RA Publications
JMST Journal of Medical Science & Technology

Original Article **Open Access**

Study of relationship of heredity with blood pressure, serum lipids and anthropometric indices among Saurashtra population of Gujarat

***Siji Mathew [1, 2], T.M. Chary [3]**

[1] Department of Biochemical Sciences, Rajiv Gandhi Institute of Information Technology and Biotechnology, Bharati Vidyapeeth Deemed University, Pune, India
[2] Department of Biochemistry, Shri M.P. Shah Medical College, Jamnagar. Gujarat, India
[3]Department of Biochemistry, Pushpagiri Institute of Medical Sciences & Research Centre, Tiruvalla, Kerala, India.

Abstract

To study the effect of heredity on blood pressure, serum lipids, body mass index and waist-hip-ratio according to age and gender. The study population consisted of 400 normal healthy individuals as controls and 746 first time detected untreated hypertensive subjects of 30-80 years of age. The control and hypertensive subjects were regrouped into two subgroups i.e. subjects with and without family history within two age-groups: 30-50years and 51-80 years. Blood samples were drawn from all the subjects following an overnight fast and total cholesterol was measured by CHOD-PAP method, serum triglyceride by GPO-PAP method and HDL-C by HDL-C plus method using "Accucare" kits. Serum LDL-cholesterol (LDL-C) was calculated by Fredrickson-Friedwald formula. Blood pressure (BP) and anthropometric measurements were measured by a standardized protocol. Highly significant increases in TC, TG, LDL-C, BMI and atherogenic indices while a decrease in HDL-C were observed in heredity hypertensive groups as compared to non-heredity controls. In women, a significant increase was seen with heredity. Gender wise, men had increased values compared to women except in HDL-C and BMI wherein vice versa was found. Also with increase in age each of the parameters increased in both, the controls as well as the hypertensive subjects except the atherogenic indices. Heredity show an increase in the level of blood pressure, serum lipids and BMI which may be the major contributor to increased risk of cardiovascular morbidity and mortality and in precipitation of HT.

KEY-WORDS: Blood pressure, BMI, Heredity, serum lipids, Waist Hip Ratio (WHR).

*Corresponding Author: Dr. Siji Mathew Asst. Professor, Dept of Biochemical Sciences, Bharati Vidyapeeth Deemed University, Rajiv Gandhi Institute of Information Technology and Biotechnology, Pune, India. E.mail: sijimathew710@gmail.com.
Received: January 13, 2013. Accepted: April 11, 2013. Published: May 20, 2013.

Introduction

Cardiovascular diseases (CVD) are one of the world's leading causes of morbidity and mortality. Researchers estimate that its prevalence will continue to rise over the next few decades. [1] Until now, public prevention strategies have relied predominantly on managing environmental factors that contribute to CVD, such as obesity, smoking and lack of exercise. It is also observed that a positive family history is not an independent primary risk factor of the same magnitude as hypertension (HT), hyperlipidaemia, diabetes mellitus (DM), obesity or smoking but showed that in younger patients the positive family history of premature CHD is consistent with an inherited susceptibility to the action of coronary risk factors. [2] With growing evidence that CVDs have a sizable hereditary component [3-5], more emphasis needs to be given to genetic predisposition as a risk factor to achieve a

better understanding of the disease development. Although a positive family history is widely accepted as an independent risk factor for CVD there is little published evidence to support this view. With this background, the present study was conducted to study the relationship of heredity with blood pressure (BP), serum lipids, body mass index (BMI) and waist-hip-ratio (WHR)

Methodology

Study population: The study population included subjects from Jamnagar with varied lifestyle based on their occupation. The ethical approval was obtained from Institutional ethics committee prior to study initiation. A total of 1146 subjects (593 women and 553 men) participated in the study and informed consent was obtained from all participants. Individuals' ≥ 30 years of age were included in the study. Random subjects free of not only HT but any disease and also who were not on any particular medication were included in the study as controls. The control group consisted of 400 healthy people (217 women and 183 men) in the age group of 30 to 80 years. In the patient group, first time detected untreated hypertensive subjects of 30-80 years of age and a total of 746 (376 women and 370 men) individuals were enrolled. HT was considered to be present if the SBP ≥ 140 mm Hg or the DBP ≥ 90 mm Hg, recent JNC VII [6] classification were used for classifying HT. Control and patient volunteers were further divided into two subgroups i.e. positive family history and negative family history, wherein, subjects having a family history of HT, CVD and DM were considered to be cases with positive family history.

Measurement of blood pressure: It was done by mercury sphygmomanometer. Instruments were calibrated from time to time. Two readings in lying down posture at 10-15 minutes interval were taken and only second reading (being lower and more realistic) was considered.

Collection of samples: Venous blood withdrawn after an overnight fast of 12 hrs was used to estimate the parameters.

Biochemical markers: Total cholesterol was measured by CHOD-PAP method, serum triglyceride by GPO-PAP method and HDL-C by HDL-C plus method using "Accucare" kits. The test was carried out according to the manufacturers' instructions. Measurements of serum cholesterol, triglycerides and HDL-C, were done on the ERBA semi-auto analyzer. (Germany). LDL-C concentration was calculated using Fredrickson-Friedwald formula. [7] The cut-off values for abnormal lipid levels were considered according to the National cholesterol education programme (NCEP) guidelines Adult treatment panel III (ATP III). [8] Atherogenic ratios [TC/ HDL] >5 and [LDL-C/HDL-C] >3.5 was taken as elevated.

Anthropometric measurements: A weighing (bathroom) scale and stadiometer were used to measure the weight (nearest 0.5 kg) and height (nearest 0.1 cm) of each subject using standard procedure. Body Mass Index (BMI) [weight in kg/ height in m^2] was calculated. The waist circumference and the hip were measured using a non-stretchable measuring tape. Waist-hip-ratio (WHR) was calculated from the data.

Statistical analysis: Two separate analyses were performed, 1 by age group (30 - 50 and 51-80 years) and 1 by gender. Mean, standard deviation and standard error were calculated. Student't' test was applied using Minitab statistical software. '*P*' values were calculated to assess significance of the results.

Results

The control and hypertensive subjects were grouped sex wise into two subgroups i.e. positive family history and negative family history within two age-groups: 30-50 years and 51-80 years.

In control group, BP, serum lipids, atherogenic ratios and anthropometric indices expressed a significant rise (Table 1a) in 30-50 age group in females with positive family history expect HDL-cholesterol (HDL-C), which showed a fall, whereas an insignificant increase in all the parameters in 51-80 age group was analyzed. (Table 1) Also, in males a decrease in BP, serum lipids and atherogenic ratios in both the age groups and a rise in triglyceride (TG) and anthropometric indices were observed when compared to negative family history. (Table 1) On the contrary, the hypertensive patients having positive family history illustrated a significant rise (Table 2a) in BP and HDL-C in both the age groups except HDL-C of males in 30-50 age groups when compared to negative family history hypertensive patients. (Table 2) The anthropometric indices displayed a specific trend of increase in both the age groups, but WHR remained alike in 51-80 age group in both the sexes. (Table 2) Moreover, TC, LDL-C and atherogenic ratios lowered in both the sexes of the two age groups whereas TG had a rise in 30-50 age group and nearly related values in 51-80 age

ISSN: 1694-1217 JMST. An open access journal

group in both the sexes. (Table 2) The values of BP, serum lipids, atherogenic ratios and anthropometric indices of hypertensive patients with positive family history were much higher than those seen in control group with negative family history (Table 3) and were enormously significant (P< 0.001) except HDL-C. (Table 3a). An intragroup comparison of control and hypertensive cases with positive family history put forward a parallel manner of increase with age in all parameters (Table 1 & 2) except in control group of males in HDL-C (Table 1). Significance was seen in only BP and anthropometric indices in hypertensive cases (Table 2a) while in control group only BP showed significance. (Table 1a) Moreover in male and female evaluation, normotensive subjects of both age groups and hypertensive patients of 30-50 age group, displayed increase in BP, serum lipids and BMI and a decrease in TG, atherogenic ratios and WHR among the females than the males of the corresponding age groups. (Table 1&2) In hypertensive patients, the females in 51-80 age group had lower values in serum lipids, atherogenic ratios and WHR whereas higher BP, HDL-C and BMI were observed than those of males. (Table 2

Table 1: Effect of heredity on BP, Serum Lipids, BMI and WHR in Normotensive cases

Age Group	Heredity	G	N	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	TC /HDL-C	LDL-C/ HDL-C	BMI	WHR
30- 50	Negative Family History (A_1)	M	35	127.5 ± 2.1	83.1 ± 1.2	180.9 ± 3.1	108.3 ± 6.1	42.74 ± 1.6	116.5 ± 2.7	4.40 ± 0.15	2.85 ± 0.12	24.64 ± 0.61	0.94 ± 0.01
		F	76	122.8 ± 1.2	80.6 ± 0.77	171.0 ± 2.7	91.4 ± 5.4	48.17 ± 1.1	104.6 ± 2.7	3.69 ± 0.10	2.28 ± 0.08	25.30 ± 0.46	0.82 ± 0.006
	Positive Family History (A_2)	M	66	123.0 ± 1.1	80.7 ± 0.68	176.3 ± 3.7	118.9 ± 5.0	42.02 ± 1.2	110.5 ± 3.7	4.39 ± 0.15	2.80 ± 0.13	26.39 ± 0.54	1.02 ± 0.01
		F	82	125.7 ± 1.4	82.4 ± 0.87	184.2 ± 1.9	106.5 ± 3.9	46.44 ± 1.0	116.5 ± 2.2	4.09 ± 0.08	2.62 ± 0.08	26.83 ± 0.55	0.87 ± 0.04
51- 80	Negative Family History (B_1)	M	34	130.5 ± 2.5	85.9 ± 1.7	187.9 ± 5.5	107.6 ± 6.5	41.40 ± 2.0	125.0 ± 5.9	4.83 ± 0.24	3.26 ± 0.22	24.37 ± 0.61	0.94 ± 0.01
		F	39	129.3 ± 2.3	82.2 ± 1.0	179.5 ± 3.7	114.9 ± 3.5	48.8 ± 1.9	107.7 ± 3.6	3.87 ± 0.16	2.37 ± 0.14	25.56 ± 0.80	0.83 ± 0.009
	Positive Family History (B_2)	M	33	126.4 ± 1.4	83.7 ± 1.6	180.5 ± 4.7	122.2 ± 5.7	41.0 ± 1.7	115.0 ± 4.2	4.63 ± 0.21	3.01 ± 0.19	26.30 ± 0.62	0.96 ± 0.01
		F	14	133.7 ± 4.9	86.4 ± 2.3	186.9 ± 6.6	99.8 ± 10.0	50.64 ± 2.0	116.3 ± 7.3	3.74 ± 0.17	2.36 ± 0.19	27.93 ± 1.10	0.84 ± 0.01

Values are Mean ±S.E. G=Gender, SBP – Systolic Blood Pressure, DBP – Diastolic Blood Pressure. TC – Total Cholesterol , WHR - Waist hip ratio

Table 1a: Statistical Analysis of Table 1

Comparison	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	TC/ HDL-C	LDL-C/ HDL-C	BMI	WHR
A_1M vs A_2M	P<0.05	P<0.05	NS	NS	NS	NS	NS	NS	P<0.01	P<0.001
A_1F vs A_2F	P<0.05	P<0.05	P<0.001	P<0.01	NS	P<0.001	P<0.001	P<0.005	P<0.01	NS
B_1M vs B_2M	NS	NS	NS	P<0.05	NS	NS	NS	NS	P<0.01	NS
B_1F vs B_2F	NS	P<0.05	NS	NS	NS	NS	NS	NS	P<0.05	NS
A_2M vs A_2F	NS	P<0.05	P<0.05	P<0.05	P<0.005	NS	P<0.05	NS	NS	P<0.001
B_2M vs B_2F	NS	NS	P<0.05	P<0.05	P<0.001	NS	P<0.001	P<0.01	NS	P<0.005
A_2M vs B_2M	P<0.05	P<0.05	NS	NS	NS	NS	NS	NS	P<0.05	P<0.001
A_2F vs B_2F	P<0.05	P<0.05	NS	NS	P<0.05	NS	P<0.005	NS	NS	NS

NS= Non-significant

Table 2: Effect of heredity on BP, Serum Lipids, BMI and WHR in Hypertensive cases

Age Group	Heredity	G	N	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL.C mg/dl	TC/ HDL-C	LDL-C/ HDL-C	BMI	WHR
30-50	Negative Family History (C_1)	M	62	145.8 ± 2.0	92.6 ± 1.2	251.6 ± 7.6	187.7 ± 11.0	41.1 ± 1.4	173.0 ± 7.2	6.50 ± 0.26	4.49 ± 0.22	27.44 ± 0.61	0.96 ± 0.01
		F	56	143.3 ± 2.0	91.04 ± 1.0	236.2 ± 5.5	161.9 ± 11.0	39.9 ± 1.1	163.9 ± 5.9	6.09 ± 0.18	4.21 ± 0.17	28.25 ± 0.63	0.84 ± 0.009
	Positive Family History (D_1)	M	90	142.7 ± 1.8	90.0 ± 1.1	224.6 ± 4.2	192.7 ± 9.9	37.6 ± 0.9	148.4 ± 4.1	6.25 ± 0.18	4.15 ± 0.15	29.74 ± 0.63	1.01 ± 0.01
		F	95	146.2 ± 1.4	94.4 ± 1.0	229.3 ± 4.0	163.7 ± 8.1	44.2 ± 1.3	162.5 ± 5.5	5.53 ± 0.16	3.70 ± 0.14	31.56 ± 0.64	0.86 ± 0.006
51-80	Negative Family History (C_2)	M	94	145.0 ± 1.5	94.4 ± 0.84	240.9 ± 5.5	201.0 ± 8.6	38.19 ± 0.89	166.2 ± 6.0	6.59 ± 0.22	4.48 ± 0.20	26.67 ± 0.48	0.98 ± 0.008
		F	95	148.4 ± 1.5	92.79 ± 0.83	242.9 ± 5.2	170.4 ± 10.0	42.6 ± 1.0	166.2 ± 6.0	6.08 ± 0.23	4.24 ± 0.23	26.58 ± 0.49	0.87 ± 0.008
	Positive Family History (D_2)	M	118	148.3 ± 1.4	96.8 ± 1.2	180.5 ± 4.7	201.2 ± 8.7	40.5 ± 0.87	158.6 ± 4.2	6.16 ± 0.15	4.08 ± 0.13	27.75 ± 0.38	0.97 ± 0.006
		F	128	151.8 ± 1.4	98.6 ± 1.2	186.9 ± 6.6	169.4 ± 7.0	45.4 ± 0.94	152.6 ± 4.2	5.38 ± 0.14	3.58 ± 0.13	30.09 ± 0.45	0.85 ± 0.005

Values are Mean ±S.E.

Table 2a: Statistical Analysis of Table 2

Comparison	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	TC/ HDL-C	LDL-C/ HDL-C	BMI	WHR
C_1M vs C_2M	NS	P<0.05	P<0.001	NS	P<0.01	P<0.001	NS	NS	P<0.01	P<0.001
C_1F vs C_2F	NS	P<0.01	NS	NS	P<0.01	P<0.05	P<0.01	P<0.01	P<0.05	NS
D_1M vs D_2M	P<0.05	P<0.05	NS	NS	P<0.05	NS	P<0.05	P<0.05	P<0.001	NS
D_1F vs D_2F	P<0.05	P<0.001	P<0.05	NS	P<0.01	P<0.05	P<0.005	P<0.01	P<0.001	P<0.05
C_2M vs C_2F	P<0.05	P<0.001	NS	P<0.01	P<0.001	NS	P<0.001	P<0.01	P<0.01	P<0.001
D_2M vs D_2F	P<0.05	NS	NS	P<0.005	P<0.001	NS	P<0.001	P<0.005	P<0.001	P<0.001
C_2M vs D_2M	P<0.01	P<0.001	P<0.001	NS	P<0.01	P<0.05	NS	NS	P<0.005	P<0.001
C_2F vs D_2F	P<0.005	P<0.01	NS	NS	NS	NS	NS	NS	P<0.05	NS

Table 3: Effect of heredity on BP, Serum Lipids, BMI and WHR in Normotensive and Hypertensive cases

Age Group	Heredity	G	N	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	TC/ HDL-C	LDL-C/ HDL-C	BMI	WHR
30- 50	Non Heredity Control (A_1)	M	35	127.5 ± 2.1	83.1 ± 1.2	180.9 ± 3.1	108.3 ± 6.1	42.74 ± 1.6	116.5 ± 2.7	4.40 ± 0.15	2.85 ± 0.12	24.64 ± 0.61	0.94 ± 0.01
		F	76	122.8 ± 1.2	80.6 ± 0.77	171.0 ± 2.7	91.4 ± 5.4	48.17 ± 1.1	104.6 ± 2.7	3.69 ± 0.10	2.28 ± 0.08	25.30 ± 0.46	0.82 ± 0.006
	Heredity HTsive (C_2)	M	90	142.7 ± 1.8	90.0 ± 1.1	224.6 ± 4.2	192.7 ± 9.9	37.6 ± 0.9	148.4 ± 4.1	6.25 ± 0.18	4.15 ± 0.15	29.74 ± 0.63	1.01 ± 0.01
		F	95	146.2 ± 1.4	94.4 ± 1.0	229.3 ± 4.0	163.7 ± 8.1	44.2 ± 1.3	152.5 ± 4.1	5.53 ± 0.16	3.70 ± 0.14	31.56 ± 0.64	0.86 ± 0.006
Above 50	Non Heredity Control (B_1)	M	34	130.5 ± 2.5	85.9 ± 1.7	187.9 ± 5.5	107.6 ± 6.5	41.40 ± 2.0	125.0 ± 5.9	4.83 ± 0.24	3.26 ± 0.22	24.37 ± 0.61	0.94 ± 0.01
		F	39	129.3 ± 2.3	82.2 ± 1.0	179.5 ± 3.7	114.9 ± 3.5	48.8 ± 1.9	107.7 ± 3.6	3.87 ± 0.16	2.37 ± 0.14	26.56 ± 0.80	0.83 ± 0.009
	Heredity HTsive (D_2)	M	118	148.3 ± 1.4	96.8 ± 1.2	239.3 ± 4.6	201.2 ± 8.7	40.5 ± 0.87	158.6 ± 4.2	6.16 ± 0.15	4.08 ± 0.13	27.75 ± 0.38	0.97 ± 0.006
		F	128	151.8 ± 1.4	98.6 ± 1.2	231.9 ± 4.0	169.4 ± 7.0	45.4 ± 0.94	152.6 ± 4.2	5.38 ± 0.14	3.58 ± 0.13	30.09 ± 0.45	0.85 ± 0.005

Values are Mean ±S.E.

Table 3a: Statistical Analysis of Table 3

Comparison	SBP mmHg	DBP mmHg	TC mg/dl	TG mg/dl	HDL-C mg/dl	LDL-C mg/dl	TC/ HDL-C	LDL-C/ HDL-C	BMI	WHR
A_1M vs C_2M	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001
A_1F vs C_2F	P<0.001	P<0.001	P<0.001	P<0.001	P<0.01	P<0.001	P<0.001	P<0.001	P<0.001	P<0.001
B_1M vs D_2M	P<0.001	P<0.001	P<0.001	P<0.001	NS	P<0.001	P<0.001	P<0.001	P<0.001	P<0.01
B_1F vs D_2F	P<0.001	P<0.001	P<0.001	P<0.001	P<0.05	P<0.001	P<0.001	P<0.001	P<0.001	P<0.05

Discussion

In the present study an effort was made to study the effect of heredity and its relation to BP, serum lipids and anthropometric indices in hypertensive volunteers. There was a significant increase in BP in the hypertensives with positive family history in both genders along with their corresponding age groups. HT is a serious disease that can result in heart attack, strokes or kidney failure and scientists have known for some time that HT is a heritable condition that runs in families. Elizabeth *et al* [9] showed significantly higher BP and total cholesterol (TC) in men less than 60 years of age with positive history of heart attack. Heller [10] also found on an average the first degree relatives of hypertensive patients to have higher BP than those relatives of normotensive controls. Some studies have suggested that high blood pressure may be associated with the presence of E_4 allele, when studies aimed to find out the association of apo E genotypes with HT were carried out [11,12]. The genetic variations at apo E have also been shown to affect lipid and lipoprotein levels in the general population [13].

With regard to lipids in this study, TC and LDL-C concentrations showed an insignificant decrease in hypertensive with positive family history while TG concentration did not vary in both age groups and their genders when compared to hypertensives with negative family history. But yet, when compared to the normotensive negative family history all serum lipids demonstrated tremendous significant increase among the hypertensives with positive family history except HDL-C concentration. One of the best understood inborn error of metabolism determining elevated levels of plasma cholesterol and LDL-C is the autosomal dominant disorder of familial hypercholesterolemia (FH). Studies indicate that 5-10% of individuals with premature MI may have FH [14]. A study on FH indicated that there exist mutational heterogeneity if the LDL receptor gene among the Indians, wherein, none of the mutation were reported to be common among Indian immigrants [15]. In recent times new markers like the apo E gene polymorphism stated earlier have been found associated with CHD and are being extensively studied [16,17]. Mowar *et al* [18] also studied coronary risk factors in family members, close or distant relatives of patients having MI and observed significant TC and TG concentrations in brothers and sisters in comparison to those without having MI.

A genetic predisposition to CAD is suggested by high levels of lipoprotein in Asian Indians [19]. Backer [20] observed (LDL-C+VLDL-C)/apo B and HDL-C/apoA-1 values to be very high in offspring group in comparison to the control group. HDL-C concentration also was less and TC/HDL-C ratio was high in those with positive family history which suggested that the protective role of HDL-C against atherosclerosis was very much reduced. Widhalm *et al* [21] showed that the progeny of families with manifestation of CHD could be distinguished from children with negative family history with the help of HDL-C, TC/HDL-C and LDL-C/HDL-C, where as apoA-1/apo B was the strongest discriminator.

The causes of obesity are many, but there is little doubt that genetic factors play an important role in its etiology. In this study the anthropometric indices, BMI and WHR showed a positive correlation with hypertensive positive family history patients.

Statistical analyses suggest that 50% or more of the variation between individuals in BMI has a genetic basis [22] but these effects are dominated by polygenic environmental interactions that reflect many genetic influences. The increasing epidemic of obesity has stimulated interest in identifying or predicting individuals who are at greatest health risk at an early age [23]. These observations emphasize the need to monitor HT and lipid metabolism in those with a positive family history to reduce the susceptibility to CAD.

However genes alone don't explain the spurt in HT and heart diseases among the young. The answer, in a word, is lifestyle. A recent study indicated that people with a family history of CAD have two times the risk of having a significant elevation in cholesterol [24, 25]. The solution to these, sky-rocketing figures are – Simply tweaking our lifestyles a little.

CONCLUSION

This study showed all the parameters except HDL-C in females and in males, only TG and anthropometric indices had an increase in control group with positive family history. Whereas in the hypertensive cases, excluding TC and atherogenic ratios, other parameters had an increase when compared to negative family history group. Therefore, understanding the genetics behind the onset and development of CVD is a critical part of the prevention and management part of CVD. While genetics put an individual at risk for disease, environmental factors may increase or decrease a person's chances of developing the disease. In a nutshell "Genetics load the gun, lifestyle pulls the trigger".

References

1. World Health Organization. *World Health Report 2002: Reducing risks, promoting healthy life.* Geneva, WHO 2002.
2. Rohan M.C., Risteard M. Is a family history of CHD an independent coronary risk factor. BRI Heart J. 1985; 53: 378-381.
3. Dhanraj T.J., Acker M.S., Danaraj W., *et al*. Ethnic differences in CHD in Singapore: an analysis of necroscopy. Am Heart J. 1959; 58: 516-526
4. Enas E.A., Yusuf S. and Mehta J.L. Prevalence of CHD in Asian Indians. Am J Cardiol. 1992; 70: 445-449
5. Jha P., Enas E.A., Yusuf S. CAD in Asian Indians: Prevalence of risk factors. Asian Am Pac Islander J Health 1993; 1: 161-175.
6. Chobanian A.V., Bakris G.L., Black H.R. The seventh report of the joint national committee on prevention, detection, evaluation and treatment of high blood pressure- the JNC 7 report. JAMA 2003; 289:2560-2572.
7. Friedwald W.T., Levy R.I. and Fredrickson D.S. Estimation of the concentration of LDL-C in plasma, without the use of the preparative ultracentrifuge. Clin Chem 1972; 18: 499
8. Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adults Treatment Panel III). JAMA 2001; 285:2486-2497.
9. Elizabeth B.C., Kay-tee Khaw. Family history of heart attack as an independent prediction of death due to CVD. Circulation 1984; 69: 1065-1069.
10. Heller R.F., Robinson N., Peart W.S. Value of BP measurements in the relatives of hypertensive patients. Lancet 1980; ii: 1206-1208.
11. Yilmag H, Isbir T, Agachan B, *et al*. Is epsilon 4 allele of apo E associated with more severe end-organ damage in essential HT? Cell Biochem 2001; 19: 191-195.
12. Dembinska Kice A, Kaweela-Jaszez K, Kwasnaik G.I., *et al.* Apo E isoforms, insulin output and plasma lipid levels in essential HT. Eur J Clin Invest 1998; 28: 95-99.
13. Breslow J.L.. Apolipoprotein genetic variation and human disease. Physiol Rev 1988; 68: 85-98.
14. Goldstein J.L., Brown M.S. Familial hypocholesterolemia. *In*: Scriver R, Beaudet A.L., Sly W.S., Valle D., eds. *The Metabolic and Molecular Bases of Inherited Disease*. New York: McGraw Hill, 1995; 1981-2030.
15. Ashavaid T.F., Kondkar A.A. and Nair K.G. Identification of two LDL receptor mutation causing FH in Indian subjects. J Clin Lab Anal 2000; 14: 293-298.
16. Siest G, Pillot T, Regis-Bailly A, Leininger-Miller B, *et al*. Apo E: an important gene and protein to follow in laboratory medicine. Clin Chem 1995; 41: 1068-1086

17. Lynn B.J., Roger R.W.. Relation between family history of CAD coronary risk variables. Am J Cardiol 1988; 62: 708-713.
18. Mowar S.M., Pal S.K., Ghosh K.K. Coronary risk factors in families. Ind Heart J 1977; 29: 112-117.
19. Jha P, Enas E, *et al*. CAD in Asian Indians: Prevalence and Risk factors. Asian AM Pac Isl J Health 1993; 1(2): 163-175.
20. De Backer G, Hulstarrt F, *et al*. Serum lipids and apoproteins in students whose parents suffern prematurely MI. Am Heart J. 1996; 112: 478-484.
21. Widhalm K, Koch S, Pakesta R, *et al*. Serum lipids, lipoproteins and apolipoproteins in children with and without family history of premature CHD. J Am College of Nutr 1992; 11(suppl): 323-355.
22. Allison D, Matz P.E., Pietrobelli A, et al. Genetic and environmental influences on obesity. In: Bendich A, Deckelbaum RJ, eds. *Primary and secondary preventive nutrition*. Totowar NJ: Humana Press, 2001: 147-164.
23. Ezzati M, Lopez A.D., Rodgers A, *et al.* Comparative Risk Assessment Collaborating Group. Selected major risk factors and global and regional burden of disease. Lancet 2002; 360: 1347-1360
24. Shih P.B., O'Connor D.T Hereditary determinants of human hypertension: strategies in the setting of genetic complexity. Hypertension. 2008 June; 51(6): 1456–1464.
25. Kuklina E, Yoon P, Keenan N. Prevalence of coronary heart disease risk factors and screening for high cholesterol levels among young adults, United States 1999-2006. Ann Fam Med. 2010;8:327-333