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Original article

EFFECT OF FAMILY HISTORY OF CHRONIC DISEASE ON CARDIOVASCULAR RISK FACTORS: ETHNIC AND SEX-BASED VARIATIONS AMONG CO-INHABITANTS

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ABSTRACT

Background: Family history of the chronic disease (FHD) has been considered as an important factor in lifestyle behaviour in addressing cardiovascular disease risk. Numerous studies have reported how positive FHD triggers lipid parameters, resulting in escalating risk factors. Variation in ethnicity and lifestyle behaviour results in differences in disease outcome despite sharing common ecology.
Methods: This cross-sectional study, with 625 participants from different ethnicities, focused on anthropometric variables with lipid parameters and family history of disease. The participants in the age group 18-35 years, with different ethnicity, were considered in this study.
Results: Considering both sexes, Bengali participants displayed notably higher mean values for most CV risk factors compared to their Bhumij counterparts. Multivariate analyses explained pronounced differences in body composition and lipid profiles across FHD categories, with both ethnicity and sex serving as significant covariates. Ethnicity accounted for 33–35.6% of the variance, while sex explained almost 80.4%, underscoring the substantial roles of these factors in modulating the impact of FHD on CV risk.
Conclusion: The study concludes that a positive family history of disease (FHD) significantly elevates CV risks, moderated by both ethnicity and sex. While FHD is strongly associated with increased CV risk factors, particularly among Bengali participants, both ethnicity and sex substantially influence this relationship. These findings are sufficient to understand population-specific CVD (cardiovascular disease) prevention strategies that indicate familial, ethnic, and sex-based risk considerations.

KEY WORDS: CVD risk, family history of chronic disease, ethnicity, young adults, modifiable factor

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INTRODUCTION

Cardiovascular disease is a global burden that accounts for significant morbidity and mortality worldwide. The CVD (cardiovascular disease) result due to abnormal functioning of the cardiovascular health (CVH), which comprises of heart, blood vessel, blood pressure and circulatory system. CVD is complex because of its multifactorial (interaction between environmental, genetic, and physiological conditions) nature. Nearly 32% of overall deaths in 2030 will be accounted for by CVD alone (Iloh et al., 2013). Studies have shown that the magnitude of the CVD risk varies across age groups, sexes, and ethnicities due to FHD (Pandey et al., 2013). The term "FHD" (family history of chronic disease) is an umbrella term under which conditions like hypertension (Yucel et al. (2012), diabetes, hyperlipidemia, etc. have been explained as the reason for CVD risk. The FHD magnified the predisposition because of shared environment, genetic, and behavioral traits. Further, this predisposition will develop into CVD and other NCDs (non-communicable diseases). The presence of a family history of diabetes will raise the CVD by 200% as compared to non-diabetic FHD (Tohidi et al., 2010). The prevalence of the family history of disease keeps on increasing from 7.1% to 8.4% in six consecutive years from 2000-2006, as reported by the Anti-HIV Drug study (2008).This indicates the Escalating rates of FHD.

The family history of disease is an intrinsic genetic trait that encompasses more than biological and physiological conditions through its expressivity. The family members resemble the disease risk because of shared condition (Valdez et al., 2010). Those who inherit certain genes will have a predisposition to IR (insulin resistance), dyslipidemia hypertension, obesity. The degrees of relation with relatives like first, second, and third also decide the magnitude of the impact (Friedlander et al., 1998). Studies have published the salt retention capacity vs hypertension among black people (Schutteet al 2017). Beyond genetic factors, lifestyle factors play a vital role in addressing CVD risk factors (Schutte, 2019) because, enough physical activity, a healthy diet, and cessation of tobacco and alcohol consumption will reduce the potentiality of FHD traits. These above lifestyle behaviors have been referred by AHA (Lloyd-Jones et al., 2022), and if not adhered will increase disease susceptibility. To understand the actual phenomenon of FHD role play, the interaction of other factors like diet, physical activity, sex, age group, and environment (region) should be considered. Identifying a positive family history and mitigating its potential (Valdez et al.,2010) through modified lifestyle behaviors is not a good option but a mandatory choice to tackle the effect of predisposing CVD risk.

This study will further investigate the effect of FHD on risk factors across sex and ethnicity. Interestingly here the regional ethnicity sets a frame or boundary because two ethnic groups residing in the same environment aligned with similar availability of diet, physical activity, more or less lifestyle conditions.

OBJECTIVES

1. To compare the ethnic differences in CVD risk factors of the participants across ethnic groups.
2. To find the association between FHD and CVD risk factors among both sexes.
3. To evaluate the independent influence of FHD on CVD risk factors controlling for ethnicity and sex.

METHODS

Study Area & Participants

This cross-sectional study was conducted between 2022 and 2023 with its overall aim to explore the effect of selected factors on CVD risk variables. North 24 Parganas, a district of West Bengal, India was chosen depending on the "concentration and co-inhabitants" of the participants. These areas with shared inhabitants have been given importance in this study because of two different ethnic groups residing in a particular place. The two ethnic groups are Bengali Hindu (caste population) and Bhumij (Scheduled Tribe). The sample size was calculated using the formula $N=Z^2pq/d^2$ (Iloh et., 2013), where N= minimum sample, Z= 1.96, p=portion of population estimated (here 30% or 30/100, as per Krishnamoorthy et al, 2020), q=1-p, e= 5% margin of error or 5/100. The final sample size was 720 participants, out of which only 625 were found suitable for this particular study. The prefixed inclusion criteria were: 1) adults between 18-35 years and, 2) willing to take part and; the exclusion criteria were: 1) participants taking any hormonal therapy or drugs (antihypertensive, diabetic, etc.) and, 2) pregnant or outcomes recently became mother (less than 6 months).

Study Variables

The data have been collected with the predesigned schedule by trained specialists. Depending on the objectives of the study, lipid parameters, body composition, and FHD have been given importance. Moreover, individual demography has been recorded. The anthropometric variables like Weight, Height, WC (waist circumference), and HC (hip circumference) have been measured nearest to 0.1 kg and 0.1 cm Weir et al., 2019). The BMI (body mass index) and FM were calculated using a standardized formula (Heyward, 1996). The weight and body fat were accessed through a body scanner (Karadascan - Omron HBF 375, Tokyo, Japan), and the lipid parameters (in mgdl) like TC (Total Cholesterol), TG (Triglyceride), LDLc (low-density lipoprotein Cholesterol), HDLc (high-density lipoprotein Cholesterol), and VLDLc (Very-LDLc) along with this FBG (fasting blood glucose) has been collected with a portable lipid analyser (Lipidometer, SD Biosensor, South Korea) after mandatory fasting for more than 9 hours. The Blood pressure (BP) was analysed using an Omron (HEM 7142T1) digital machine. The participants were asked to sit quietly for at least 15 minutes before the test. BP was recorded on the right hand, in sitting posture two times with a gap of five minutes (Kshatriya et al, 2022). If the result of two consecutives were different by more than 5 mmHg, then again it is measured and the most different value was discarded. Before the day of the check up, they were advised to avoid alcohol, tobacco, milk products, sweets, etc. For all the above measurements, the standard anthropometric technique has been followed (Lohman et al., 1988). The Mets (Metabolic Syndrome) criteria of NCEP ATP III with Asian modification have used (Soewondo et al., 2010). Family history of chronic disease has been noted using a pre-structured schedule. Not only the presence or absence of disease but also the type of disease and, its relation with a person has been noted in the main study. Out of which the presence and absence of FHD were used in this part. In order to maintain their integrity, the self-reported

FHD details were cross-checked with back-references with pedigree analysis.

Statistical Analysis

After cleaning the data in Excel, normality with the Shapiro-Wilks test has been checked and the skewed data were dealt with Box-Cox transformation (Wang et al., 2018). The frequency distribution of the FHD has been obtained. Further to compare the mean difference within the group and between groups and ethnicity " ANOVA" test has been performed due to its robustness. To find out the association between FHD and CVD risk factors multivariate test - "MANCOVA" was used with FHD as a fixed factor and ethnicity as a covariate for both males and female. Similarly, ethnicity and sex were kept as covariates for the pooled data set. The output was reported in the form of descriptive and inferential statistics with their p-value, Wilk's λ, and partial η^2.All the analysis has been done with IBM SPSS (version-25.0). The statistical significance was set at $p<0.05$.

RESULTS

Table 1 details the frequency distribution of the family history of disease among participants. Notably, a significant proportion of participants, specifically 35.81% of Bengali individuals and 27.24% of Bhumij individuals, reported a positive family history of the disease.

T t-test was conducted to compare the mean differences in cardiovascular disease (CVD) risk variables between participants of the same sex across ethnic groups. The results, presented in Table 2.1, reveal significant differences in various CVD risk factors between Bengali and Bhumij males. Bengali males exhibited significantly higher mean values for WHR (t=3.082, p<0.01), %BF (t=2.891, p<0.01), TG (t=5.203, p<0.000), LDLc (t=2.580, p<0.01),, and VLDLc(t=5.203, p<0.000) as compared to Bhumij males. Conversely, Bhumij males demonstrated significantly higher mean value HDLc (t=10.534, p<0.000)

A similar pattern emerged when comparing females from the two ethnic groups. Bengali females displayed significantly higher mean values for WC (t=2.940, p<0.01),WHR (t=4.026, p<0.000), %BF (t=3.379, p<0.01), FM (t=2.664, p<0.01),TC (t=3.571,p<0.000), TG (t=4.507, p<0.000), LDLc (t=2.256, p<0.05),, and VLDLc(t=4.507, p<0.000) and FBG (t=2.689, p<0.01),compared to Bhumij females. However, Bhumij females had a significantly higher mean value for HDLc.

Table 2.2 presents the mean differences in cardiovascular disease (CVD) risk variables among male participants with positive and negative family histories of chronic disease. Notably, Bengali males with a positive family history of disease (FHD) exhibited significantly higher mean values for several CVD risk factors, including BMI (t=2.957, p<0.01), WC (t=3.392, p<0.01), WHR (t=2.163, p<0.05), BF% (t=3.280, p<0.01), FM (t=3.351, p<0.01), DBP (t=2.514, p<0.05), TC (t=2.487, p<0.05), TG (t=2.531, p<0.05), VLDLc (t=2.531, p<0.05), compared to those with a negative FHD. In contrast, among Bhumij males, only two CVD risk factors, namely BMI (t=1.996, p<0.05) and TC (t=2.451, p<0.05), demonstrated significant mean differences between individuals with positive and negative FHD. These findings suggest that a positive family history of chronic disease is associated with a higher risk of CVD among Bengali males, whereas the

relationship is less pronounced among Bhumij males.

Table 2.3 presents the mean differences in CVD risk variables among female participants with positive and negative family histories of chronic disease. Notably, among Bengali females, those with a positive family history of disease exhibited significantly higher mean values for SBP ($t=2.089$, $p<0.05$) and DBP ($t=2.379$, $p<0.05$) compared to those with a negative FHD. In contrast, among Bhumij females, a positive FHD was associated with significantly higher mean values for several CVD risk factors, including BMI ($t=2.686$, $p<0.01$), WC ($t=2.857$, $p<0.01$), BF% ($t=2.168$, $p<0.05$), FM ($t=3.008$, $p<0.01$), and FBG ($t=2.130$, $p<0.05$). These findings suggest that a positive family history of chronic disease is associated with a higher risk of CVD among Bhumij females, particularly in terms of anthropometric and metabolic risk factors.

The NCEP ATP III has been used to isolate the raised BP, FBG, WC, TG and reduced HDLc of the participants (Table 2.4). Interestingly it has been found those Bengali males showed pronounced frequency of raised variables like BP, WC, TG, and reduced HDLc as compared to Bhumij males, but have low FBG. Among females, Bengali had surpassed the frequency of raised variables than Bhumij counterpart.It is very clear that Bengali participants were more hypertensive, having higher fasting blood glucose with abdominal obesity, and elevated triglyceride with low HDLC than their counterparts indicating a disturbed metabolic function.

Table 3.1 presents the results of a MANCOVA analysis conducted on the combined male participant data, to check the influence of FHD on combined CVD risk factors ethnicity as the covariate. Substantial differences emerged in the mean values for BMI, WC, %BF, FM, TC, TG, LDLc, and VLDLc, with p-values reaching significance at both the 0.05 and 0.01 levels. In contrast, no significant differences were noted for WHR, SBP, DBP, HDLc, and FBG. Notably, participants with a positive family history of disease consistently exhibited higher mean values in these risk factors compared to those without. Among male participants, family disease history was found to significantly influence combined CVD risk factors when controlled for ethnicity, as indicated by $F(12,296) = 2.311$, $p < 0.05$, Wilk's $\lambda = 0.914$, and a partial η^2 of 0.086. This effect size suggests that family history of disease accounts for 8.6% of the explained variance in CVD risk factors within the model. These findings underscore a meaningful association between family history and CVD risk profiles.

Ethnicity itself was a significant covariate, further moderating the relationship between family history and CVD risk factors. The analysis yielded $F(12,296) = 2.311$, $p < 0.01$, Wilk's $\lambda = 0.644$, and a partial η^2 of 0.356, indicating that ethnicity accounts for 35.6% of the variance in the model. These results point to a pronounced role of ethnicity in shaping the impact of family disease history on cardiovascular risk factors (Table 3.1).

Multivariate Analysis of Covariance (MANCOVA) analysis was performed on combined female participants to assess the association between family history of disease and cardiovascular disease risk factors, with ethnicity controlled as a covariate (Table 3.2). The analysis reveals a pattern somewhat consistent with

findings among male participants, showing significant distinctions in CVD risk factors across FHD categories. In particular, participants with a positive FHD exhibited significantly elevated mean values for BMI, WC, BF%, FM, SBP, and DBP, with p-values below 0.01 and 0.05, compared to those without a family history of disease. However, the analysis did not find a significant association between FHD and the overall set of combined CVD risk factors, as indicated by $F(12,301) = 1.088$, $p > 0.05$, Wilk's $\lambda = 0.958$, and a partial η^2 of 0.042, suggesting that FHD explains only 4.2% of the total variance in CVD risk factors within the model but this is not significant. This minimal explained variance points to an insubstantial relationship between FHD and the broader profile of CVD risk factors in this group.

Ethnicity, controlled as a covariate, demonstrated a modest but statistically significant role in influencing the association between FHD and CVD risk factors with $F(12,301) = 13.231$, $p < 0.01$, Wilk's $\lambda = 0.653$, and a partial η^2 of 0.347, the results suggest that ethnicity accounts for 34.7% of the variance in the model. This notable effect underscores the nuanced role of ethnicity in affecting the impact of FHD on CVD risk, indicating that the strength of the FHD association with CVD risk factors is distinctly modulated by ethnic background. These findings are detailed in Table 3.2.

A comprehensive MANCOVA was conducted on pooled participant data (Table 4), with family history of disease (FHD) as the fixed factor, while controlling for the effects of ethnicity and sex, to isolate the specific impact of FHD on cardiovascular disease (CVD) risk factors. The analysis revealed that participants with a positive FHD consistently exhibited significantly higher mean values across CVD-related variables, including body composition indicators (BMI, WC, WHR, %BF, FM), blood pressure (SBP and DBP), lipid parameters (TC, TG, LDLc, and VLDLc), and fasting blood glucose (FBG), all with p-values below 0.05 and 0.01. HDLC was the only variable that did not show a significant difference. The findings underscore a significant mean difference in CVD risk factors associated with FHD, as indicated by $F(12,610) = 2.429$, $p < 0.05$, Wilk's $\lambda = 0.954$, and a partial η^2 of 0.046. This suggests that FHD accounts for 4.6% of the variance in CVD risk factors within the pooled sample. Notably, the influence of FHD appeared more pronounced in male participants, while it was not significant among females. As a result, when combining both sexes in the pooled analysis, the overall potential effect of FHD on CVD risk was diminished.

Sex emerged as a highly significant factor in explaining variance, with a partial η^2 of 0.804, $F(12,610) = 208.529$, $p < 0.01$, indicating that sex alone accounts for approximately 80.4% of the total variance in the model. Ethnicity, although contributing less variance compared to sex, still showed a statistically significant effect, with a partial η^2 of 0.330, $F(12,610) = 25.040$ and $p < 0.01$, indicating that ethnicity accounts for 33% of the variance in CVD risk factors. These results suggest that both sex and ethnicity substantially modulate the influence of FHD on CVD risk factors, with sex having the most pronounced effect. Consequently, the association between FHD and CVD risk factors demonstrates variation across sex and ethnic groups, as detailed in Table 4.

DISCUSSION

This study sought to examine the association between family history of disease and CVD risk factors, particularly those consistently prioritized by the World Health Organization (WHO) and the American Heart Association (AHA). The frequency table effectively illustrates the prevalence of FHD among participants, revealing notable distinctions. Specifically, Bengali males exhibit a higher prevalence of FHD compared to Bengali females, with both groups showing greater prevalence than Bhumij participants. This disparity highlights an ethnic variation in FHD prevalence. Additionally, the analysis of variance T-test shows a significantly higher mean prevalence among Bengali participants, further underscoring these ethnic differences.

The essence of the FHD in Tables 2.2 and 2.3 illustrates its significant influence on CVD risk factors. Through t-test mean comparisons, the data reveals that within any given ethnic or sex group, those with a "positive FHD" consistently show higher mean values for CVD risk factors, indicated by a significant t value. This aligns with findings from Valdez et al. (2010), who reported increased odds for Type 2 diabetes and CVD associated with positive FHD, with the odds doubling when moving from one to two first-degree relatives with a history of these conditions, thus underscoring the potential of FHD. For example, Friedlander et al. (1998) found a notable odds ratio of 1.57 (95% CI: 1.27–1.95) for primary cardiac arrest among individuals with first-degree relatives affected by CVD. Supporting this, a comprehensive review by Pandey et al. (2013) showed that, even after adjusting for age, gender, and ethnicity, those with positive FHD had a higher odds ratio for coronary heart disease (CHD), emphasizing that FHD significantly increases the likelihood of developing CVD and coronary disease. Consistent with these studies, the present results reveal a higher mean CVD risk for individuals with positive FHD, highlighting its critical role in assessing risk across different demographic groups. A study by Kumar (2015) highlights the influence of parental obesity on lipid profiles and body composition, showing significant differences in the mean values of BMI, LDLc, and TC. Our findings align with this study, echoing a higher mean across most body composition and lipid parameters, underscoring the impact of family history through inherited traits. Similarly, a family-based study by Yucel et al. (2012) identified a family with coronary artery disease (CAD) and hypertension (HT) in three brothers and their mother. The 10-year Framingham risk assessment revealed a 13% risk for the mother (with only hypertension), 4% for the elder brother (with only CAD), and 7% for the younger and older brothers (with both CAD and HT). Multivariate analysis in our study highlights "sex" as a significant factor in FHD impact, explaining 80.4% of the variance in its influence. Jowell et al. (2023), in a study of 166,714 individuals with self-reported family history of heart disease (FFHD), observed a significant prevalence of hypertension, diabetes, cholesterol, lipoprotein imbalances, and elevated mean levels of TC, LDLc, TG, ApoA, and ApoB among participants with positive FFHD. These results support the relation of lipid parameters with FHD, which resembles significant mean differences across FHD categories in our study.

In our study's multivariate analysis further clarifies FHD's role, explaining approximately 4% to 8% of the variance in CVD risk factors. Parallel results have been noted from, Galema-Boers et al. (2018) that among individuals with familial hypercholesterolemia, even those undergoing lipid-lowering treatment had a high incidence of secondary cardiac events, especially among participants with hypertension and a family history of premature CVD. Reflecting these results, Tohidi et al. (2010) found that the prevalence of FHD-related premature CVD was higher among diabetic and non-diabetic participants with CVD compared to those without CVD. A recent study from 2023 by Taylor et al. shows a similar trend in the results in line with this study, by confirming that offspring with a positive parental history of CVD were exhibiting higher mean for lipid parameter, diabetes, and hypercholesterolemia with $p<0.001$ as compared to those without parental history of CVD. In their adjusted (age and sex) model, the odd ratio in CVD outcomes in offspring due to parental CVD was 1.71(1.33-2.21, 95%CI, $p<0.001$). The study by Mehta et al. (2020) demonstrates a pattern consistent with our findings, revealing those individuals with a positive family history of disease—specifically coronary heart disease in their case exhibited higher mean levels of TC, TG, and LDL, along with a lower mean level of HDLc. Furthermore, their research highlighted significant ethnic disparities in the prevalence of FHD, particularly between white and black participants. Similarly, our study underscores the role of ethnicity in modulating CVD risk, accounting for 34.7% of the variance in combined CVD variables and indicating a subtle yet meaningful influence. Ethnic differences in body composition and CVD risk profiles are shown in Table 2.1. These disparities align with studies reporting ethnic variations in adiposity and body composition as key determinants of CVD risk by Carter et al. in 2023 during their study among Malay, Indian, Chinese, and White adults. Even Chaturvedi in 2003 highlighted that Asiansare more prone to atherosclerosis because of their smaller coronary vessel size as compared to Europeans peoples. In line with the above results, the present found the potentially significant role of ethnicity in describing the influence of FHD on CVD risk factors. These findings affirm that the impact and intensity of FHD on CVD risk factors vary distinctly across ethnic groups.

CONCLUSION

All the analyses applied in this study indicate an impactful association of FHD with CVD risk variables. It should be taken into consideration of how sex and ethnicity account for CVD risk variables before concluding about FHD. The t test analyses clear the difference in risk factors across ethnicity and FHD. Those who had positive FHD exhibits significantly higher mean as compared to their counterparts. Interestingly, sex as a dynamic covariate has enough potential to maneuver the impact of FHD. It is due to dimorphic nature of human body, reflected through different biological attributes and characteristics. This interaction needs some refined, multidimensional approach in evaluating CVD risk that combines genetic, sex-specific, and ethnic dimensions with cultural behaviour especially lifestyle attributes to capture the full scope of FHD's impact.

Moreover, monitoring, diagnosing, and treating FHD is the only way to keep safe

from CVD events. Without such interventions, individuals will likely to experience an escalated risk.

Limitations

In this present study, though the sample is enough as per the sample estimation formulae when we classified more FHD groups the distribution becomes uneven for representation. If more focus had been given to the generation and degrees of association of FHD with CVD risk factors, then the magnitude of impact would have been cleared. Though we have tried to crosscheck the degrees of FHD among relatives till the previous 3rd generation (traced back to the father's and mother's side) but felt some gap, because most of them were unaware about their FHD.

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Conflict of Interest

The authors have no conflicts of interest to declare.

Ethics Statements

The Institutional Ethical Committee of Sidho-Kanho-Birsha University, Purulia has approved this study with vide no-Ref/IEC/465/01/C/a/22 issued on 12.04.22. Written consent was obtained from each participant prior to the commencement of the study.

Author's Contributions

Field work and data collection were done by CSU and IB. MD designed the study and analyzed the data. Further, the manuscript was written by CSU and checked formatted by MD. All the Authors approved the final version.

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Table 1. Frequency of family history of chronic disease among participants by ethnicity and sex.

Ethnicity & Sex	Family History of Disease			
	Positive		Negative	
	n	%	n	%
Bengali Male	52	32.91	106	67.09
Bengali Female	60	38.71	95	61.29
Bhumij Male	41	26.97	111	73.03
Bhumij Female	44	27.50	116	72.50

Table 2.1. Mean difference in the CVD risk factors between Bengali and Bhumij participants

	Bengali Male (n=158)		Bhumij Male (n=152)		t value sig.	Bengali Female (n=155)		Bhumij Female (n=160)		t value sig.
	Mean	±SD	Mean	±SD		Mean	±SD	Mean	±SD	
BMI	21.90	3.51	21.88	3.00	.055	21.98	4.19	21.46	3.52	1.186
WC	76.73	9.93	75.58	9.29	1.060	75.56	12.24	71.95	9.39	2.940**
WHR	0.88	0.07	0.85	0.06	3.082**	0.82	0.07	0.79	0.05	4.026***
BF%	21.34	6.27	19.27	6.35	2.891**	32.24	5.83	30.08	5.53	3.379**
FM	12.58	5.06	11.66	5.23	1.566	16.12	5.51	14.58	4.73	2.664**
SBP	130.45	14.20	129.09	13.40	.865	124.35	16.12	123.97	15.24	.215
DBP	80.32	11.06	79.14	11.12	.930	78.15	11.16	79.02	10.82	.688
TC	136.96	29.56	134.70	29.14	.679	130.88	28.159	142.10	27.60	3.571***
TG	141.73	71.97	100.71	66.59	5.203***	97.05	55.68	71.96	42.44	4.507***
LDLc	78.01	24.64	70.88	23.97	2.580**	75.68	26.21	82.09	24.18	2.256*
HDLc	31.74	8.41	43.63	11.29	10.534***	36.13	8.85	46.26	9.70	9.647***
VLDLc	28.34	14.39	20.14	13.31	5.203***	19.41	11.136	14.39	8.48	4.507***
FBG	106.4	15.46	106.09	17.67	.203	110.98	43.52	101.10	15.99	2.689**

* p<0.05, ** p<0.01, ***p<0.000"

Table 2.2. Mean comparison of CVD risk factors across FHD among males

CVD risk factors	FHD	Bengali Male (106 vs 52)		t value Sig.	Bhumij Male (111 vs 41)		t value Sig.
		Mean	±SD		Mean	±SD	
BMI	Negative	21.33	3.28	2.957**	21.58	2.95	1.996*
	Positive	23.05	3.71		22.67	3.04	
WC	Negative	74.92	9.19	3.392**	75.15	9.07	.920
	Positive	80.44	10.42		76.72	9.91	
WHR	Negative	0.873	0.07	2.163*	0.857	0.062	.037
	Positive	0.899	0.07		0.858	0.072	
BF%	Negative	20.23	6.61	3.280**	18.79	6.55	1.535
	Positive	23.61	4.81		20.57	5.64	
FM	Negative	11.66	5.06	3.351**	11.28	5.27	1.472
	Positive	14.45	4.56		12.69	5.05	
SBP	Negative	129.95	15.38	-.626	128.52	13.57	.861
	Positive	131.46	11.49		130.63	12.97	
DBP	Negative	78.79	11.02	2.514*	79.05	11.51	.165
	Positive	83.42	10.57		79.39	10.12	
TC	Negative	132.93	29.40	2.487*	131.23	29.90	2.451*
	Positive	145.18	28.41		144.08	24.97	
TG	Negative	131.75	63.64	2.531*	94.97	64.18	1.760
	Positive	162.07	83.53		116.24	71.20	
HDLc	Negative	31.65	8.55	.204	43.30	11.81	.583
	Positive	31.94	8.20		44.51	9.77	
LDLc	Negative	75.79	52.0	1.624	69.15	23.50	1.472

	Positive	**82.53**	**23.31**		**75.57**	**24.88**	
VLDLc	**Negative**	**26.35**	**12.72**	**2.531***	**18.99**	**12.83**	**1.760**
	Positive	**32.41**	**16.70**		**23.24**	**14.24**	
FBG	**Negative**	**104.85**	**11.48**	**1.891**	**105.586**	**11.89**	**.580**
	Positive	**109.76**	**21.1**		**107.46**	**28.05**	

* p<0.05, ** p<0.01"

Table 2.3. Mean comparison of CVD risk factors across FHD among females

CVD risk factors	**FHD**	**Bengali Female (95 vs 60) Mean**	**±SD**	**t value sig.**	**Bhumij Female (116 vs 44) Mean**	**±SD**	**t value sig.**
BMI	**Negative**	**21.60**	**4.40**	**1.425**	**21.01**	**3.43**	**2.686****
	Positive	**22.58**	**3.80**		**22.65**	**3.51**	
WC	**Negative**	**74.88**	**12.32**	**.869**	**70.67**	**9.06**	**2.857****
	Positive	**76.37**	**12.13**		**75.32**	**9.51**	
WHR	**Negative**	**0.822**	**0.06**	**.848**	**0.791**	**0.06**	**1.917**
	Positive	**0.832**	**0.07**		**0.811**	**0.06**	
BF%	**Negative**	**31.94**	**6.00**	**.808**	**29.50**	**5.32**	**2.168***
	Positive	**32.75**	**5.58**		**31.60**	**5.83**	
FM	**Negative**	**15.75**	**5.75**	**1.053**	**13.90**	**4.44**	**3.008****
	Positive	**16.70**	**5.10**		**16.36**	**5.06**	
SBP	**Negative**	**122.22**	**13.88**	**2.089***	**123.48**	**15.66**	**.653**
	Positive	**127.72**	**18.78**		**125.25**	**14.18**	
DBP	**Negative**	**76.41**	**10.69**	**2.379***	**78.85**	**11.48**	**.313**
	Positive	**80.90**	**12.55**		**79.48**	**9.15**	

TC	**Negative**	**128.97**	**27.38**	**1.062**	**141.45**	**27.74**	**.488**
	Positive	**133.90**	**29.32**		**143.83**	**27.48**	
TG	**Negative**	**96.20**	**52.86**	**.241**	**70.22**	**41.12**	**.840**
	Positive	**98.41**	**60.31**		**76.54**	**45.89**	
HDLc	**Negative**	**35.94**	**7.70**	**.377**	**46.06**	**9.97**	**.422**
	Positive	**36.50**	**10.49**		**46.79**	**9.01**	
LDLc	**Negative**	**73.94**	**24.07**	**1.043**	**81.85**	**24.20**	**.176**
	Positive	**78.44**	**28.41**		**82.64**	**24.41**	
VLDLc	**Negative**	**19.24**	**10.57**	**.241**	**14.04**	**8.22**	**.840**
	Positive	**19.68**	**12.06**		**15.30**	**9.17**	
FBG	**Negative**	**108.37**	**30.04**	**.936**	**99.46**	**12.28**	**2.130***
	Positive	**115.10**	**58.97**		**105.43**	**22.72**	

* $p<0.05$, ** $p<0.01$

Table 2.4. Classification of the risk factors on the basis of NCEP ATP III for metabolic syndrome

Mets criteria	Bengali Male (%)	Bhumij Male (%)	Bengali Female (%)	Bhumij Female (%)
Raised BP	**29.1**	**22.4**	**20**	**18.1**
Raised FBG	**30.4**	**31.6**	**23.9**	**21.9**
Raised WC	**12.7**	**7.2**	**38.1**	**22.5**
Raised TG	**36.7**	**21.7**	**11.6**	**6.9**
Reduced HDLc	**43.5**	**38.2**	**68.6**	**60.6**

Table 3.1. Multivariate analysis of CVD risk factors by FHD among pooled male participants

	Family History of Disease							
	Negative (217)		Positive (93)		Wilk's	F value	p	η^2
	Mean	±SD	Mean	±SD				
CVD risk factor					**0.914**	**2.311**	**p<0.05**	**0.086**
BMI	**21.46**	**3.11**	**22.88**	**3.42**		**12.753**	**p<0.01**	**0.040**
WC	**75.04**	**9.11**	**78.80**	**10.31**		**9.818**	**p<0.01**	**0.031**
WHR	**0.87**	**0.06**	**0.88**	**0.07**		**2.790**	**0.096**	**0.009**
BF%	**19.49**	**6.60**	**22.27**	**5.38**		**11.719**	**p<0.01**	**0.037**
FM	**11.47**	**5.16**	**13.67**	**4.83**		**11.654**	**p<0.01**	**0.037**
SBP	**129.22**	**14.47**	**131.10**	**12.10**		**1.085**	**0.298**	**0.004**
DBP	**78.93**	**11.25**	**81.65**	**10.51**		**3.724**	**0.055**	**0.012**
TC	**132.06**	**29.60**	**144.69**	**26.81**		**12.227**	**p<0.01**	**0.038**
TG	**112.94**	**66.38**	**141.87**	**81.23**		**9.453**	**p<0.01**	**0.000**
LDLc	**72.39**	**24.45**	**79.46**	**24.14**		**4.827**	**p<0.05**	**0.015**
HDLc	**37.61**	**11.87**	**37.48**	**10.87**		**0.334**	**0.564**	**0.001**
VLDLc	**22.58**	**13.27**	**28.37**	**16.24**		**9.453**	**p<0.01**	**0.030**
FBG	**105.23**	**11.67**	**108.75**	**24.32**		**2.923**	**0.088**	**0.009**
Ethnicity (covariate)					**0.644**		**p<0.01**	**0.356**

Table 3.2. Multivariate analysis of CVD risk factors by FHD among pooled female participants

	Family History of Disease							
	Negative (211)		Positive (104)		Wilk's	F value	p	η^2
	Mean	±SD	Mean	±SD				
CVD risk factor					0.958		0.370	0.042
BMI	21.27	3.90	22.61	3.66		7.821	p<0.01	0.024
WC	72.56	10.83	76.08	11.07		5.649	p<0.05	0.018
WHR	0.81	0.06	0.82	0.07		3.416	0.064	0.011
BF%	30.60	5.75	32.25	5.68		4.158	p<0.05	0.013
FM	14.73	5.14	16.56	5.06		7.287	p<0.01	0.023
SBP	122.91	14.86	126.67	16.95		3.991	p<0.05	0.013
DBP	77.75	11.14	80.29	11.21		3.965	p<0.05	0.013
TC	135.83	28.21	138.11	28.84		1.243	0.266	0.004
TG	81.91	48.42	89.16	55.50		0.478	0.490	0.002
LDLc	78.30	24.69	80.22	26.74		0.825	0.364	0.003
HDLc	41.51	10.32	40.85	11.09		0.318	0.573	0.001
VLDLc	16.38	9.68	17.83	11.10		0.478	0.490	0.002
FBG	103.47	22.50	111.01	47.23		2.638	0.105	0.008
Ethnicity (covariate)					0.653		p<0.01	0.347

Table 4. Multivariate analysis of CVD risk factors by FHD among pooled participants

	Family History of Disease							
	Negative (428)		Positive (197)		Wilk's	F value	p	η^2
	Mean	±SD	Mean	±SD				
CVD risk factor					**0.954**		**0.004**	**0.046**
BMI	**21.37**	**3.52**	**22.74**	**3.54**		**19.871**	**0.000**	**0.031**
WC	**73.82**	**10.06**	**77.36**	**10.78**		**15.116**	**0.000**	**0.024**
WHR	**0.83**	**0.07**	**0.85**	**0.07**		**6.305**	**0.012**	**0.010**
BF%	**24.97**	**8.32**	**27.54**	**7.45**		**15.139**	**0.000**	**0.024**
FM	**13.08**	**5.40**	**15.20**	**5.15**		**18.825**	**0.000**	**0.029**
SBP	**126.11**	**14.98**	**128.76**	**14.99**		**4.779**	**0.029**	**0.008**
DBP	**78.35**	**11.20**	**80.93**	**10.88**		**7.510**	**0.006**	**0.012**
TC	**133.92**	**28.95**	**141.22**	**28.02**		**9.931**	**0.002**	**0.016**
TG	**97.64**	**60.19**	**114.04**	**73.56**		**7.890**	**0.005**	**0.013**
LDLc	**75.312**	**24.72**	**79.86**	**25.48**		**4.188**	**0.041**	**0.007**
HDLc	**39.53**	**11.09**	**39.26**	**11.09**		**0.706**	**0.401**	**0.001**
VLDLc	**19.59**	**12.03**	**22.80**	**14.71**		**7.890**	**0.005**	**0.013**
FBG	**104.36**	**17.85**	**109.94**	**38.09**		**5.222**	**0.023**	**0.008**
Covariate								
Ethnicity					**0.670**		**0.000**	**0.330**
Sex					**0.196**		**0.000**	**0.804**