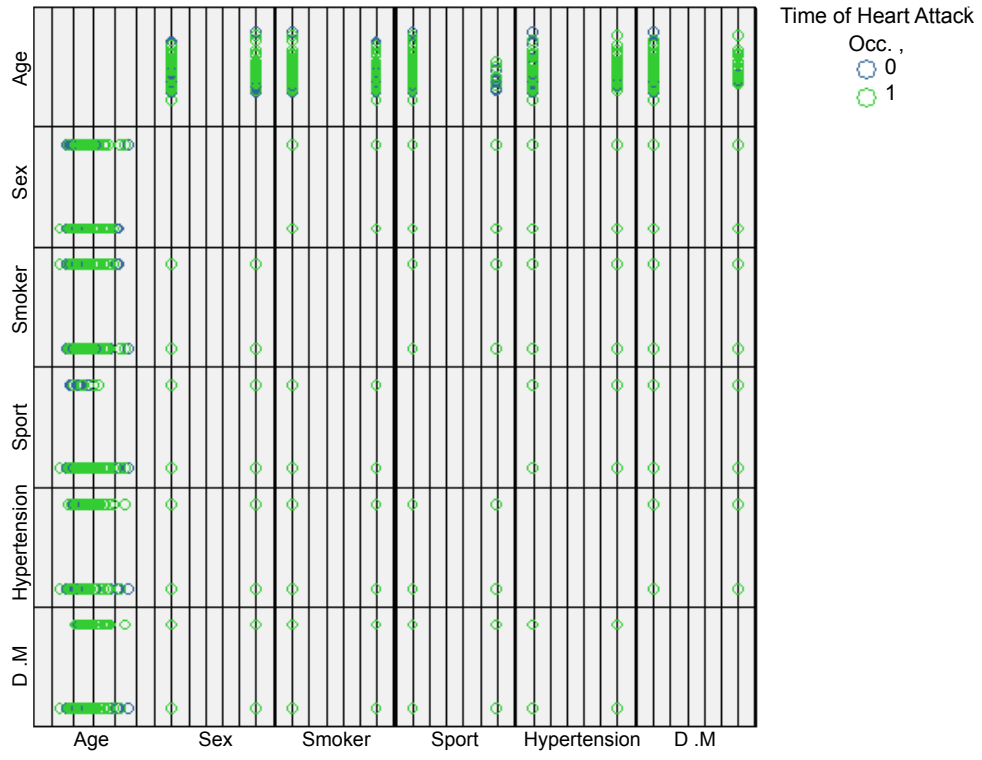

Figure 2: The matrix scatter of demographic variables in correlation with healthy-unhealthy heart subjects.

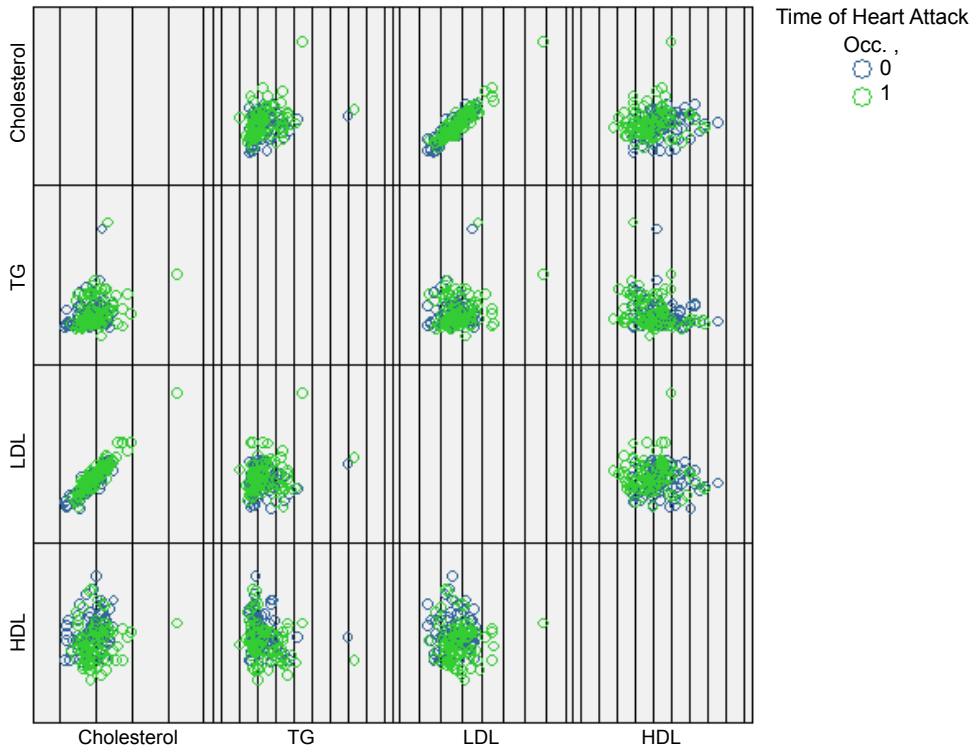

Figure 3: The matrix scatter of blood lipids in correlation with healthy- unhealthy heart subjects.

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