Extensive search has been carried out through pubmed, epidemiological journals and general internet to identify the scientific publications concerning the various cardiovascular risk factors and the familial aggregation of blood pressure among different populations until April, 2012. The present study has included a wide range of references from 1942 to 2012 to discuss the various aspects related to cardiovascular diseases.

2.1. Cardiovascular Diseases

It is now established that non-communicable diseases especially cardiovascular diseases (CVD) are major causes of death and disability in low income countries, including India (Gersh et al., 2010). Over 82% of the mortality burden is caused by ischaemic or coronary heart disease, stroke (both hemorrhagic and ischaemic), hypertensive heart disease or congestive heart failure. Prevalence of CVD and its risk factors is rapidly increasing (Gupta et al., 2008) and it causes major burden on healthcare systems (Reddy et al., 2005; Gupta et al., 2011). In 2008, CVD caused an estimated 17 million deaths and led to 151 million disability/adjusted life years (DALYs) representing 10% of all DALYs lost in that year. Behavioural risk factors such as physical inactivity, tobacco use and unhealthy diet explain nearly 80% of the CVD burden (Gaziano et al., 2010).

According to Gupta et al. (2006) a wide range of variation was seen in mortality rates due to CVD in various states of Punjab as shown in figure 2.1. States of Goa, Punjab, Tamil Nadu and Andhra Pradesh have high cardiovascular disease mortality rates, while, sub-Himalayan states of Nagaland, Meghalaya, Himachal Pradesh and Sikkim are low cardiovascular disease mortality regions.
2.2. Cardiovascular Risk Factors

There is an incomplete understanding of the precise etiology and mechanisms that lead to study the epidemiology of CVD among Indians. Some of the risk factors of CVD have higher prevalence among the Asian Indian population, including insulin resistance, glucose intolerance, central or abdominal obesity, hypertriglyceridaemia and increased level of low density lipoprotein cholesterol (LDL-c) (McKeigue et al., 1991; Enas et al.,
1992; Gupta et al., 1995; Enas, 2000, Kusuma and Das, 2008). Factors such as genetic predisposition, which appears to be mediated by elevated levels of lipoprotein (a) and apolipoprotein (E), as well as changing lifestyle (including physical inactivity) may also increase the coronary risk profile among Indians (Ghosh et al., 2003). Reviews of epidemiological studies suggest that all the major risk factors are increasing in India (Pais and George, 1998; Reddy et al., 2002; Gupta, 2004; Gupta et al., 2008; Tandon et al., 2011; Gupta and Kapoor, 2012).

2.2.1. Non-modifiable Risk Factors:

2.2.1.1. Age and gender: Cardiovascular risk factors in men and women are known to increase with age, hypertension, blood lipids, glucose levels and central obesity, but, there is a marked difference between the two sexes (Sakurai et al., 2006; Tyagi et al., 2008). The incidence of CVD has been found to be three times higher and mortality to be five times higher among men than the women. But with the increase in the age it increases more sharply in women. The differences in sexes are particularly due to the factors like high density lipoproteins (HDL), blood pressure, body mass index (BMI) and diabetes (Thelle, 1990; Thom et al., 1992; Kuhn and Rackley, 1993; Rich-Edwards et al., 1995; Njolstad et al., 1996; Jousilathi et al., 1999; Sarraf-Zadegan et al., 1999; Hatmi et al., 2007; Rosamond et al., 2007; Andersen et al., 2010; Ghosh et al., 2010). Wang et al. (2012) assessed the age and gender specific population attributed risks (PARs) for cardiovascular diseases and found higher prevalence in women than in men. This is due to the low levels of high density lipoproteins (HDL) secretions in post-menopausal women.

2.2.1.2. Ethnicity: According to Bhopal et al. (2002), ethnicity means group the group a person belongs to as a result of mix of cultural factors including language, diet, religion and ancestry. There is considerable variation in cardiovascular disease distribution between different ethnic groups especially between traditional and non-traditional societies. This variation of CVD may be due to socio-cultural factors rather than genetic background (Dressler, 1999; Jafar et al., 2003; Kuller, 2004; Dwivedi and Beevers, 2009; Tungdim and Kapoor, 2009; Fernandes et al., 2011).
Bhopal *et al.* (2002) studied the ethnic and socio-economic coronary heart diseases, diabetes and risk factors in Europeans and South Asian population. The authors concluded that the inequality pattern of coronary heart disease (CHD), diabetes and risk factors in Europeans is being established in South Asian men and women at different pace in different sub groups. They further suggested that future studies of inequalities should be large and studied in separate populations, such as Indian, Pakistani and Bangladeshi populations to track changes over time.

Chaturvedi (2003) studied the ethnic differences in cardiovascular diseases (CVD) among South Asians (Mostly from Indian origins), Europeans and Caribbean’s. He found that South Asians have increased risk of heart diseases as compared to Europeans and Caribbean’s have low risk of heart diseases as compared to Europeans. He concluded from his study that these differences are due to increased level of insulin resistance and associated factors such as inflation and endothelial dysfunction, adaptation to western lifestyle and dietary factors like consumption of ghee and raised level of trans fatty acid and lower level of linoleic and linolenic acids in adipose tissue, all of which adversely effect to the risk of CVD.

Sidhu *et al.* (2005) conducted the study on 2000 adult Punjabi females, from the residential areas of Amritsar, Hoshiarpur, Ludhiana and Pathankot cities of Punjab in India. The information regarding age, occupation, income, education, caste and family history was collected and blood pressure was measured and found that hypertension was maximum in Bania followed by Arora, Jat Sikh and then Sikh Harijan females, respectively. The difference between the four caste groups was statistically significant ($\chi^2= 34.3$, df = 3, P<0.01). The reason for Bania females having higher prevalence of hypertension was higher socio-economic status of this group, sedentary lifestyle, increased level of stress, changes in dietary practices and heredity factors as compared to other groups. Sikh Harijan females had minimum hypertension because they were involved in manual labour (physical activity) and less obesogenic diet. The Jat Sikh and Sikh Harijan females, both had great physical activity but the hypertension had higher prevalence in Jat Sikh population because of good diet supplements to the Jat Sikh females. Thus, they concluded that the health of Punjabi population has been severely affected by socio-economic and demographic developments.
Holland et al. (2011) studied the prevalence of coronary heart disease (CHD), stroke and peripheral vascular disease (PVD) across Asian-American subgroups (Asian Indian, Chinese, Filipino, Japanese, Korean and Vietnamese) and non-Hispanic white subjects and found that there is a considerable heterogeneity across the Asian subgroups for the prevalence of CHD, stroke and PVD. They were found to be higher in Filipino women and men and Asian Indian men and significantly lower among Chinese men and women when compared with non-Hispanic whites.

2.2.1.3. Family aggregation and heritability:

There is much epidemiological evidence that environmental cofactors and anthropometric characteristics are directly and consistently correlated with cardiovascular diseases in developing countries (Badaruddoza and Afzal, 1999, 2000; Wang et al., 1999; Yu et al., 2000; Lasser and Breckenkamp, 2005; Tompson and Lip, 2005; Badaruddoza and Kumar, 2009). It has been reported that almost 30% of risk factors for cardiovascular diseases are accounted by genetic heritability and at least approximately 70% of risk factors are familial in nature (Brand et al., 1992; Hopper and Carlin, 1992; Knuiman et al., 1996a, b; Feuntes et al., 2000; Harrap et al., 2000). However, many authors have contradicted that to what extent the observed familial aggregation on both systolic and diastolic is due to genetic or environmental reasons. Some authors have argued that familial aggregation on diastolic blood pressure is more than systolic blood pressure or vice versa (Knuiman et al., 1995). Although the heritability of blood pressure in western population has been well described (Perusse et al., 1989; Abney et al., 2001; Bochud et al, 2005; Van-Asselt et al., 2006; Van-Rijn et al., 2007; deOliveira et al., 2008), however, there are very few studies available in Indian populations (Sidhu et al., 2004; Ghosh et al., 2010; Kumar and Badaruddoza, 2010; Badaruddoza and Pathari, 2012). In India the pattern of risk factors for cardiovascular diseases are different to cut across the cultural patterns and geographic regions. Therefore, in Indian context the paucity of family based information and complex etiology of this disease made it difficult to understand how these factors contribute to the cardiovascular diseases. Therefore, important studies related to familial aggregation and heritability of blood pressure from the present research group with other studies are mentioned below.
Badaruddoza and Sawhney (2009) studied familial aggregation of blood pressure with respect to anthropometric variables in a business community of Punjab. They evaluated a total of 75 families, constituting 305 individual from three generations such as offspring, parental and grandparental. The data were analyzed through familial correlations, multiple regressions, percent of variance and univariate analysis. The data indicated a strong familial aggregation of blood pressure in this population, especially, in offspring generation and showed that such a familial influence on blood pressure can be detected from the different anthropometric variables, genetic factors, shared household environment and age. They found that these effects were strong in SBP and moderate in DBP. SBP and DBP showed higher genetic correlation with many anthropometric characters in offspring generation as compared to other generations. These correlations were negligible in male grandparental generation. The results suggested that almost all measured variables are significant multivariate correlates with blood pressure.

Badaruddoza and Kumar (2009) studied cardiovascular risk factors and familial aggregation of blood pressure with respect to anthropometric variables in a scheduled caste population in Punjab. They studied a total of 1096 adult individuals, constituting 350 families for blood pressure, pulse rate, pulse pressure and anthropometric variables. They estimated correlation among blood pressure phenotypes with other significantly associated variables and stepwise multiple regression analysis was carried out for both offspring and parent all generations. The hypothesis for common household effects was examined by likelihood ratio tests. Almost all anthropometric variables were found to be significant with blood pressure between both generations. The percent of variance for regression ($R^2$) was found to be higher for offspring generation than for parent generation. The data indicated strong familial aggregation of blood pressure and the authors concluded that anthropometric measurements would be a useful tool for screening cardiovascular risk factors with the elevated blood pressure.

Kumar and Badaruddoza (2010) studied related familial aggregation and heritability for cardiovascular risk factors in a family based study in Punjab. The work was conducted through house to house family study among three generations such as offspring, parent and grandparent in a scheduled caste community (Ramdasia) in Punjab. A total of 1400
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individuals, constituting 380 families were surveyed for blood pressure, pulse rate, pulse pressure and anthropometric measurements to study familial aggregation and heritability for cardiovascular risk factors. The analysis represented a multivariate model which includes the each individual family data for estimation of familial correlation and heritability. The study showed that all the risk factors had positive familial correlation but magnitudes were different in various pairs of combinations. Correlations generally are higher among genetically close relatives such as brother-sisters or parent-offspring and are lower among spouses. The estimated heritabilities were 22% for systolic and 27% for diastolic blood pressure, 19% for BMI and 17% for WHR. The study concluded that these results indicate a strong familial aggregation of cardiovascular risk factors such as SBP and DBP in this population and also showed that this familial influence can be detected from anthropometric measurements and genetic closeness. Almost all anthropometric variables were found to be significantly associated with blood pressures among three generations.

Badaruddoza and Kaur (2012) studied related familial aggregation of blood pressure with respect to anthropometric variables among the Lobana (nomadic origin) population in Punjab, a North Indian state. They have studied on 505 individuals, constituting 116 families of three generations (offspring, parental and grandparental). The study represented a multivariate model analysis, which included family data with respect to blood pressure phenotypes and other metric measurements such as height, weight, body mass index, waist and hip circumferences, waist-to-hip ratio (WHR), and four skinfold measurements. A higher correlation for almost all sets of anthropometric variables with blood pressure was found among the offspring generation as compared with the parental and grandparental generations. The study confirmed that the familial aggregation of blood pressure with respect to anthropometric measurements is strong in the offspring generation. The findings suggested that sharing a household environment has a significant effect on familial aggregation especially for systolic blood pressure.

Badaruddoza and Patharia (2012) studied relative heritability of some anthropometric and physiometric phenotypes among three caste populations in Punjab. The study included a total of 150 families, 50 families each of the three castes such as Brahmin, Khatri and Bania populations which constituted a total of 517 individuals. The
anthropometric measurements taken were height, weight, waist circumference, hip circumference, biceps skinfold, triceps skinfold, suprailliac skinfold and subscapular skinfold. The physiometric variables included measurement of systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse rate. The estimation of heritability was calculated from the degree of resemblance between relatives. They found that almost all heritabilities of anthropometric and physiometric phenotypes were found significant with caste populations. Although heritabilities for some phenotypes (WHR, supra-iliac skinfold and pulse rate) were relatively low as compared to other studies in literature. Khatri population has showed greater variability for both generations whereas, Brahmin population for both generations have showed minimum heritability. Since, caste effects were significant for most of the phenotypes in comparison, therefore, the authors suggested that inter-caste differences among these castes are more prominent. However, heritabilities were different in magnitudes across the caste groups. Thus, heritability pattern of anthropometric and physiometric phenotypes observed in this study exhibited significant variations among Punjabi Brahmin, Khatri and Bania population.

Therefore, from many previous studies it has been observed that familial clustering of CVD has lead to a variety of studies on familial aggregation of cardiovascular risk factors. Almost all studies demonstrate that there is familial aggregation of cardiovascular risk factors. Still, there is a disparity over extent to which observed aggregation is due to genetic rather than environmental causes. The genetic and cultural transmission mechanisms along with shared physical and social environments are responsible to maximize similarities of individuals within families. There is ample evidence that high blood pressure tends to cluster in families. Such clustering may be due to shared genetic factors or shared environment or an interaction of both. Familial aggregation in blood pressure may vary in different populations with basic difference in genetic characteristics, culture and lifestyles (Feinleib et al., 1979; Wang et al., 1999; Guo 2000; Harrap et al., 2000; Jee et al., 2002; Yoon et al., 2002; Sneider, 2004; Scurrah et al., 2008; Ghosh et al., 2010).

In the World Health Report (2002), it was estimated that high blood pressure caused 13% of all deaths world-wide and 4.4% of disability/adjusted life years lost (DALYs). High blood pressure was estimated to account for 12.7% of deaths in men and 15.1% in
women. Genetic factors that increase susceptibility of an individual to hypertension have been studied in many populations (Jeunemaitre et al., 1992; Hata et al. 1994; Lifton, 1996). It is documented that hypertensive individuals have relatives with elevated blood pressure more commonly than in relatives of normotensive individuals indicating familial resemblance of blood pressure phenotypes. Blood pressure and obesity are complex quantitative traits and thought to be influenced by variety of physiological, behavioral and sociocultural factors (Ward, 1983, 1990; Majumdar et al., 1995; McGill, 1996). Nath et al. (2002) compared the hypertension prevalence in two populations from India and found that the observed familial resemblance of blood pressure in two populations was primarily due to cultural rather than genetic influences.

Importance of family history in assessing familial health risk does not remain constant throughout life. It changes as families grow, as family members age and increase their exposure to the environment, and as the status of their health evolves CVD. The blood pressure in childhood and adolescence is considered a predictor of elevated blood pressure later in life (Lauer et al., 1986; Bao et al., 1995) and weight loss in overweight adolescents has shown association with a decrease in blood pressure (Rocchini et al., 1988; Kelley et al., 2003).

Both genetic and environmental factors predispose individuals to cardiovascular disease. An important contribution to assess the role of environmental factors in cardiovascular diseases could arise from similarity of cardiovascular risk factors between marital partners. Spouses are usually genetically unrelated but share a common environment and if the spousal concordance for cardiovascular risk is found, then family environment might pose a threat for spouses of patient who have a high risk of developing a disease (Chow et al., 2007; Di Castelnuovo et al., 2009). Several studies have been conducted to investigate correlation between spouse and majority have found positive correlation (Garrison et al., 1979; Rao et al., 1982; Perusse et al., 1989). This is due to some environmental factors that contribute cohabitation on sharing the same lifestyle. The individuals who cohabit show concordance in cardiovascular risk factors that have association with lifestyle and such concordance should increase with duration of cohabitation. Husband and wife are not genetically related, therefore, any concordance in cardiovascular risk factors must be due to assortative mating. Knuiman
et al. (1996b) did a community based study on 1319 nuclear families and he considered SBP, DBP, BMI, triceps skinfold and cholesterol as cardiovascular risk factors and concluded that all the risk factors have shown a positive familial correlation, with correlation being lower in spouses than parent-offspring correlation. Spouse correlation showed a little variation with ages suggesting that the observed correlations are primarily due to assortative mating and not due to cohabitation. Spouse’s correlation showed increasing pattern with age. Parent-offspring showed a little variation with age. This suggests that observed correlation with CVD risk factors is primary due to genetic influence rather than environmental (Annest et al., 1983; Harrap et al., 2000).

Kaur and Singh (1981) studied the intra-familial correlation, involving the parent and offspring, of body measurements and heritability in a rural Indian population and found that mean height of offspring was greater than parent but the skinfold at different sites were higher in parents than offspring. Intra-familial correlation of different body measurements were low and statistically non-significant in husband-wife correlation. Parent-offspring correlation was highest in longitudinal measurements like height, sitting height and arm length but lowest in arm girth and skinfolds. Sib-Sib correlation showed the highest correlation for body weight and other parameters like parent offspring correlation.

Rao et al. (1987) studied the Swedish twin family study to obtain the maximum – likelihood estimation of familial correlation considering high density lipoprotein cholesterol and total cholesterol. The study indicated that there is strong familial resemblance for both traits, but, little cross-trait resemblance. A large number of investigations indicated that in spite of large environmental effects on blood pressure 20-40% of this variability within a population is due to polygenic factors (Feinleib et al., 1979). The familial nature and the inheritance of blood pressure have been under investigation for several decades. The overall relationship between blood pressure of first degree relatives and their propositi strongly supports the hypothesis that blood pressure levels in the general population are determined multifactorially and that a family factor is involved (Miall et al., 1967).
Familial concentration of blood pressure among genetically close relatives such as parent and offspring, brother and sister has been suggested in many studies (Knuiman et al., 1996b; Feng et al., 2008; Badaruddoza and Sawhney, 2009; Kumar and Badaruddoza, 2010; Badarudoza et al., 2011b). Hence, it appears that the degree of aggregation of blood pressures with respect to anthropometric measurements and other physiological traits have been well reported from western populations as well as Indian populations (Livshits et al., 1999; Charturvedei, 2003; Wada et al., 2006). There is strong evidence, from investigations in genetic epidemiology, about the importance of the family history in blood pressure values (Gu et al., 1998; Levy et al., 2000; Arya et al., 2002; Austin et al., 2004; Li et al., 2006; Campos-Rodriguez et al., 2007). This influence is the result of the sharing of genes and a common environment of the members of the same family (Campos-Rodriguez et al., 2007). It is estimated that the 25% (Campos-Rodriguez et al., 2007) to 58% (Arya et al., 2002) of the blood pressure variability can be determined genetically. Sive et al. (1971) examined the role of 18 biological and environmental variables in blood pressure variation among a variety of Israeli groups and reported 16.6% to 29% of variation in SBP are due to these variables and about 80% due to age, pulse rate, and weight/height. India, with a homogenous population and high cardiovascular disease morbidity and mortality, is well suited for family studies aimed to investigate the familial aggregation of blood pressure.

Studies conducted on populations with very diverse geographical, cultural and environments reported familial aggregation of blood pressure (Tyroler, 1977). Genetic differences among individuals are ascertained with estimates of the fraction of phenotypic variation in blood pressure (Borhani, 1979). They concluded that unspecified environmental factors are the main determinants of blood pressure. Both genetic and environmental influences in the variations of blood pressure have been thoroughly studied by many investigators (Havlik et al., 1979; Canessa et al., 1980; Delles et al., 2010; Fung et al., 2011; Wang et al., 2011). Some other researchers used path analysis to separate the genetic and environmental influences in blood pressure variations (Weinberg et al., 1979; Krieger et al., 1980; Morton et al., 1980; Perusse et al, 1989; Rice et al., 1989, 1992).
The results of a study from French-Canadian descent population and certain other studies indicated that about half of the total phenotypic variation in blood pressure is due to familial factors, whereas the remaining one half is due to the shared genes and environment (Biron et al., 1976, 1977; Brion and Mongeau, 1978; Annest et al., 1979a, b; Mongeau and Biron, 1981). In subjects of all ages, weight is probably the most important correlate of blood pressure. The familial aggregation of blood pressure may therefore, to a certain extent, be owing to the familial aggregation of obesity. Schieken et al. (1992) addressed this question in a pediatric population of 11 years old twins. The study of the genetics of the mechanisms involved in blood pressure regulation in children might bring us closer to the casual mechanisms (Sneider, 2004).

Wang et al. (1999) investigated 1183 Chinese nuclear families having mother, father and first two children and assessed the SBP and DBP of second sibling in relation to parents and first sibling and showed significant correlation among mother, father and first sibling with each other and independently with second sibling. The rate of high SBP in second sibling was lowest (2.3%) when both parents and first sibling had low SBP. Fuentes et al. (2000) studied 184 families in Finnish population to evaluate the association of environmental factors and familial aggregation of blood pressure to study genetics of high blood pressure in families. Significant results were found for mother-offspring correlation of SBP (r=0.18) and father offspring for MBP (r=0.20). The offspring had highest quartile of SBP and MBP when mother had history of high blood pressure when compared with offspring of mother without the history of hypertension.

Harrap et al. (2000) studied 783 families each comprising both parents (40-70 years) and atleast one adult offspring (18-30 years), were recruited in Melbourne, Australia to explore the familial pattern of cardiovascular risk factors. Both generations included 461 monozygotic and dizygotic twins as pairs or singletons. All traits showed evidence for additive genetic variation, explaining from 55% (height) to 26% (pulse) of age- and sex-adjusted variance. An effect persisting into adulthood of shared family environment during cohabitation explained from 39% (BMI) to 13% (SBP) of variance. The shared environmental effects were strongest within twin pairs, less so for sibling pairs, and least for parent-offspring pairs.
Jee et al. (2002) studied 1891 Koreans families for familial correlation of risk factors. SBP, DBP, BMI total cholesterol showed highest correlation in parent-offsprings i.e. 0.2 for BMI, 0.11 for SBP and 0.17 for DBP. The lowest correlation was seen in spouse (mother-father). Cui et al. (2002) studied 767 nuclear families having total number of 2912 individuals including 66 pairs of monozygotic and 84 pairs of dizygotic twins, respectively. The cross-trait correlation coefficient of SBP, DBP and BMI were found to have greatest magnitude of correlation among monozygotic twins and least among spouses.

In India, studies on inheritance of height and other anthropometric traits have been reported in different ethnic communities (Kaur and Singh, 1981; Rama Devi and Reddy, 1982, 1983; Poosha et al., 1984; Sharma et al., 1984; Byard et al., 1985; Kapoor et al., 1985; Dasgupta et al., 1997; Susanne, 1997; Kumar and Badaruddoza, 2010). These studies have revealed differences in heritability estimates and its relative contributions, genetic and environmental influences. These are based on different populations, but comparison of heritability estimates in the same ethnic population, but, living in different regions is of little importance. Such studies help to detect the consistency in the heritability estimates in the space and time to detect the differences due to population structure or assessing the changes in environmental influences. Another approach to investigate the age-dependency of genetic and environmental effects is to compare parent-offspring data with data from siblings. If there are age-dependent genetic or environmental effects on the phenotype, one would expect the parent-offspring correlation to be lower than siblings.

According to Kaur and Singh (1981), the midparent-offspring correlation had the same direction and variability as the parent-offspring correlation but consistently higher heritability of body measurement as given by the midparent–offspring regression ranged 0.15-0.92 for different body measurements. Heritability was found highest for stature and arm length but lowest for skinfolds. Rice et al. (1992) reported higher cultural heritability estimates for SBP (about 40%) than for DBP (15%). They also reported that path analysis of blood pressure suggested inbreeding effects with genetic variance for SBP being lower in the sample that included inbred families.
Annest et al. (1979b) reported heritability for SBP of 0.34 and proportion of variability attributed to be 0.11. In terms of correlation between full-sibs, this includes 61% attributed to shared genes and 39% shared environment. Rotimi et al. (1999) studied 510 families including 1552 individuals (320 father, 370 mother, 475 son, and 387 daughters) in Nigerian families to study heritability ($h^2$) for blood pressure measurements that were adjusted for the effect of age, separately for father, mother, sons and daughter to accommodate the gender and generational heterogeneity. They concluded low prevalence of obesity in these populations. Mothers were 2 BMI units heavier then father, and value of SBP and DBP were not significantly different in these two groups. Whereas, in offspring generation daughters are found heavier than sons and DBP is 2 mm Hg units higher in daughters and borderline significant ($p= 0.05$). The maximum heritability was estimated as 45% for SBP and 43% for DBP and these values were estimates of both genetic and familial environment factors and non-significant spouse co-relation suggested that the majority of the effect is due to shared genetic factors.

Saadat et al. (2001) investigated the genetic influence variation in blood pressure by performing heritability analysis on Iranian population and found that heritability of SBP (0.58) is remarkably higher in Iranian population than western population but DBP (0.30) and MBP (0.60) variability is almost same as studied from other population.

Raychaudhuri et al. (2003) estimated the heritability of height and weight in Mahishya caste of Chakpota village in West Bengal. The authors concluded from the inter-familial correlation that most of the correlations are statistically significant ($p<0.05$) except father-mother. Sibling correlation for height was found to be highest for sister-sister combination followed by brother-brother combination. All heritabilities were higher for stature (62%) than weight (54%). The authors also did the comparison between Mahishya caste of Chakpota village with south 24 Pargana districts about 100 km away and found differences of heritabilities. Weight showed more heritabilities (54%) in this region than 24 Parganas suggesting the role of environment in heritability.

de Oliveira et al. (2008) estimated ‘heritability’ in the study and used the midparent-offspring model where ‘heritability’ ($h^2$) was equivalent to regression co-efficient (b).
The regression sum of square (RSS) and total sum of squares (TSS) ratio were also calculated both for mid parent-offspring and single parent-offspring. This ratio was considered as a measure of ‘heritability’ in the study with consideration that RSS is the variation due to genetic factor and the TSS is due to genetic and other additive factor. Heritability estimates for cardiovascular risk factor traits were all high, ranging from 26 to 51% and BMI was the highest. All heritability estimates were highly significant ($p<0.0001$). Heritabilities of systolic and diastolic blood pressure were 15% and 16.4%, waist circumference - 26.1%, triglycerides - 25.7%, fasting glucose - 32.8%, HDL (c) - 31.2%, TC - 28.6%, LDL (c) - 26.3%, and BMI - 39.1%.

Ghosh et al. (2010) studied 24 nuclear families in Calcutta, India. Only first degree relatives are included in the study of proband. Heritability of anthropometric and blood pressure measurements were analyzed through midparent-offspring and single parent-offspring model and heritability for SBP in mid-parent offspring model was 0.16 to 0.44 and 0.05 to 0.54 for single parent-offspring model. Thus, indicating high blood pressure measurement aggregation in family.

Wu et al. (2011) estimated the genetic and environmental influences on blood pressure and body mass index by twin study analysis in Chinese population. They included 1243 monozygotic and 833 dizygotic Han Chinese twins. The authors found 46% and 30% heritability for SBP and DBP, respectively. They concluded that the positive correlations of BMI with SBP ($r=0.26$) and with DBP ($r=0.27$) were due to genetic factors (approximately 85%). Genetic factors accounted for 6% and 7% of total variance for SBP and DBP, respectively. The gene-obesity interaction analysis showed that environmental influences on SBP increased with increasing levels of BMI, resulting in lower heritability at higher BMI levels, whereas for DBP the heritability remained unchanged at higher BMI levels.

2.2.2. Modifiable risk factors

2.2.2.1. Blood pressure: It has been well documented that hypertension is the commonest cardiovascular disorder affecting at least 20% of adult populations in several countries and an important cause for cardiovascular mortality accounting for 20-30% of all deaths (Saadat et al., 2001; Messner et al., 2003;; Banik, 2007; Badaruddoza and
Sawhney, 2009; Badaruddoza and Kumar, 2009; Pawar et al., 2010; Mahmood et al., 2011; Badaruddoza and Patharia, 2012). It is posing a major public health burden to societies in epidemiological, socio-economic and demographic transition, causing two fold higher risk of developing coronary heart disease, four times higher risk of congestive heart failure and seven times higher risk of cardiovascular disease as compared to normotensive individuals (Abate and Chandalia, 2003; Gaziano et al., 2006; Ulasi et al., 2011).

It is estimated that the worldwide prevalence of hypertension would increase from 26.4% in 2000 to 29.2% in 2025 (Kearney et al., 2005). Hypertension is an important risk factor for cardiovascular disease and has become a major global burden on public health (Lawes et al., 2006; Wong et al., 2007; Ong et al., 2008).

Erem et al. (2009) estimated the prevalence, awareness and control of prehypertension and hypertension among Turkish adults and its associations with demographic factors, socioeconomic factors, family history of selected medical conditions and lifestyle factors. The prevalence of hypertension was 44.0% (46.1% in women and 41.6% in men) and of prehypertension was 14.5% (12.6% in women and 16.8% in men). Overall, only 41% of the hypertensive individuals had been previously diagnosed. The prevalence of hypertension increased with age, being highest in the 60-69 year old age group (84.4%) but lower again in the 70 years above age group. The prevalence was 16.9% in the 20-29 year old age group. Through multinomial logistic regression analysis, the hypertension was found to be significantly associated with age, male gender, BMI, low education level, nonsmoking, positive family history of selected medical conditions, occupation, and parity. They concluded that patients who are unaware of their status and treated uncontrolled hypertensives are at high risk of early cardiovascular morbidity and mortality.

Yu et al. (2008) estimated the prevalence of pre-hypertension and identified its risk factors among Chinese adults. They sampled 15,540 Chinese adults aged 35 to 74 years and observed that overall, 21.9% of Chinese adults had prehypertension. The prevalences were 25.7% in men and 18.0% in women. The prevalence of prehypertension was higher among residents in northern China compared with their
counterparts in southern China. They also found that the pre-hypertension was high among men and women who were overweight (38.4% and 27.8%) as well as with central obesity (37.8% and 25.9%). Multivariate analysis revealed that increased BMI, waist circumference, rural and urban residences were associated with prehypertension. High odds ratios of prehypertension were found in overweight and central obese adults.

Pang et al. (2010) estimated the prevalence of hypertension and associated factors among older rural adults in Liaoning Province, China. Overall, the prevalence rates of hypertension were 57%, 64.4% and 64.9% for the age groups 60–69, 70–79 and above 80 years, respectively. The prevalence of hypertension was positively correlated with age, female gender, Mongolian ethnicity, overweight and obesity, smoking and drinking habits, whereas income level was a protective factor for hypertension. The rates of awareness, treatment and control among older rural adults were very low (35.2%, 28.7% and 1.0%, respectively).

**Hypertension in India:** A recent systematic review reported that the prevalence of high blood pressure in low and middle-income countries is coming at par with high income countries (Pereira et al., 2009). The prevalence of hypertension varies considerably from one region of India to another. Yet no nationwide epidemiological studies to determine the prevalence of hypertension have been carried out. Sporadic studies from different parts of the country provided data on the epidemiology of hypertension in India. Chopra and Chopra (1942) reported the first epidemiological study on hypertension in urban north India, and reported that mean blood pressure of Indians was less than that in Europeans and the problems of high blood pressure was not that much prevalent, following which, many studies in urban and rural areas of India have been carried out. Vakil (1949) reported high incidence of hypertension among urban Indians, due to demographic changes and culture transition.

Indian urban population studies in the mid-1950s used the standardized World Health Organization (1959) guidelines (hypertension diagnosed if SBP ≥160 and/or DBP ≥95 mm Hg), have shown increasing trend in hypertension prevalence. Studies from Agra (Mathur and Sapru, 1963) and Rohtak (Gupta et al., 1978) using WHO criteria showed prevalence 4.35% and 6.43%, respectively. A majority of the surveys in urban areas
using the criteria of $\geq 160/95$ mmHg, showed a prevalence rate of 6-13 per cent, except a few, which reported a prevalence of 15% (Malhotra, 1970; Dalal, 1979). The Jaipur Heart Watch study (Gupta et al., 2002) and the Chennai Urban Rural Epidemiology Study (CURES) (Mohan et al., 2007) reported the prevalence of hypertension to be 37% and 20% using the JNC-VII guidelines (hypertension diagnosed if SBP $\geq 140$ and/or DBP $\geq 90$ mm Hg) (Chobanian et al., 2003). The term 'prehypertension' (SBP range of 120 to 139 mm Hg or DBP range of 80 to 89 mm Hg) represents the onset of arterial hypertension and thus may be considered a starting point in the occurrence cardiovascular disease (Chobanian et al., 2003; Russell et al., 2004). There is strong correlation between urbanization and increase in hypertension prevalence ($r = 0.92$, $P < 0.01$). According to 2001 census, there are 600 million adults in India, of whom 420 millions are in rural and 180 million in urban areas and the hypertensive in India shall be 31.5 million rural and 34 million urban subjects total constitute 65.5 millions and 70% them would be at stage-I hypertension (Gupta, 2004). Pooling of epidemiological data showed that hypertension was present in at least 25% of the urban and 10% of the rural adult population in India (Gupta, 2007). So, it could be hypothesized that these socio demographic and lifestyle factor are concentrating the hypertension epidemic currently sweeping India and other developing countries (Pradeepa and Mohan, 2008).

Reddy and Prabhu (2005) studied the prevalence of hypertension and its risk factors among adults in urban Andhra population. They found the overall prevalence of hypertension to be 8.6%. Out of the total hypertensives, 83.7% were aware of their hypertension; all of those aware were under treatment; among the treated, only 41.7% had satisfactory control of their hypertension. Higher prevalence of hypertension was found with history of cardiovascular events (50.0%), diabetes mellitus (33.3%), family history of hypertension (23.3%), smoking (22.4%), age more than 50 years (22.2%), alcohol intake (20.0%), lack of physical exercise (15.8%), BMI $>$ 25 (14.9%), non-vegetarian diet (8.8%) and saturated fat intake (8.8%). The mean systolic as well as diastolic blood pressures were found to be higher among men, higher age groups, and in business occupation of the respondents.

Mohan et al. (2007) studied the prevalence of hypertension in Chennai representing urban south Indian population. The overall prevalence of hypertension in the study
population was 20% and it was higher in men than in women (men: 23.2%, women: 17.1%, p<0.001). The prevalence of hypertension steadily increased with age in both sexes and was 3.8% in men and 3.1% in women at the age group of 20-29 years, which increased rapidly and reached a prevalence of 50.8% in men and 51% in women at the age of 60 years and above.

Yadav et al. (2008) studied the prevalence of pre-hypertension and hypertension, as well as their association with cardiovascular risk factors, in a north Indian upper socio-economic population. They recruited a total of 1746 adults (age ≥30 years) urban high-income group of Lucknow. They analyzed the variables contributing significantly to pre-hypertension and hypertension by multiple logistic regression analysis and observed that sex adjusted prevalence of prehypertension and hypertension were 32.3% and 32.2%. In contrast to hypertension, which was highest in the age group 60-69 years (64%), prehypertension was highest (36%) in the group 30-39 years. Two or more of the cardiovascular risk factors were present in a higher proportion of hypertensive (66%) and pre-hypertensive (56%) compared to normotensive subjects (39%).

According to Todkar et al. (2009) the overall prevalence of hypertension in the rural Maharashtra population is 7.24%. The prevalence increased gradually with the increasing age and it was maximum (31.25%) in age group of 79-90 years, while it was minimum (0.41%) in age group of 19-28 years. The odds ratio was found significantly increasing gradually with increasing age. Of the total subjects with hypertension, 6.55% and 7.92% were males and females, respectively. A higher hypertension prevalence rate was observed in females as compared with males, however, this difference was statistically not significant.

Gupta and Kapoor (2012) studied blood pressure (BP) levels and their association with different indices of body fat in adult Baniyas (a caste group) (aged 25-60 years) in Delhi. The prevalence of systolic prehypertension was 49.4% and 46.0% among males and females, respectively whereas percentage of systolic hypertensive males and females was 21.8% and 7.4%, respectively. Prevalence of prehypertension and hypertension was higher among males and prevalence of obesity was higher in females.
Blood pressure was significantly correlated with all obesity measures and odds ratio of hypertension was higher in males than in females.

Durrani and Fatima (2011) determined the percentile of SBP and DBP and investigated distribution of blood pressure and its association with anthropometric variables in school children (aged 12-16 years) of Aligarh. The overall prevalence of hypertension was 9.4%, with 9.36% and 9.46% among boys and girls, respectively. Means of systolic blood and diastolic blood pressure were higher as the range of weight, height and BMI increased and blood pressure of children showed positive correlation with anthropometrics characteristics.

Gupta et al. (2011) studied the prevalence of essential hypertension among rural population of Haryana and investigated the diseases and risk factors involved in its etiology. They found that the overall prevalence of essential hypertension was 38.2%. The prevalence was 59.2% and 40.8% among males and females, respectively. Multivariate logistic regression analyses revealed that the risk of essential hypertension was significantly associated with BMI, smoking status and family history of essential hypertension.

Kaur (2012) assessed age related trends of blood pressure and prevalence of hypertension in rural and urban women and correlation of blood pressure with obesity indicators (WC, BMI and WHR) in Jat Sikh women population of Haryana. The author found an age associated increase in mean values of systolic and diastolic blood pressure in rural and urban women. Urban women showed significantly higher overall mean value of SBP, DBP and pulse rate (SBP: 133.93; DBP: 84.34; pulse rate: 81.72±6.27) as compared to rural women (SBP: 130.79; DBP: 82.81; pulse rate: 80.94±9.06). The overall prevalence of hypertension was found to be 9% in rural and 26.66% in urban women as per JNC VII criteria. Increased prominence of hypertension among urban Jat women may be attributed to their modern lifestyle having more stress, less manual work and faulty dietary habits. There was a very low awareness of hypertension in the rural subjects (37%) than their urban (72%) counterparts. Rural and urban women revealed a positive and significant association of SBP with BMI, whereas only urban women
displayed positive correlation of waist circumference with SBP (r=0.183) and DBP (r=0.151).

However, the prevalence of hypertension of various studies is not comparable because of variations in socio-economic status of the subjects, their lifestyle, genetic makeup and periodic dissimilarity, but the criterion used to define hypertension is same in all the studies. In Punjab, not enough studies have been done on the prevalence of hypertension, only a few studies have been reported (Mohan et al., 2004; Sidhu et al., 2005; Badaruddoza and Kumar, 2009; Badaruddoza and Sawhney, 2009). Sharma et al. (1985) has shown increased prevalence of hypertension in Ludhiana (Punjab). Ahlawat et al. (2002) reported the hypertension in Chandigarh as 43.7% men and 45.8% in women. But, Swami et al. (2002) also studied Chandigarh population and reported prevalence rate of 52.3% and 62.2% in males and females respectively. Thus it is evident from this meta-analysis that the prevalence of hypertension in India has been increased drastically during the last five decades (Sidhu, 2007).

Mohan et al. (2004) conducted a study to evaluate the prevalence of hypertension and obesity in apparently healthy school children, aged between 11–17 years, from rural and urban areas of Ludhiana. In urban areas, prevalence of hypertension was 6.69% and in rural areas it was 2.56% with more number of hypertensive males in both rural and urban areas. The means of both SBP and DBP of hypertensive population in both urban and rural population were significantly higher than their normotensive counterparts (urban normotensive SBP:115.48±22.74 mmHg, urban hypertensive SBP: 137.59±11.91 mmHg, rural normotensive SBP: 106.31±19.86 mmHg, rural hypertensive SBP: 131.63±10.13 mmHg, urban normotensive DBP: 74.18±17.41 mmHg, urban hypertensive DBP: 84.58±8.14 mmHg, rural normotensive DBP: 68.84±16.96 mmHg, rural hypertensive DBP: 79.15±7.41 mmHg). There was significant increase in prevalence of hypertension in both rural and urban population with increased BMI. They concluded that the prevalence of hypertension is on the rise in urban area even in younger age groups. Blood pressure is frequently elevated in obese children as compared to lean subjects. This is possibly related to their sedentary lifestyle, altered eating habits, increased fat content of diet and decreased physical activities.
Sidhu et al. (2005) observed that Punjabi population show higher incidences of hypertension like other Indian population. Prevalence of hypertension, pre-hypertension and hypertension in adult population were 25.4%, 13.56% and 61%, respectively. The data also suggested that hypertension (26.6%) is higher in females than in males (23.5%) and the difference between males and females was significant ($\chi^2=6.33$ df=1 P=0.05). Similarly stage I and II of hypertension is more prevalent in females than the males. Urban populations show higher incidence of hypertension than their rural counterparts. Subsequent studies also showed that there is gradual increasing prevalence of hypertension in rural population, also.

Many factors are considered determinant for increased blood pressure such as age, sex, salt intake, obesity, use of alcohol, smoking, sedentary lifestyle, genetic factors and genetic-environmental interactions (Saadat et al., 2001; Silventoinen et al., 2003; Garcia et al., 2004; Bose et al., 2005; Banik, 2007; de Araujo et al., 2007; Badaruddoza and Sawhney, 2009; Badaruddoza et al., 2011a,b). Elderly are especially at risk for hypertension and its related morbidity and mortality thus suggesting the role of aging in health problems (Haslam, 2008; Latiffah and Hanachi, 2008). Brown et al. (2000) havestudied significant association of obesity with high blood pressure, high blood cholesterol and low level of high density lipoprotein-cholesterol, HDL (c) in men and women in the diverse race/ethnic groups. Latiffah and Hanachi (2008) determined the association between obesity, dyslipidemia and hypertension among older people. Although hypertension is more frequently present in adults, its prevalence in childhood and adolescence may range from 2% to 13% in different parts of the world.

2.2.2.2. Anthropometric variables: Inspite of the advancements in the modern techniques, anthropometric measurements such as height, weight, BMI and WHR are traditionally important methods to study the genetic structure and prediction of risk factors of many complex diseases in human health (Seidell et al., 1989, 2001 Badaruddoza and Afzal, 2000; Reddy and Rao, 2000; Reddy et al., 2004; Sidhu et al., 2004; Badaruddoza et al., 2008, 2009; 2010; Badaruddoza and Hundal, 2009; Badaruddoza and Kumar, 2009; Klaus-Peter and Habil, 2010; Kaur and Talwar, 2011). The importance of blood pressure as a risk factor in cardiovascular disease is well established (Badaruddoza and Afzal, 2000; Saadat et al., 2001; Messner et al., 2003;
Merlo et al., 2004; Banik, 2007; Pawar et al., 2010; Mahmood et al., 2011; Ulasi et al., 2011). However, how blood pressure is influenced by different factors such as WHR, BMI, adiposity and environmental factors is the key for the understanding of coronary diseases.

World statistics from developed and many developing countries suggested that the prevalence of obesity has increased substantially in the last few decades and probably this trend will continue (WHO, 2011). Thus, increase in body fat is a serious and wider spread problem around the whole world. Obesity is associated with increased risk of hypertension, hyperlipidemia and diabetes mellitus (Nemeth et al., 1999; Brown et al., 2000; Szmodis et al., 2004; Tyagi et al., 2005; Latiffah and Hanachi, 2008; Sauvaget et al., 2008; Sinha et al., 2008; Sanyal et al., 2009).

Many prospective and cross sectional studies have been done in order to evaluate the anthropometric measurement methods to assess patients with elevated blood pressure, which is a dominant cardiovascular risk factors (Lurbe et al., 2001; Fuchs et al., 2005; Yalcin et al., 2005; Scholze et al., 2007; Wang et al., 2007; Zafar et al., 2007; Tuan et al., 2009). Many Indian studies claimed that there is a strong relation between the different anthropometric measurements and elevated blood pressure (Gupta et al., 1995; Bose et al., 2003; Shanthirani et al., 2003; Badaruddoza et al., 2008; 2010; Badaruddoza and Kumar, 2009; Badaruddoza and Sawhney, 2009; Badaruddoza and Kaur; 2012; Kaur et al., 2012). Different anthropometric measurements like BMI, waist circumference (WC), Waist to hip ratio (WHR), WHR, biceps skinfold, triceps skinfold and subscapular thickness are investigated for this purpose (Ketel et al., 2007; Sinha et al., 2008; Huxley et al., 2010; Brar and Badaruddoza, 2012).

Many studies, found that total body fat or body mass index (BMI), rather than its distribution, is the stronger predictor of metabolic risks. (Mykkanen et al., 1992; Spiegelman et al., 1992; Han et al., 2002; Bray, 2003; Ghosh and Bandyopadhyay, 2007). Body mass index (BMI) is widely used for classification of overweight and obesity, but it does not account for wide variations of the fat distribution. It was supposed to be most widely used indicator for obesity, but during recent years many epidemiological studies have suggested that measurements for abdominal obesity, such
as waist circumference (WC) or waist-to-hip ratio (WHR), might include information independent of general obesity or even be superior predictors of later adverse health effects compared to BMI (Folsom et al., 1990, 1998; Terry et al., 1992; Rimm et al., 1995; Rexrode et al., 1998; Ko et al., 1999; Savva et al., 2000; WHO, 2002; Dalton et al., 2005; Welborn et al., 2003; Janssen et al., 2004; Gupta et al., 2007; Ketel et al., 2007; Zafar et al., 2007; Badaruddoza et al., 2009, 2011a,b). Therefore, there is currently overwhelming evidence that android obesity (described as high proportional of abdominal fat) is a greater risk for cardiovascular disease (CVD) than general obesity. (Gerber and Stern, 1999; Ghosh et al., 2000; Lahti-Koski et al., 2000; Livshits and Gerber, 2001; Mueller et al., 2001; Seidell et al., 2001; Teixeria et al., 2001; Janssen et al., 2002; Badaruddoza, 2004; Benetou et al., 2004; Bose et al., 2005; Yalcin et al., 2005; Ghosh, 2007; Badaruddoza and Kumar, 2009).

WHR is so far the most widely used index of central fat distribution due to its ease in routine monitoring and assessment in patients. Although, abdominal viscera adipose tissue measured by computed tomography (CT) or magnetic resonance imaging (MRI) may more accurately reflect fat distribution and more correctly predict metabolic risks, (Fujioka et al., 1987; Peiris et al., 1989; Bjorntorp, 1990; Mueller et al., 1991; Caprio et al., 1996; Ho et al., 2003), but, some studies suggest that waist circumference (WC) is the best anthropometric index of abdominal visceral adipose tissue (Ferland et al., 1989; Despres et al., 1991; Pouliot et al., 1994; Bray, 2003, Ghosh and Bandypadhyay, 2007) and may also be the best index for predicting cardiovascular risks (Pouliot et al., 1994; Ko et al., 1997; Reeder et al., 1997). Few studies have compared the degree of association of WHR and WC with cardiovascular risks after adjustment for total fat or BMI. Also, limited data have compared the degree of correlation of WHR or WC (controlling for BMI) and BMI (controlling for WHR) with metabolic and lipid profiles. (Ho et al., 2001).

Waist circumference reflects the abdominal adiposity and total fat mass. Therefore, it is complemented with the BMI for the evaluation of obesity-associated cardiovascular diseases (CVD) risks by providing a measure of fat distribution (Ross et al., 1992; Pouliot et al., 1994; Han et al., 1995; Zhu et al., 2002, 2004). It has been also documented from various studies (Stevens et al., 1992; Pouliot et al., 1994; Lean et al., 2001).
that waist circumference (WC) is complimentary or superior to BMI in its association with CVD risks factors. It has been reported that WC cutoffs are linked with overweight and obesity. Waist circumference cut-off points associated with increased risk have been developed for adult men and women (Yalcin et al., 2005). A WC of 90 cm for men and 83 cm for women conferred a risk CVD equivalent to BMI of 25 and a WC of 100 cm for men and 93 cm for women is equivalent to CVD risk of BMI 30 in white population (Zhu et al., 2002). A large number of studies have developed reference waist circumference percentiles for children and adolescents in different countries (Moreno et al., 1999; McCarthy et al., 2001; Katzmarzyk et al., 2004; Eisenmann, 2005; Hatipoglu et al., 2008). However, many studies demonstrated that combining both WC and BMI were superior to using only one of these parameters (Chan et al., 2003; Wang et al., 2005, 2010; Badaruddoza et al., 2009, 2011; Takahashi et al., 2009). The efficiency of WC and WHR to detect the presence of CVD has been controversial (Norgan, 1994; Gallagher, 1996; Ko et al., 1999; WHO, 2002; Zhu et al., 2002; Dalton et al., 2005; Welborn et al., 2003; Grundy et al., 2004; Janssen et al., 2004; Al-lawati and Jousilahti, 2007; Huxley et al., 2008; Liu et al., 2011).

WHO has advocated that a lower limit of normal BMI in Asian Indians are more prone to diabetic having lean body mass but high upper-body adiposity. To overcome this problem, Snehalatha et al. (2003) found the cutoff values for BMI, WC and WHR by computing their risk association with diabetes and concluded the normal BMI cutoff value for men and women was 23 kg/m² and WC 85 cm for men and 80 cm for women and WHR-0.88 and 0.81 for men and women respectively. These values are significantly lower as compared to the corresponding values of whites.

Abundant literature is available related to anthropometric variables and blood pressures in world-wide populations. It is not possible to mention all these studies in this review, therefore, only important, recent and directly related works to the present study are included.
Al-Sendi et al. (2003) studied 504 school children in the age group 12-17 and suggested that WC and WHR are useful anthropometric predictors for developing high blood pressure. In view of the observed relationship between WC, Intra-abdominal fat deposition and cardiovascular disease risk factors in children, concluded that WC could be adopted as an alternative on additional measurement to BMI. Yalcin et al. (2005) studied Turkish population to identify the better anthropometric predictors closely related to blood pressure. Each subject’s weight, height, waist and hip circumference, triceps skinfold and blood pressure were measured and WHR and BMI were calculated. The authors found that the mean systolic and diastolic blood pressures were 123.49 ± 17.60 and 78.79 ± 10.37 mmHg. According to body mass index 23.7% of the subjects were obese (>29.9 kg/m$^2$). When waist circumference cut-off points were compared with waist to hip ratio the android obesity ratio was doubled (32.3% versus 16.6%). About 6.8% were not obese according to body mass index but had waist circumference measurements above the cut-off points suggesting a high cardiovascular risk. In the linear regression models, waist circumference was found to be an independent risk factor for blood pressure in men; however body mass was more important index and waist circumference somewhat less so for women.

Deshmukh et al. (2006) studied the relationship of anthropometric indicators with blood pressure levels in rural population in India and concluded that BMI, waist circumference, WHR and WHtR are important anthropometric indicators for predicting the obesity and cardiovascular risk factors. He found that all these measurements are significantly correlated to SBP and DBP except WHR and DBP. Manova et al. (2006) found an association between BMI and SBP in both genders ($r= 0.3$ in men, $r = 0.33$ in women, $p< 0.0001$) as well as between BMI and DBP ($r=0.28$ in men, $r = 0.32$ in women, $p<0.0001$) in both gender. Obese had higher prevalence of hypertension as compared with normal weight men (11.08% vs 1.81%, $p<0.0001$). Similarly, obese women also had higher prevalence of essential hypertension compared with normal weight (8.63% vs 1.48%, $p<0.0001$).

Moy et al. (2008) concluded that 44.6% of their participants had more than 10% risk for developing CHD in the next ten years. Hence, they were suitable target candidates for
the promotion of a healthy lifestyle such as smoking cessation, weight control, healthy dietary patterns and increased physical activities.

Al-Lawati et al. (2008) determined the gender-specific cut-off points for BMI, WC and WHR for Omani Arabs using current levels of risk factors for CVD and the Framingham risk score. They found a high distribution of CVD risk among Omanis. He suggested the BMI cut-off values of $>22.6 \text{ kg/m}^2$ and $>22.9 \text{ kg/m}^2$, WC of $>78.5 \text{ cm}$ and $>84.5 \text{ cm}$ and WHR of $>0.96$ and $>0.98$ for Omani men and women. They also found a larger area under the ROC curves for WHR followed by WC and BMI in both genders, suggesting WHR as a better indicator of CVD.

Badaruddoza et al. (2009) studied age specific relation of blood pressure with anthropometric variables among 19-24 years Punjabi female youth of Amritsar city. The study samples included a total of 800 Punjabi urban female youth. The results showed strong positive correlation of age and other anthropometric variables with blood pressures.

Chakraborty and Bose (2009) studied the relationship between body mass index (BMI) and percent body fat (PBF) with central adiposity measures. The study tested the relative efficacy of waist circumference (WC), hip circumference (HC), waist hip ratio (WHR) and coincity index (CI) to predict BMI and PBF among Bengalee Hindu male slum dwellers. Waist circumference showed the strongest significant ($p<0.001$) partial correlation with BMI and PBF (0.82 and 0.77, respectively). Stepwise multiple linear regression analyses revealed that WC had the strongest impact on BMI and PBF. On the other hand WHR, HC and CI had weaker effect. Moreover, WC alone accounted for about 67% and 60% of the variations, respectively, of BMI and PBF. They concluded that in Bengalee Hindu rural population, WC may be preferred over other measures of central adiposity in studies dealing with obesity and cardio vascular disease risk factors.

Badaruddoza et al. (2010) undertook cross-sectional study to assess the inter-relationship of blood pressures with BMI, WHR and subcutaneous fat among university going Punjabi Sikh and Hindu females. No significant differences of all the measured mean values of the traits have been found between these two groups. Further analysis of the data showed that BMI, WHR and skinfold measurements have significant ($p<0.05$)
effect on blood pressure phenotypes. They concluded that BMI and WHR would be good predictors for the chronic diseases like hypertension.

Bishnoi et al. (2010) estimated the correlation and regression relationship between phenotypes of blood pressure, anthropometric measurements, lifestyle factors and metabolic variables among three population groups namely, Bishnoi, Sikh and Hindu from Punjab and Rajasthan. A total of 310 females were recruited from the three populations. Females of Hindu population were at higher risk, Sikh at moderate risk and Bishnoi at lower risk in case of comparison with respect to BMI, weight, waist circumference, total-cholesterol, triglyceride, HDL and Cholesterol-HDL ratio. They concluded that CVD risk in females should not be taken casually rather this risk factor should be addressed much earlier than menopause.

Hotchkiss and Leyland (2010) investigated the relationship between body mass index (BMI), waist circumference (WC) or waist to hip ratio (WHR) and all-cause mortality or cause-specific mortality. The authors suggested that BMI-defined obesity (≥30 kg/m²) was not associated with increased risk (hazard ratio=0.93) of mortality, whereas the overweight category (25–30 kg/m²) was associated with a decreased risk (hazard ratio=0.80). In contrast, the hazard ratio for a high WC was 1.17 and a high WHR was 1.34. There was an increased risk of cardiovascular disease (CVD) mortality associated with BMI-defined obesity, a high WC and high WHR categories; the hazard ratio estimates for these were 1.36, 1.41 and 1.44, respectively. They concluded that it might be prudent not to use BMI as the sole measure to summarize body size. The alternatives WC and WHR may more clearly define the health risks associated with excess body fat accumulation. The lack of association between elevated BMI and mortality may reflect the secular decline in CVD mortality.

Taylor (2010) compared the magnitude of association of BMI, WC, WHR and skinfolds with CVD risk factors. He suggested that the measurements of central adiposity and other regional measurements (i.e. skinfold thickness) of fat mass compared with BMI were not more strongly related with CVD. The magnitude of associations of BMI with incident CHD and CVD risk factors were similar to those with the measurements of central obesity (WC, WHR, WHtR) and more direct measurement of fat mass (skinfold
thickness). He also concluded that central obesity measurements, as well as BMI, were associated with all-cause mortality.

Schneider et al. (2011) showed that measures of abdominal obesity including WHtR, WC, and WHR predict death, cardiovascular death, and cardiovascular events clearly better than BMI. In the ROC analysis, the AUCs of WHtR were significantly larger than the AUCs of the other anthropometric parameters. They suggested that WHtR is the best indicator of future cardiovascular risk and overall mortality among different measures of abdominal obesity. Although cardiovascular risk conditions at baseline accumulated among obese subjects, mediator analysis showed little or no effect modification by these baseline conditions. This indicated that incident cardiovascular events were not relevantly mediated by other baseline cardiovascular diseases and risk factors.

Badaruddoza et al. (2011b) studied association of anthropometric and metabolic variables with cardiovascular disease among urban and rural Punjabi population. This cross-sectional study was carried out on a total of 400 urban and rural origin Punjabi males (200 each from urban and rural). The anthropometric, physiometric and metabolic assessments were through standard procedures. Statistical analysis includes descriptive statistics, correlation, multivariate regression analysis and odds ratios. It was observed that males of rural population were at a higher risk to develop cardiovascular diseases compared to their urban counterparts. Rural males had significantly (p<0.001) higher mean values of cardiovascular risk factors with respect to BMI, weight, waist circumference, WHR, fasting glucose, total cholesterol, triglyceride, HDL and CHO-HDL ratio. SBP and DBP have positive association with waist-to-hip ratio, body mass index; waist circumference, skinfolds, pulse pressure, alcohol consumptions, food habit, HDL and triglyceride. They concluded that cardiovascular disease risk is found more in rural male Punjabi population due to consumption of more dairy products and leading of more sedentary lifestyle due to the overuse of mechanized substances for agriculture and personal use.

Liu et al. (2011) studied the association of BMI, WC, WHR and WHtR to predict the risk of obesity related diseases in Chinese population. They suggested that BMI, WC
and WHtR were associated with cardiovascular risk. The cut-off values of BMI in males and females were suggested to be 22.85 kg/m$^2$ and 23.30 kg/m$^2$, respectively. For WC the cut-off were 91.3 cm and 87.1 cm and for WHtR 0.51 and 0.53 in males and females, respectively. All of these parameters equally predict multiple metabolic risk factors. The optimum values can be maintained by modifications in diet and exercise.

According to Gharakhanlou et al. (2012), overweight and obesity are important public health problems in society, due to their association with various chronic diseases. They determined the prevalence and distribution of overweight and obesity, using different anthropometric measurements and studied the anthropometric indicators which were more closely related to cardiovascular disease (CVD) risk factors in an Iranian urban population.

The cross-sectional study measured body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR) and percentage of body fat. They indicated that WHR and WHtR were the anthropometric indicators that best predicted CVD risk factors in men and WHR and WC in women.

In epidemiological and population based studies (Badaruddoza and Afzal, 1999, 2000; Badaruddoza and Kumar, 2009) the technique of anthropometric measurement such as BMI, WHR, waist circumference and subcutaneous skinfold thickness are used to assess adiposity, topography of adiposity and their relationship with cardiovascular risk factors. Various statistical techniques could be applied to examine the association between the risk factors and cardiovascular diseases. To simplify dimensions of cardiovascular risk, multivariate data reduction techniques such as principal component factor analysis (PCFA) have been employed to extract uncorrelated factors from numerous inter-correlated phenotypes (Susanne et al., 1998; Bellis et al., 2005; Goodman et al., 2005). Few PCFA based studies related anthropometric and cardiovascular diseases are given below.

Badaruddoza et al. (2011a) studied principal component factor analysis of anthropometric, physiometric and metabolic risk traits associated with cardiovascular diseases in north Indian Punjabi adults. The clustering of variables was evaluated by PCFA with varimax rotation on 616 individuals (350 males and 266 females). They
derived 6 and 5 principal factors accounting for 87% and 84% of the total variance derived among males and females, respectively. Factor 1 was loaded with glucose and lipids for males; glucose and blood pressure for females. Factor 2 was loaded with obesity in males; glucose and lipids in females. Factor 3 was loaded with blood pressure in males and obesity in females. These findings indicated the importance of PCFA to identify clusters of risk factors for chronic disease like CVD.

Kaur et al. (2012) studied cardiovascular risk factors among females of Punjabi Brahmin and Jat Sikh populations using principal component analysis (PCFA). It has been used for seventeen factors including anthropometric, physiometric, metabolic and glucose tolerance. A total of 428 females (199 Brahmin and 229 Jat Sikh) were recruited for the study. Blood samples from 100 (50 Brahmin and 50 Jat Sikh) out of total 428 individuals were obtained. PCFA reduced 16 risk factors to 7 uncorrelated components that explained maximum (87%) of the total variance among the females of both the groups. Factor 1 has high loading of the traits that reflects obesity related traits like body mass index (BMI), waist circumference (WC), hip circumference (HC) and waist to hip ratio (WHR) for both female populations and explained the largest portion of total variance (36% for Brahmin; 34% for Jat Sikh). Factor 2 was loaded predominantly with total cholesterol (TC), high density and low density lipoproteins (HDL & LDL), LDL-HDL ratio and TC-HDL ratio for Brahmin female population. Comparably, factor 2 is loaded with SBP, DBP and pulse pressure (PP) among Jat Sikh female population. Therefore, factor 2 was identified as responsible for dyslipidemia for Brahmin and hypertension for Jat Sikh.

2.2.2.3. Socio-economic status: Cardiovascular disease (CVD) is the leading cause of morbidity and mortality among high-income countries of the industrialized world, accounting for more than one-third of total deaths (Murray and Lopez, 1997; Lopez et al., 2006). It has been argued that the burden of CVDs could be shifting and could be more in the poor subjects in countries in economic transition such as India (Gupta and Gupta, 2009; Jeemon and Reddy, 2010). It is also leading in low- and middle-income countries, accounting for almost 25% of total deaths (Levenson et al., 2002; Xavier et al., 2008) and, by the year 2030, is projected to be the leading cause of death worldwide (Murray and Lopez, 1997; Lopez et al., 2006). Smoking, diabetes, hypertension,
abdominal obesity, psychosocial factors, fruit/vegetable consumption, physical activity and alcohol consumption are the modifiable risk factors for CVD. Therefore, the modification of these individual risk factors will significantly improve cardiovascular health (Menotti et al., 2003; Marmot and Wilkinson, 2006; Chow et al., 2009). Socioeconomic status include the conditions in which people are born, grow, live, work and age, and are shaped by the distribution of money, power and resources at global, national and local levels (WHO, 2002). Because the prevalence of some cardiovascular risk factors (eg, obesity, hypertension and diabetes) is rising worldwide (Yusuf et al., 2001a; Lopez et al., 2006; Mathers and Loncar, 2006), it is necessary to focus efforts on understanding the role of the ‘causes of the causes’ (the social determinants of health) to help bridge the current gap in equality (Kreatsoulas and Anand, 2009). Socio-economic status (SES) is determined or evaluated according to individuals monthly income and educational attainment (Fukuda et al., 2005).

The growing epidemics of cardiovascular diseases worldwide are due to their rapidly increasing incidence in low/middle income economies, especially India, China and Eastern Europe (Murray and Lopez, 1996). In India, the prevalence of coronary heart disease in urban areas has increased from about 2% in 1960 to 6.5% in 1970, 7.0% in 1980, 9.7% in 1990 and 10.5% in 2000 and in rural areas it increased from 2% in 1970 to 2.5% in 1980, 4% in 1990 and 4.5% in 2000 (Gupta, 2005). The poor are burdened with the risk factors associated with these diseases. A study on a semi-urban population in southern India found that higher socio-economic status was associated with greater prevalence of CVD risk factor (Reddy et al., 2002). In contrast, a study of industrial workers found that risk factors (tobacco use and hypertension) for CVD were concentrated among the lesser educated in both urban and rural areas, however, the prevalence of diabetes and being overweight increased with better education (Reddy et al., 2007). Rural northern India has a greater exposure to cardiovascular risk factors such as smoking, increasing incidence of atherosclerotic risk factors (obesity, diabetes, dyslipidemia and hypertension), poor working and living conditions, stress, lower rates of formal education, and reduced access to health care and health education (Berkman and Kawachi, 2000; Bodzsar and Zsakai, 2002; Levenson et al., 2002; Reddy, 2004; Marmot and Wilkinson, 2006; Joshi et al., 2007; Sugathan et al., 2008; Rao et al.,
Illiteracy and low educational status are highly prevalent in low income countries. It is well known that poverty is associated with greater ill health and mortality (Leon and Walt, 2001) and low educational status is a major determinant of disease as well as mortality (Marmot and Wilkinson, 2006). Low educational status is associated with under-nutrition, greater infant and maternal mortality, and acute and chronic infections (Leon and Walt, 2001). Case control studies in two large Indian cities have shown that those with low levels of education are at two times higher risk of experiencing a heart attack as persons as compared to high educational attainment (Rastogi et al., 2004a).

Cardiovascular risk factors have been studied in the younger population – children, adolescents and youth in the different parts of the world but these studies are nearly done on the high income countries and rare data is available from low income countries. In India some studies have reported a moderate to high prevalence of multiple
atherosclerosis risk factors in adolescents and young. Studies from metropolitan cities in non-governmental schools report a high prevalence of obesity. In low income schools in Indian urban and rural areas there is low prevalence of obesity but high prevalence of tobacco use. (Yusuf et al., 2001a; Beaghehole and Yach, 2003; Reddy, 2004; Lynch and Smith, 2005; Gupta et al., 2006, 2009; Divakaran et al., 2010).

Lynch et al. (1996) conducted the study on eastern Finnish population. He considered information on medical, biological, behavioral, psychological and social risk factors to mediate between SES and cardiovascular mortality. Socioeconomic status was assessed by the income (personal and household income), education, lifetime occupation, housing tenure and material living conditions. But personal income was preferred over household income as the literature reveals that personal income is a good predictor of SES. Similarly other finding was taken for SES factors like education and occupation.

Yu et al. (2000) investigated the association between four socio-economic indicators (education, occupation, income and marital status) with the three risk factors of cardiovascular disease (blood pressure, body mass index and smoking) and concluded that education level seemed to be the most important measure of the four socio-economic indicator in relation to cardiovascular risk factors in studied population of China with lower socio-economic status having higher level of cardiovascular risk factors. The association between socio-economic status and cardiovascular risk factors were more consistent among women than men.

Bovet et al. (2002) examined the prevalence of selected modifiable risk factors of CVD, blood pressure, BMI and smoking habits and their associations with indicators of SES in a representative sample of adult population of Dares Salaam, Tanzania and found that there is high prevalence of cardiovascular risk factors in urban population of such low income country. They documented that SES was associated inversely with blood pressure and smoking and directly with BMI. Blood pressure tended to be related more with education than wealth, BMI related more with wealth than education and smoking related to both.

A study from Jamaica (Mendez et al., 2003) suggested that the participants in both the lowest and highest income groups had elevated blood pressure and hypertension
prevalence relative to those in intermediate categories. Mean blood pressure and hypertension were generally highest in the top income group, creating a J-shaped pattern. Compared with the group with lowest mean blood pressure, participants in the top income category had 2-5 mm Hg higher SBP, and 4-5 mm Hg higher DBP. Distribution of blood pressure and hypertension by level of education were also J-shaped among men, with highest levels among college educated men.

Gulliford et al. (2004) evaluated the effects of income and education levels on blood pressure in general population of Trinidad and Tobago. Middle income countries are regarded as being at an earlier stage of epidemiological transition than high-income countries. Authors found a negative association of systolic blood pressure with increasing income and education in women. But no consistent association was found between education and income and blood pressure in men.

Monteiro et al. (2004) reviewed the socio economic status and obesity in adult population of developing countries. It was based on the studies conducted on adult population from developing countries. The authors reviewed the studies that presented the estimates of obesity prevalence, stratified by some indicators of SES in adult male and female populations from developing countries. They concluded that obesity can no longer be considered solely a disease of high SES group in the developing countries. The burden of obesity in a particular developing country tends to shift towards the groups of lower SES. They also suggested that the shift of obesity towards the poor apparently occurs at earliest stages of economic development among women than among men.

Dhargupta et al. (2009) conducted a study comprising the socio-economic variables like age, sex, education, family education status on Toto, Santal, Sabar and Lodha respondents of Jalpaiguri and Purulia district of West Bengal, India. The practice of education was found common in Santal, Lodha and Toto compared to Sabar. Majority of the Sabar respondents were under low-income group. Family size was large in Santal; than other three tribes. Education, occupation, income, house types were found to be highly significant in relation to health status among Santal than other three tribes.
Family education status, land type, land holding, family size, family type and personal cosmopolite were highly significant to health status among the Lodhas.

Braig et al. (2011) analyzed the relationship between cardiovascular diseases (CVD) and status inconsistency and assessed the influence of behavior related risk factors on this association. They sampled 8960 men and 6070 women, aged 45-65 years, from the EPIC-Heidelberg cohort (European Prospective Investigation into Cancer and Nutrition) were included. Socioeconomic status was assessed by education/vocational training and their occupational position at recruitment. Overweight was associated with status groups characterized by low education in combination with high occupation. Low education in combination with high occupation was related to CVD in men, but not in women. Men with low educational status working in a higher occupational position had a nearly had a nearly two-fold increased incidence of CVD than men with high educational status in lower occupation position.

Pednekar et al. (2011) performed a cohort study to determine the influence of education on CVD mortality. Subjects were classified according to their educational status into illiterate, primary school (≤ 5 years of formal education), middle school (6-8 years), secondary school (9-10 years) and college (> 10 years). In men inverse association of literacy status was observed with the CVD mortality, which was significantly greater in low educational status subjects while the association was not clear in women.

2.2.2.4. Lifestyle: Various hypothesis, put forward to explain consequences of urbanization such as lifestyle pattern, diet and stress have been implicated. Current urbanization rate in India is 35% as compared to 15% in 1950. With growing urbanization, socio-developmental changes have been taken place over last 40-50 years. Dramatic changes in lifestyle from traditional to modern have lead to physical inactivity due to technological advances. Furthermore, increasing population growth at the current rate to about 2% in each year and technological advances have shrunken the employment opportunities as particularly among young generation leading to stress and hypertension in young persons, including students and labours (Hussain et al., 1988; Chobanian et al., 2003; Das et al., 2005; Steptoe and Marmot, 2005; Matthews et al., 2006; Chida and Steptoe, 2010; Hilmert et al., 2010).
According to WHO (2002), an epidemiological transition has resulted in shifts in the living styles of the people. Higher consumption of junk food with increased saturated fats and refined carbohydrates, sedentary lifestyle, and cigarette smoking were the major determinant of increased CVD prevalence. Widely available high calories convenience foods and sedentary life styles have led to obesity epidemic across all socioeconomic levels, including the young and socio-economically disadvantaged (CDC, 2006).

2.2.2.4.1. Food habits: In India a substantial portion of the population, approximately 35%, follows a traditional vegetarian diet and has done so for many generations (Refsum et al., 2001; Key et al., 2006). Vegetarians are the individuals who do not eat any meat, poultry or fish. It has been suggested that diet rich in whole grains, legumes, vegetables, nuts and fruits, excluding meat and high fat animal products, along with regular exercise pattern is associated with low blood cholesterol levels, low blood pressure, less obesity and thus, lower incidence of stroke, heart diseases and mortality (Messina and Burke, 1997; Snowdon and Phillips, 1985; Dwyer, 1988). Studies have consistently reported that plasma cholesterol is lower in vegetarians than in non-vegetarians, by 0.61 mmol/l in the Health Food Shoppers Study, 0.43 mmol/l in the Oxford Vegetarian Study and 0.39 mmol/l and 0.35 mmol/l in men and women, respectively in EPIC-Oxford (Key et al., 1999). The effect of a vegetarian diet on plasma cholesterol is dependent on the exact composition of the diet, particularly in relation to saturated and unsaturated fatty acids (Key et al., 2006). Earlier studies have suggested that vegetarians have lower blood pressure than similar non-vegetarians, with differences of 2-10 mmHg in systolic and diastolic blood pressure associated with the lower BMI of the vegans (Beilin et al., 1988; Toohey et al., 1998; Appleby et al., 2002). African-American were associated with 35-44% lower risk of overall mortality due to frequent consumption of nuts, fruits and green salads (Fraser et al., 1997).

According to a British study, fresh fruits consumption lowered mortality due to heart disease by 24% and due to cerebrovascular disease by 32% and regular consumption of raw salad decreased mortality from heart disease by 26% (Key et al., 1999). Jenkins et al. (1997) induced healthy volunteers to follow strict vegetarian diet for two weeks. After two weeks they showed 25%, 33%, 20% and 21% decrease in total cholesterol,
LDL, triglycerides and total cholesterol-HDL ratio, respectively. Adults who have been following vegetarian food habits have lower SBP in reference to their lower body weights (Melby et al., 1988). Vegetarians in Northern Mexico had lower body weights, higher potassium and lower sodium intakes and hence lower MBP than the non-vegetarians (Wyatt et al., 1995). Kwok et al. (2000) compared the risk of ischemic heart disease among older Chinese vegetarian women with that of older non-vegetarian women living in Hong-Kong and found lower risk among the vegetarian group in addition to lower serum cholesterol levels.

A WHO report (2003) stated that a diet high in fat (particularly saturated fat), sodium and sugar and low in complex carbohydrates, fruit and vegetables increases the risk of CVD. It has been recommended that the percentage food energy derived from fat should be 35%, with 11% from saturated fat. The National Diet and Nutrition Survey in 2000/2001 found that the total energy intake from fat was 36% in men and 35% in women with 13% from saturated fat. It also found that the average intake of fruit and vegetables was fewer than 3 portions per day compared with the recommended 5 portions (National Health Survey). In the same survey salt intake was 11 g/day for men and 8.1 g/day for women. However, the Scientific Advisory Committee on Nutrition suggests that salt intake should be no more than 6 g/day. Trans-fatty acids reduce high-density lipoprotein (HDL) and increase low-density lipoprotein (LDL) cholesterol and can increase CHD risk. A meta-analysis showed that a 2% increase in the energy intake from trans-fatty acids increased CHD incidence by 23% (Mozaffarian et al., 2006). Eating oily fish rich in omega-3 fatty acids has been shown to reduce CHD mortality (Bays et al., 2008).

2.2.2.4.2. Physical activity and exercise: There are convincing evidences that physical activity reduces CVD and all causes of mortality among men (Rodriguez et al., 1994; Paffenbarger and Lee, 1997; Crespo et al., 2002). Substantial data from the literature indicates that physical activity is an important modifiable risk factor for many diseases. Regular physical activity has been linked to a reduced risk of coronary heart disease (Sundquist et al., 2005; Li et al., 2006), hypertension (Pereira et al., 1999; Barengo et al., 2005), stroke (Wendel-Vos et al., 2004; Hu et al., 2005a,b), obesity (Hill and Wyatt, 2005; Littman et al., 2005; Slentz et al., 2005), as well as decreased cardiovascular and
overall mortality (Andersen et al., 2000; Hu et al., 2004, 2005a,b). According to the World Health Organization (WHO), physical inactivity is responsible for 1.9 million deaths globally every year (WHO, 2005). Women have been consistently found to have lower rates of participation in physical activity than men (Jones et al., 1998; Sallis and Owen, 1999; Kaplan et al., 2001; Martinez-Gonzalez et al., 2001; Lim and Taylor, 2005; Pitsavos et al., 2005; Schaller et al., 2005). As physical activity occurs in multiple social aspects, other domains of physical activity are particularly important contributors to energy expenditure for women because they tend to spend more time in housekeeping, shopping, and food preparation activities (He and Baker, 2005). Rapid economic development in China has been associated with an increased prevalence of overweight and several chronic diseases, paralleling changes in lifestyle, including physical activity (Bell et al., 2001; Bell et al., 2002; He et al., 2008). It is important to understand that there is separate effect of different forms of physical activity on CVD because some individuals increase their physical activity and other may get the health benefits from being more physically active because of their way of work.

Epidemiological studies have shown that low physical activity is strong and independent risk factors for cardiovascular diseases (Powel and Blair, 1994). It is estimated that 30% of death of coronary heart diseases are due to sedentary life style and 14% of all death in US have been estimated and attributed to physical inactivity and unhealthy diet (McGinnis and Foege, 1993). Only few studies have been conducted among women (Lissner et al., 1996; Mensink et al., 1996; Anderson et al., 2000). Most of the previous studies (Rosengren and Wilhelmsen, 1996; Hedblad et al., 1997; Davey Smith et al., 2000) have focused on health effects of leisure time physical activity where as the evidence on the associations of occupational physical activity with CVD and total mortality is limited and inconsistent (Eaton et al., 1995; Rosengren and Wilhelmsen, 1996; Anderson et al., 2000; Crespo et al., 2002).

Regular physical activity is extremely important for CVD prevention (Pearson et al., 1993). The recommended physical activity is 30-to-45 minutes of moderate-intensity activity such as brisk-walking every day (Gupta et al., 2011). A strong inverse relationship is seen between physical activity and CVD development and mortality in men, women, ethnic minorities and the elderly. In the Harvard alumni cohort of 10,269
men, those who were moderately active (>4200 kJ or 1000 cal/wk) had a 23% lower risk of death as compared to less active group (Paffenbarger et al., 1993).

In the Health Professional Follow-up Study (Tanasescu et al., 2002) of 44,542 men, brisk walking ≥30 minutes daily, running ≥1 hour weekly, weight lifting ≥30 minutes weekly, and rowing ≥1 hour weekly, all reduced the risk of cardiovascular diseases. Higher levels of fitness and physical activity were associated with the greatest health benefit. Peak exercise capacity and physical fitness are the strongest predictors of CVD and mortality in both men and women.

Kelley et al. (2003) studied the effect of exercise on blood pressure in children. They performed meta-analysis studies that subjected children to an intervention of at least 8 weeks of exercise. They suggested reductions of 1% (SBP) and 3% (DBP) in those participating in exercise. Many other studies (Boreham et al., 1999; Bouziotas et al., 2004) have investigated associations between physical activity and blood pressure in children and adolescents. Leary et al. (2008) performed the study on large, contemporary population of 11- to 12-years-old. Authors suggested an association between higher levels of physical activity and lower levels of blood pressure. Their results also suggested that the volume rather than the intensity of the activity was important.

Barengo et al. (2004) investigated whether moderate or high leisure time physical activity, occupational physical activity, and commuting activity are associated with a reduced cardiovascular disease among 30-59 years Finnish subjects. They concluded that moderate and high levels of leisure time and physical activity are associated with a reduced CVD and all-cause mortality among both sexes. The relation remained significant even after adjusting for the most common risk factors for CVD and for other forms of physical activity. Commuting activity seemed to be inversely associated with CVD and all-cause mortality among women.

Albert et al. (2006) assessed the relationship between education and income, traditional and novel CVD risk factors and incident CVD events among 22,688 apparently healthy female health professionals participating in the Women’s Health Study. They found that more educated women were less likely to be smokers; had a lower prevalence of
hypertension, diabetes, and obesity; and were more likely to participate in vigorous
physical activity than less educated women. They concluded that there is a decrease in
incident CVD events with increasing levels of education and income. In contrast to the
relationship between income and CVD events, the relationship of CVD events with
education was explained only partially by traditional and novel risk factors for CVD.

Forman and Bulwer (2006) suggested that increasing physical activity improves lipid
metabolism, autonomic balance endothelial function, myocardial work efficiency and
even modifies fundamental inflammatory factors that underlies prosperity, increase
insulin sensitivity and lowers appetite – all key assesses in ameliorating and metabolic
syndrome.

Ito et al. (2008) studied the effect of education on the cardiovascular disease in
Japanese population. They sampled 39228 individuals aged 40-59 years. The authors
obtained information on education and lifestyle variables and found that <10 years of
education was associated with significantly higher mortality from all causes (hazard
ratio (HR) = 1.22, 95% confidence interval (CI): 1.05–1.42) and cardiovascular disease
(HR = 1.44, 95% CI: 1.01–2.06) as compared with >12 years of education, but was not
associated with higher incidence of cardiovascular disease (HR = 0.96, 95% CI: 0.78–
1.18) or higher mortality. They suggested that lower education is associated with higher
mortality from all causes and cardiovascular disease among the Japanese population
that is not totally attributable to lifestyle differences or higher cardiovascular disease
incidence.

Bishnoi et al. (2010) estimated the significant association of environmental factors such
as measures of sedentary lifestyle or physical inactivity with elevation in CVD risk.
Sedentary lifestyle such as number of hours spent reading per day also had a strong
relationship with CVD risk.

2.2.2.4.3. Stress: It is very difficult to define the term stress, however, it is commonly
used to identify different categories such as depression, anxiety, panic attack, social
isolation, lack of social support, acute and chronic life events, psychological work,
hostility and personality disorders. The INTERHEART study on risk factors including
psychological factors on 11,119 myocardial infarction cases and 13,648 controls across
52 countries reported that composite variables of subjective stress (homework, financial stress, low self efficiency, depression, etc.) appeared to be associated with increased risk of developing an acute myocardial infarction across gender, nationality, ethnic groups, independent of smoking and socio-economic stress. Few authors in their review work (Smith and Ruiz, 2002; Dimsdale, 2008) related to psychological and psychosocial stress and cardiovascular disease, discussed the mechanisms and impact of these stress on coronary heart disease. These stress increase CHD mortality and morbidity.

Tyroler (2000) divided different factors in various categories: inherited genes and culture, biomedical life style and psychological risk factors at individual level, social political and economic factors at group or aggregate level and social medical care, physiochemical and biological exposure at environmental level. Kivimaki et al. (2002) has studied related work stress and risk of cardiovascular mortality in a prospective cohort study of industrial employees.

Hitendrasinh et al. (2004) studied the effect of physical and mental activities on blood pressure on school children aged 10 years and above. Authors classified the children into two groups, those who were engaged in outdoor games for more than two hours and those for less than two hours daily. They concluded that both physical and mental stress had significant relationship with blood pressure. Physical stress reduced the blood pressure and BMI while mental stress affected only blood pressure. Boys and girls who spent more than two hours in outdoor activities had lower blood pressure and BMI.

Wu et al. (2010) studied related genetic influence on cardiovascular stress reactivity. They tested the hypothesis linking frequent exposure to psychosocial stress to adverse outcomes in cardiovascular health. To assess the importance of genetic factor, they performed meta-analysis on all published twin study. They estimated pooled heritability ranged from 0.26 to 0.43 for reactivity on mental stress. The study also revealed number of genes that may account for part of heritability of cardiovascular stress reactivity.

2.2.2.4. Smoking and high alcohol consumption: Kahmijin et al. (2002) suggests that lifestyle factors such as cigarette smoking and alcohol drinking are suitable for intervention targeted to individual in the community. Even though the incidence of
CVD and associated disease is decreasing in North America in recent decades, the opposite trend has been occurring in aboriginal population in Canada and the United States (Howard et al., 1999; Shah et al., 2000; Sewell et al., 2002). The increased prevalence of CVD in these groups is due to influence of the western lifestyle habits. Cigarette smoking is one of the such western lifestyle and leading cause of cardiovascular diseases and cancer (Young, 1994).

It is documented that increase in blood pressure and smoking are major risk factors for cardiovascular diseases (CVD) including coronary heart disease and stroke. Increase in blood pressure and smoking together cause more than 20% globally premature deaths (Ezzati et al., 2002; WHO, 2002; Asia pacific Cohort study, 2003, 2005), hence a combination of raised blood pressure and smoking may have synergistic impact on CVD (Labarthe, 1998; Nakamura et al., 2008). Reddy et al. (2005) pointed out from a national survey of tobacco use in 2002, that the Indian subcontinent is second only to China in both the production and consumption of tobacco products. India had an alarming rate of current tobacco use of 56% among men aged 12-60 years. Reddy et al. (2006) also observed in a survey of sixth and eight graders attending school in an urban setting that the prevalence of tobacco use (any history of use or current use) was 2-3 times higher among sixth graders compared with eighth graders. They suggested a new wave of smoking among India’s youth that may lead to serious future public health consequences.

In addition to the high prevalence of cigarette smoking, other forms of tobacco use are common in South Asia, including reported prevalences of smoking beedies (a small amount of tobacco wrapped in a temburini leaf) of 5.9% among males, and of smokeless tobacco (chewing tobacco or chewing paan) of 7.3% (5.5% in women and 7.6% in men) (Teo et al., 2006). Little data have existed regarding the association between the use of other forms of tobacco and the risk of CVD, however, a recent analysis of data from the INTERHEART case-control study of risk factors for acute myocardial infarction (MI) has documented that there is an increased risk of myocardial infarction associated with all forms of smoke and smokeless tobacco (Yusuf et al., 2004).
In addition, there was a graded increase in risk associated with the number of beedies smoked per day. The risk of having an acute myocardial infarction associated with the use of chewing tobacco was also increased (OR 2.23, 95% CI 1.41-3.52). Furthermore, the INTERHEART investigators found that 40 per cent of people who used smokeless tobacco also smoked cigarettes, and in these individuals there was a compounded risk of acute myocardial infarction associated with the use of both chewing tobacco and smoking cigarettes. Therefore, use of tobacco in forms other than cigarette smoking (beedies) is both common and important contributors to the CHD burden in South Asia (Goyal and Yusuf, 2006).

Alcohol consumption is analogous to the proverbial double-edged sword, and perhaps no other factor in cardiovascular health is capable of cutting so deeply in either direction depending on how it is used (O’Keefe et al., 2007). Most epidemiological studies have found a positive association between alcohol consumption and coronary heart disease, apart from an excess of coronary disease in alcoholics (D’Alonzo and Pell, 1968; Marmot, 1984). There is mounting evidence from both clinical and epidemiological studies that alcohol consumption, especially at higher levels, is associated with elevations in blood pressure (BP), both systolic and diastolic, although the elevation in systolic blood pressure is generally of greater magnitude. Previous studies have varied greatly in their assessment of potential confounding factors that might be involved in this association. A consistent earlier finding have showed that drinkers at the highest levels of alcohol consumption had an increase in blood pressure (Clark et al., 1967; Dawber et al., 1967; D’Alonzo and Pell, 1968; Gyntelberg and Meyer, 1974; Myrhed, 1974; Robinson et al., 1974; Klatsky et al., 1977; Harburg et al., 1980; Naimi et al., 2003; Rehm et al., 2003; Mukamal et al., 2003). It has been found that moderate amounts of alcohol may protect from coronary heart disease, with a relative risk of 0.5 although this suggestion remains controversial (Shaper et al., 1987; Beevers and Maheswaran, 1988). Most studies report that light to moderate drinkers have less risk than abstainers, and heavy drinkers have the highest risk. A recent meta-analysis of over 1 million individuals showed that consumption of 1 drink daily by women and 1 or 2 drinks daily by men was associated with a reduction in total mortality of 18% (DiCastelnuovo et al., 2006; Brien et al., 2011; Ronksley et al., 2011).
Marmot et al., (1994) estimated effect of blood pressure by the amount and pattern of alcohol consumption. They defined heavy drinker as the individual who consumed $\geq 300$ ml alcohol/week corresponding to $\geq 34$ g alcohol/day. Different countries have different standard for drink, 8-10 g and 13 g of alcohol in Britain and USA, respectively. Authors concluded that men consuming alcohol 200-400 ml/week had on an average 2.7/1.6 mmHg higher blood pressure than non-drinkers and men drinking 500 ml/week had 4.6/3.0 mmHg higher blood pressure. For women consuming alcohol $\geq 300$ ml/week had blood pressure 3.9/3.1 mmHg higher than non-drinker women.

Todkar et al. (2009) studied the prevalence and socio-demographic factors of hypertension in rural Maharashtra. A total of 1297 persons of age $\geq 19$ years from 256 houses were sampled. Significant association with hypertension were found for BMI, educational status, diabetes mellitus, smoking, socio-economic status, alcohol intake and family history of hypertension. The association between alcohol intake and hypertension shows 2.15% were having the habit of alcohol consumption, of these 28.57% study subjects were found with hypertension and 5.5 times higher risk was observed in alcohol consumers.

Brien et al. (2011) carried out a meta-analysis of interventional studies on effect of alcohol consumption on biological markers associated with coronary heart disease. The meta-analysis suggested that moderate consumption of alcohol (up to one drink or 15 g/day alcohol for women and up to two drinks or 30 g/day alcohol for men) has beneficial effects on a variety of biomarkers linked to risk of CHD. Alcohol consumption significantly increased circulating levels of high density lipoprotein cholesterol, apolipoprotein A1, and adiponectin and significantly decreased fibrinogen levels, all changes reported to be cardio-protective.

Kaur et al. (2011) estimated the prevalence of CVD risk factor like tobacco use, alcohol use and overweight in a rural population in Tamil Nadu, India. Study population included 10,500 subjects aged 25-64 years. Among them 47% were males, of which 37.6% were current smokers and 62.4% were alcohol users. Among females 15.1% were smokeless tobacco users. The authors suggested need for health promotion programs to encourage adoption of healthy lifestyle and policy interventions.