INTRODUCTION

1.1. Epidemiology

Epidemiology is defined as “the study of distribution and determinants of health related states or events in human population and its application to the prevention and control of health problems” (MacMahon and Pugh, 1970; Merrill, 2010). Thus, three closely inter-related components: frequency, distribution and determinants encompass all epidemiologic principles and methods.

The first component to be considered is measurement of disease frequency, which involves quantification of the existence or occurrence of disease. The availability of such data is a prerequisite for any systematic investigation of pattern of disease occurrence in a population. The second, the distribution of diseases considers such questions as, who is getting the disease in a population, as well as where and when the disease is occurring. Such questions may involve comparisons between different populations at a given time, between sub-groups of a population or between various periods of observations. Knowledge of such distributions is essential to describe hypothesis concerning possible causal or preventive factors. The third component, the determinants of disease, derives from the first two, since knowledge of frequency and distribution of disease is necessary to test an epidemiologic hypothesis. Therefore, identifying of determinants or determining factors of health related states or events are the primary function of epidemiology.

Hence, a cause is a specific event, condition and characteristic that precedes the health outcome which is necessary for its occurrence. And adverse health outcome can be prevented by eliminating the exposure. Identifying causal association is complex and typically requires “making a judgment” based on the totality of evidence, such as a valid statistical association time sequence of events, biology creditability and consistency among studies. Therefore, an important aspect of study of epidemiology is to identify relevant risk factors. A risk factor is behavior, environmental exposure or inherent genetic characteristics which are associated with important health condition (Last, 1995). Hence, a risk factor is condition that is associated with the increased probability of a
health related state or event. Therefore, it may be concluded that epidemiology not only involves the study of distribution and determinants of health related states or events in human population but also it involves the application of study to the prevention and control of health problem. Hence, results of epidemiological study can provide public health planner with information related to the risk of the disease to plan better and effective health programme for public.

1.2. Adolescence

Adolescence is defined as the ‘period between the beginnings of puberty until maturity’ in human development, and it extends mainly over the teen years and terminates legally when the age of maturity is reached (Thomas, 1985). Adolescent is derived from Latin word “to grow up”. It is a time of rapid change in the life of an individual, and it is the time between childhood and adulthood. While children are not just little adults and adults are not just large children, adolescents are also unique and constantly changing. So, in the present study according to the definition, adolescents aged 10 to 18 years were targeted as samples for the study. This phase of life has a very fast series of changes occurring and during this time there are some medical problems that may surface with the root for the future problems which will occur in the adult life of a person. Elevated blood pressure may be detected at this age. Since, the last two decades there is an increasing interest in the field of hypertension in adolescents (Kollias et al., 2011). Therefore, at present it is an established fact that hypertension in adolescent is not uncommon as previously believed and in most cases there is early onset of essential rather than secondary hypertension. More importantly, recent data suggest increasing trends of average blood pressure in adolescence which appears to be attributed to the increasing prevalence of obesity. Therefore, the present thesis has focused the aspects of epidemiology of elevated blood pressure among adolescents. To detect the occurrence of high blood pressure, it is important to define blood pressure and recognize the changes occurring in it.

1.3. Epidemiology of Elevated Blood Pressure in Adolescents

The pressure exerted by the blood on the walls of arteries and veins during its flow is known as blood pressure. Blood pressure is a quantitative trait that is highly variable in population studies (Parati et al., 1998). There is no specific level of blood pressure where
cardiovascular complications start to occur, thus the definition of normal blood pressure is arbitrary. When the value of blood pressure increases beyond the standard threshold, the term is given as high blood pressure.

(The details of distribution and classification of blood pressure have been given in section 3.8 of chapter 3-Materials and Methods at page number 96-100)

1.3.1. Tracking Blood Pressure from Adolescence

There is an evidence for early manifestation of the arteriosclerotic process present in adolescence (Lauer et al., 1984). However hypertension is predictive for development of cardiovascular disease in adults, it is uncertain whether elevated blood pressure, in early adolescent period has the same predictive values. The degree to which blood pressure level is maintained during early adolescence has been described by many investigators to determine whether children with high levels of blood pressure are likely to become adults with hypertension (Lauer et al., 1984; Sun et al., 2007; Chen and Wang, 2008; Soudarssanane et al., 2008). These studies have shown that there is indeed some consistency in the level of blood pressure over time, a phenomenon that has been referred to as tracking.

Attempt to track blood pressure represent an effort to develop systematic approach to predict adult blood pressure trend based on childhood and adolescence measurement. Rosner et al. (1977) studied whether measurements during adolescence are more predictive of adult blood pressures than those of early childhood, and found that “tracking correlations increased with initial age for both systolic and diastolic blood pressure for each time intervals between measurements”. However, in terms of sex, girls seem to reach at their adult or near adult blood pressure earlier than boys. It has been noted that (Berkey et al., 1991) blood pressure measurement at age 11 and 15 years in girls seems to predict most consistently where the adult blood pressure would ultimately fall. Among boys, who mature on average two years later than the girls, systolic blood pressure measured at age of 13 and 17 best predict systolic blood pressure at 40 years age (Gerber and Stern, 1999). Therefore, the study of tracking of blood pressure from childhood and adolescence to adulthood has many significant public health implications, as growing evidence indicates that hypertension is one of major modifiable risk factor for cardiovascular diseases. Recent studies (Chen and Wang, 2008; Kotchen, 2010; Raj,
 showed that increased blood pressure among adolescents is related to growing obesity epidemic. Although the evidence for blood pressure tracking from childhood and adolescence to adulthood is rich with large number of reported findings, but to the best of present knowledge, no systematic study and analysis has been conducted among Punjabi adolescent population. Hence, the present study focused to systematically evaluate epidemiological evidence of blood pressure with respect to quantitative traits from Punjabi adolescent population.

1.3.2. Magnitude and Prevalence of Adolescent Hypertension

Trends in hypertension prevalence have been comprehensively reviewed. The awareness that essential hypertension has its origin in childhood has resulted in increased emphasis on screening asymptomatic children. Research of pubmed database studies have shown high prevalence of adolescent hypertension in urban and rural areas (Kelishadi et al., 2007a; Raj et al., 2007; Gupta et al., 2009; Falkner, 2010; Raj, 2011; Narayanappa et al., 2012; Brar and Badaruddoza, 2013a,b).

There have been variable reports (Heyden et al., 1969; Shaper et al., 1969; Bello, 1993 and Harrabi et al., 2006) on the prevalence of adolescent hypertension ranging from 0% to 36%. Varying methodology, sample size, sex, age, environment and social factors could have contributed to this wide disparity. A number of reports have provided a more precise estimate of the prevalence of hypertension verified by separate measurements. A study conducted in Brazil had reported 9.4% prevalence of high blood pressure (Moura et al., 2004). Hansen et al. (2007) determined the frequency of hypertension and pre-hypertension to electronic medical record data from a well-child care visits in a cohort of over 14,000 primary care patients. With the advantage of having data on repeated blood pressure measurements on separate visits, these investigators determined the prevalence of hypertension to be 3.6% and the prevalence of pre-hypertension to be 3.4% in children and adolescents between the ages of 3 years and 18 years. A Canadian Study (McCrindle et al., 2010) looking for trends in cardiovascular risk and lifestyle factors in 20,719 adolescents (14- to 15- years-old) observed constant prevalence of stage I hypertension (5-6%) or stage II hypertension (2-4%). Katona et al. (2011) conducted a study on 10,539 adolescents with mean age 16.6 years in Hungary and found prevalence of hypertension
to be 2.53%. On the other hand, data from National Health and Nutrition Examination Survey (NHANES), a nationally representative survey of the health and nutritional status of non-institutionalized population of United States, showed hypertension prevalence in adolescents ranging from 5% (1999-2000) to 3% (2007-2008) (May et al., 2012). Similar figures were found in a Chinese study (Cao et al., 2012) that examined 88,974 scholars aged 12 to 17 years, in Changsha city in which 3.1% (4.7% in males and 1.5% in females) prevalence was reported. A recent study (Magliano et al., 2013) conducted in Brazilian adolescents reported the pooled prevalence of hypertension, estimated through random effects model, as 8.12%. In a cross-sectional study (McNiece et al., 2007) limited to the adolescence age, the prevalence of pre-hypertension and hypertension was determined in a cohort of 6,790 high school students as 11–17 years in Houston, Texas and found that nearly 20% of those adolescents were at risk of hypertension. Using the recommended repeated blood pressure measurements on those with an elevated initial blood pressure measurement, the authors found that the prevalence of hypertension was 3.2% and the prevalence of pre-hypertension was 15.7% in adolescents. The prevalence of hypertension and pre-hypertension combined was over 30% in obese boys and from 23–30% in obese girls, depending on ethnicity. Another analysis of the same two data cohorts demonstrated an overall increase in the prevalence of hypertension, from 2.7% in the 1988–1994 survey to 3.7% in the 1999–2002 survey (Din-Dzietham et al., 2007).

Few systematic studies were conducted in India which presented the prevalence of hypertension among adolescents. It has been varying widely in different Indian studies—from 0.46% to 11.9%. A study was conducted in 2007 among school students of adolescent age group in Mysore, and the reported prevalence was 6.1% (Gupta, 2004). In Shimla, the prevalence was 20% (study period 2005 to 2006) (Sharma et al., 2010). In a cross-sectional study (Saha et al., 2008) carried out to determine the prevalence of hypertension and variation of blood pressure with age among adolescents in an urban slum of Kolkata, India, a total of 1081 adolescents aged 10-19 years were sampled. The prevalence of hypertension was found to be 2.9% and highest prevalence (5.6%) was observed in the age group of 18-19 years. Khan et al. (2010) conducted a study among boys of Ahmedabad city and reported 9.78% hypertension and highest prevalence was found at 19 years of age (21.7%). In a cross-sectional population-based study of 1022
students aged 14-19 years in New Delhi reported 6.4% hypertension (Goel et al., 2010). The prevalence of hypertension and pre-hypertension was 3.4% and 10.6%, respectively, in a study carried out in adolescents of rural Wardha, Central India (Kumar et al., 2012). Among Punjabi adolescent population likewise Mohan et al. (2004) has reported the prevalence of 6.69% of sustained hypertension and it is higher in younger age group in urban areas. A recent study (Sundar et al., 2013) reported the prevalence of adolescent hypertension to be 21.5% which indicates it is major public health problem and immediate intervention is needed.

Prevalence of sustained hypertension is on the rise in urban area even in younger age groups (Mohan et al., 2004; Bansal et al., 2009; Sharma et al., 2010; Mitra et al., 2011). Mohan et al. (2004) found prevalence of sustained hypertension to be 6.69% in urban Ludhiana and 2.56% in rural Ludhiana. Males outnumbered females in both urban and rural areas. Urban adolescents are taller and heavier by possessing significant higher levels of body mass index than to their rural counterparts (Hema latha et al., 2013). 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 decreasd physical activities (Mohan et al., 2004).

Although there is generally lower prevalence of hypertension in rural Indian population, there has been steady increase over time in rural population as well (Gupta, 2004, Narayanaapa et al., 2012; Hema latha et al., 2013). This is probably due to rapid urbanization of rural India which has altered the dietary habits, level of physical activity and social pressures in life.

1.3.3. Incidence

The incidence of pediatric hypertension is not known, due to regional differences in the definition of high blood pressure, the distribution of reference blood pressure data and the blood pressure measurement methodology. Among adults, the prevalence of hypertension increases with age, indicating that new cases of hypertension in adults are diagnosed each year. Although primary hypertension is more commonly identified in adolescence than earlier in childhood, there is little information about the incident rates of hypertension in childhood. Within the National Childhood Blood Pressure database (Falkner et al., 2008),
a segment of adolescents underwent blood pressure measurement at intervals of 2 years and 4 years. An analysis of these data found that, among adolescents with pre-hypertension, 14% had developed hypertension 2 years later, which yielded an approximate incidence rate of 7% per year (Falkner, 2010). Among adolescents with high risk blood pressure values, including those designated from a single measurement as having pre-hypertension and hypertension combined, 68% of boys and 43% of girls had developed pre-hypertension or hypertension 2 years later. Despite the well-known variability in serial blood pressure measurements in children, there is now substantial evidence that blood pressure measured in childhood predicts future blood pressure. A community based study (Bao et al., 1995) of 1,505 children aged 5–14 years demonstrated tracking of systolic and diastolic blood pressures over 15 years, with statistically significant correlation coefficients between childhood blood pressure and later blood pressure levels. Among these, 116 young adult participants, who had developed hypertension, 48% and 41% had suffered elevated childhood systolic and diastolic blood pressures, respectively. The patterns may vary by race, but weight does not seem to affect population tracking data significantly (Donahue et al., 1994; Fuentes et al., 2002).

A systematic review and analysis (Chen and Wang, 2008) of 50 cohort studies that examined blood pressure tracking, documented significant blood pressure tracking correlation coefficients from childhood into adulthood. The strength of the tracking increased with baseline age and decreased with length of follow-up period. The analysis concluded that data from diverse populations show that the evidence for blood pressure tracking from childhood into adulthood is strong and that early intervention is important. Sun et al. (2007) examined serial data from participants in the Fels Longitudinal Study in Ohio and derived age- and gender-specific blood pressure levels in childhood that predicted hypertension in adulthood. They found that the earliest significant difference in childhood systolic blood pressure values among adults with and without hypertension occurred at age 5 years for boys and 8 years for girls. The interesting finding from this study was the level of childhood blood pressure that was predictive of subsequent hypertension in adults. The age- and gender-specific values for childhood systolic blood pressure among adults with hypertension were below the 50th percentile for systolic blood pressure.
pressure in children of median height, based on data in the Fourth report on diagnosis, evaluation, and treatment of high blood pressure in children and adolescents (NHBPEP, 2004). Therefore, the childhood systolic blood pressure values in the Fels Longitudinal Study that were predictive of future hypertension in adults were well below childhood blood pressure levels that are presently considered to be high risk.

1.4. Risk Factors for Adolescent Hypertension

‘Risk’ is defined as a probability of an adverse health outcome, whereas ‘risk factor’ refers to an attribute or characteristic or exposure of an individual whose presence or absence raises the probability of an adverse outcome (WHO, 2005). More than 200 risk factors have been estimated, but, the World Health Report (2002) has identified top 20 leading risk factors in terms of the burden of diseases according to the mortality status in the population.

Cardiovascular diseases are non-communicable diseases which are multi-factorial in nature and hypertension is one of them. Hypertension comprises both modifiable and non-modifiable risk factors. The association among various risk factors is mostly statistical. A particular risk factor in an individual merely increases the probability of developing a disease and does not necessarily mean that he/she will develop it. On the other hand, absence of the risk factor does not guarantee protection from the risk (Black, 2002).

Risk factors for hypertension have been exhaustively investigated and it has been found that most hypertension risk factors are non-modifiable such as advanced age, gender, ethnicity and family history of disease. Other risk factors are subjected to intervention like obesity, diabetes mellitus, dyslipidemia, socio-economic and lifestyle factors, mainly lack of physical activity. It has been realized that early management of these risk factors of lifestyle by modifications and/or pharmacological interventions can result in a significant drop in cardiovascular morbidity and mortality. This increases the importance of early detection with reversible and treatable risk factors (Rosenberg et al., 1990; Hebert et al., 1993; Paffenbarger et al., 1993; Manova et al., 2006; Flyn and Falkner, 2011; Nichols and Codogan, 2012). Thus, the risk factors of hypertension in adolescents can be broadly classified into two categories, non-modifiable and modifiable risk factors, as shown in Figure 1.1.
1.4.1. Non-modifiable Risk Factors

Non-modifiable risk factors are those risk factors which are irreversible in nature and cannot be changed by any kind of intervention. These include age, gender, ethnicity and family history.

1.4.1.1. Age and gender: The incidence of hypertension increases with individual’s age. Hypertension is characterized by early onset and greater mortality (Gupta et al., 2009). The probability of diagnosis of essential hypertension increases with age from birth onward (Bernstein et al., 2010). Children and young adolescents with blood pressure greater than 90th percentile for age have roughly threefold greater likelihood of becoming adults with hypertension compared to their peers with blood pressure at 50th percentile (Bernstein et al., 2010). There is evidence that blood pressure measured in adolescence will predict future blood pressure and adolescents with blood pressure distribution curve tend to maintain that position over time, a condition called tracking (Flynn and Falkner, 2011). High prevalence is observed in older groups of adolescents as compared to the younger ones in many recent studies (Saha et al., 2008; Kumar et al., 2012). Pre-
hypertension is shifted to hypertension if not treated as the age advances. Body mass index is found to be an important factor for detection of CVD and weight tends to increase through middle ages (Haslam, 2008; Latiffah and Hanachi, 2008; Gupta et al., 2009; Ghosh et al., 2010; Wang et al., 2012). With age the structure of heart and vessels changes and may lead to increased systolic blood pressure (Latiffah and Hanachi, 2008). Age related differences have also been found in the regional fat distributions over the body as observed by larger waist circumferences (Mazariegos et al., 1994) and greater waist-to-hip ratios (Baumgartner et al., 1993) and lower girths in the limbs in older subjects (Prothro and Rosenbloom, 1995). Rosner et al. (2013) analyzed a population-based sample of 3248 children in National Health and Nutrition Examination Survey (NHANES) III (1988–1994) and 8388 children in continuous NHANES (1999–2008), aged 8 to 17 years and found that boys were more likely to have elevated blood pressure as compared to girls of same age.

There are sex differences in the manner genetic and environmental factors influence the phenotype. Although autosomal genes are not expected to be different between males and females as a result of the random nature of the chromosomal segregation during meiosis, it is possible that some genes (or environments) differ in impact between women and men. There is a possibility that some genes contributing to blood pressure in women are distinct from genes contributing to blood pressure in men (Levine et al., 1982). A number of studies even report the same heritabilities for the two sexes, indicating that estimates for males and females could be set equal as a part of the model-fitting process used in these studies (Badaruddoza et al., 2009; Kumar and Badaruddoza, 2010).

1.4.1.2. Ethnicity: Ethnicity is a creation that encompass both genetic and cultural (language, religion, diet) differences (Anand, 1999). Individuals of different ethnic backgrounds tend to live in distinct region and societies. Variations in disease rates by ethnicity are also intervened with geographic differences. Hence, specific ethnic group with in one location adopt certain life styles, whereas same ethnic group in another location may adopt substantially different life styles. Consequently any study of variation in disease by different ethnic group is also interrupted by additional variations in life style, geography, socio economic status. Several factors may contribute to observed inter-ethnic differences in blood pressure profile. It has been documented from the seven
countries study that low CVD rate were observed in Japan and Mediterranean countries and high CVD rates in Finland and US. These differences were in large part explained by differences in diet, serum cholesterol, blood pressure, culture, life style and ethnicity (Yusuf et al., 2001).

Various countries may be experiencing different stages of epidemiological transition with varied life expectancy, diverse demographic profile, differing contributions from competing causes of death. Genetic factors are responsible for differences in individual’s susceptibility in a shared and relatively homogenous environment. They also contributed to inter-population differences due to variable frequencies of one or more genetic determinants of risk in different ethnic groups (Goldburt et al., 1994; Reddy, 1998; Suwandono and Aryastami, 1998; Zhou, 1998; Ghosh et al., 2010; Kumar and Badaruddoza, 2010; Badaruddoza et al., 2011a).

1.4.1.3. Genetics: An estimated 30–60% of the variation in blood pressure between individuals, after adjustment for age and sex, is attributed to the effect of genetic factors (Singh et al., 2010). A child with a history of hypertension in both parents and who has a sibling with hypertension, has a 40–60% chance of developing hypertension as an adult (Slocks, 1930). If the siblings are the monozygotic twins, the risk of the same increases to 80% (Slocks, 1930). The genetic susceptibility to develop primary hypertension results from the effects of multiple genes and is modulated by multiple environmental determinants (Lifton et al., 2001). Using linkage studies and positional cloning in humans, a dozen genes responsible for or associated to essential hypertension, have been identified (Meneton and Warnock, 2001). All of these genes are either mediating or involved in the regulation of renal sodium transport (Lifton, 1996). These mutations alter the blood pressure through a common pathway, changing salt and water re-absorption in the kidney (Lifton, 1996). Genes encoding components of the Renin-Angiotensin-Aldosterone system (RAAS), and angiotensinogen and angiotensin converting enzyme (ACE) polymorphisms may be related to hypertension (Singh et al., 2010). The same may also be related to blood pressure sensitivity in response to dietary salt intake (Singh et al., 2010). Normal functioning of endothelial ion channels is important in the control of vascular tone. Dysfunction of these ion channels could contribute to alterations in blood pressure (Baker, 2000). Studies indicate that genetic defects in sodium transport
across cell membranes may be important in development of primary hypertension in humans (Baker, 2000; Meneton and Warnock, 2001). Genetically mediated alterations in the regulation or expression of renal ion channels and transporters may also be important in the genesis of hypertension (Lifton et al., 2001). Variation and mutations in other genes, such as α-adducin, arterial natriuretic factor, the insulin receptor, β2-adrenergic receptor, calcitonin gene-related peptide, angiotensinase C, rennin-binding protein, endothelin-1 precursor, and G-protein b3-subunit, have also been reported to be associated with the development of essential hypertension (Williams et al., 1994; Luft, 1998, 2004; Barlassina et al., 2002; Singh et al, 2010).

1.4.1.4. Family history: Hypertension is familial in nature means individual with a close relative of this disease is itself at the increased risk (Brand et al., 1992; Di Castelnuova et al., 2009). High blood pressure is a complex trait found to be determined by the interaction of genetic and environmental factors (Badaruddoza and Kumar, 2009; Badaruddoza and Sawhney, 2009; Ghosh et al., 2010; Kumar and Badaruddoza, 2010). Therefore, determination of the relative roles of genes and environment in the etiology of high blood pressure is very important. Blood pressure level has been shown to be highest in Finish population among world (Wolf et al., 1997). However, there are regional differences in the prevalence of hypertension because of the result of favourable changes in life style at population level. The prevalence of hypertension varies around the world with the lowest prevalence in rural India (3.4% in men and 6.8% in women) and the highest prevalence in Poland (68.9% in men and 72.5% in women) (Kearney et al., 2005). This suggests that genetics play a major role on Poland population in the development of hypertension. Environmental factors also play a significant role in the observed variations in the distribution of blood pressure among different population groups. Epidemiologic interest in the genetic determination of high blood pressure received a major stimulus in 1950s and 1960s as suggested by the Platt-Pickering controversy. This controversy was regarding the nature of the inheritance of hypertension (Tyroler, 1977). Factors affecting blood pressure are diverse and complex (Burke and Motulsky, 1992; Badaruddoza and Sawhney, 2009; Zhang et al., 2010; Badaruddoza and Kaur, 2012). Many investigators tried to elucidate factors contributing to variations in blood pressure levels within and between populations. Several personal, social, and environmental factors contribute towards this variation.
With the advancing age of the subject his/her family history acts as an excellent tool for increasing awareness of risk but, it is probably not as useful when the risk of those persons compared is too low or high according to risk factors other than family history (Valdez et al., 2007). In many families high blood pressure is most likely a polygenic condition where multiple genes contribute to development of high blood pressure and in some families single gene influences the blood pressure (Badaruddoza and Sawhney, 2009; Ghosh et al., 2010; Kumar and Badaruddoza, 2010). Some genes in families are also associated with the biochemical changes in the metabolism of the body (Tyroler, 2000). Familial aggregation of blood pressure need not necessarily be genetic in nature but shared environment can also be the cause. The facts that the lifestyles and personal interest aggregate within families suggest the role of environment (Garn and Rohman, 1966; Borhani et al., 1976; Grotevant et al., 1977; Saadat et al., 2001; Gu et al., 2007).

Family studies and twin studies have documented that blood pressure and hypertension are heritable. Heritability of systolic blood pressure (SBP) and diastolic blood pressure (DBP) based on family studies generally range from 15% to 35%. In twin studies, heritability constitutes blood pressure approximately 60% for males and approximately 30% for females (Williams et al., 1991). Heritability reflects all possible genetic contributions to population’s phenotypic variances. It includes effects due to epistatic (multi-genic interactions) as well as maternal and paternal effects where individuals are directly affected by their parent’s phenotype (Falconer and Mackay, 1996). There are four major designs for inferring the heritability of a quantitative trait parent-offspring, mid parent–offspring; half sib family; and full-sib family designs (Falconer, 1989).

Traditionally, to estimate the heritability for a quantitative trait of interest, measurements are taken directly on parents and offspring. This is followed by regression of offspring measurements on parent measurements; the slope of the regression is proportional to the heritability of the trait (Lynch and Walsh, 1998).

In twin adult studies, the $h^2$ for SBP ranges from 85% - 25% and $h^2$ for DBP ranges from 64% -19%. Cultural heritability also vary from 5% - 2% for both SBP and DBP (Feinleib and Garrison, 1979; Morton et al., 1980; Burns and Lauer, 1986; Persuse et al., 1989). Onat and Sansoy (1998) studied the heritabilities of certain cardiovascular risk factors in
Turkish adult (25-75 years) population and estimated them to be 22% for systolic, 27% for diastolic blood pressure, 19% for BMI and 17% for WHR.

1.4.2. Modifiable Risk Factors

A modifiable risk factor is a determinant that can be modified by intervention, thereby reducing the probability of the disease. The following modifiable risk factors are important to mention:

1.4.2.1. Obesity: Adolescent obesity is on the rise and is associated in the literature with adverse health effects. Excessive body weight, including overweight and obesity, together with hypertension, represents major civilization threats of the 21st century. At a population level, obesity may refer to the prevalence of an arbitrarily specified level of body mass index (BMI), waist circumference (WC), waist to hip ratio (WHR), waist to height ratio (WHtR) and sum of skinfold thickness as measured at selected body sites, or some other measures. Obesity must be explicitly addressed on any particular context. The principal concepts of obesity found relevant to health must be clarified. Broadly obesity may be classified as central adiposity, fatness and overweight.

The relationship of childhood obesity with blood pressure has been examined in many studies in the past (Graf et al., 2005; Maffeis et al., 2001; Nur et al., 2008; Cobayashi et al., 2010; May et al., 2012). High blood pressure (i.e., BP >95th percentile) was seen in 35.4% of overweight children in the European pediatric cohort (l’Allemand et al., 2008). The most comprehensive study by Rosner et al. (2000) pooled data from 8 large US epidemiological studies involving over 47000 children to describe the blood pressure differences between black and white children in relation to body size. Irrespective of race, gender, or age, the risk of elevated blood pressure was significantly higher for children in the upper decile compared with the lower decile of BMI, with an odds ratio of systolic hypertension ranging from 2.5 to 3.7. Freedman et al. (1999a) reported that overweight children in the Bogalusa Heart Study were 4.5 and 2.4 times as likely to have elevated systolic blood pressure and diastolic blood pressure, respectively. Sorof et al. (2002) reported a three times greater prevalence of hypertension in obese compared with nonobese adolescents in a school-based hypertension and obesity screening. Data from an Indian study (Raj et al., 2007) covering 25000 school children in the age-group of 5–16
years reported similar figures. The first instance hypertension was seen in 10.10% of normal-weight (non-overweight and non-obese) children, 17.34% of overweight children, and 18.32% of obese children in this study. The corresponding figures for systolic (first instance) hypertension were 5.38%, 12.31%, and 14.66%, respectively and for diastolic hypertension (first instance) 6.45%, 8.86%, and 8.90%, respectively. The rate of change in BMI appears to be more significant than the absolute level of BMI in influencing pediatric blood pressure as evidenced by a recent cohort of 12129 children from India (Raj et al., 2010a,b).

Probable mechanisms of obesity-related hypertension include insulin resistance, sodium retention, increased sympathetic nervous system activity, activation of the renin-angiotensin-aldosterone system, and altered vascular function (Kotchen, 2010). Sympathetic nervous system activity is increased in obesity, particularly sympathetic activity to the kidney and skeletal muscle (Vaz et al., 1997; Alvarez et al., 2002 and Davy, 2004). The probable reasons for over activation of the sympathetic nervous system in obesity include hyperinsulinemia and/or insulin resistance; increase in leptin, adiponectin, or other adipokines; renin–angiotensin system overactivity; and lifestyle factors (Kotchen, 2010). Hypertension is also causally related to sleep apnea, possibly due to sympathetic overflow as a consequence of intermittent hypoxia (Friedman and Logan, 2009a,b). Obesity-related hypertension is associated with renal sodium retention and impaired pressure natriuresis (Hall, 2003). Obese individuals and individuals with the metabolic syndrome tend to be relatively salt sensitive (Rocchini et al., 1989; Chen et al., 2009). Activation of the renin–angiotensin system may also contribute to obesity-related hypertension (Kotchen, 2010). Several studies suggest that plasma renin activity and plasma angiotensin II concentrations are elevated in obesity (Sharma, 2004; Engeli et al., 2005). Vascular endothelial dysfunction is associated with a number of cardiovascular risk factors, including obesity, insulin resistance, and hypertension (Steinberg et al., 1996; Meyers and Gokce, 2007). Increased sympathetic over activity is a probable mechanism by which leptin may increase arterial pressure (Kotchen, 2010). Leptin activates the sympathetic nervous system both by centrally mediated effects on the hypothalamus and by local peripheral actions (Mark et al., 2009). Circulating adiponectin
levels are decreased in obesity-induced insulin resistance, and some studies suggest that adiponectin is protective against hypertension through an endothelial-dependent mechanism (Rasouli and Kern, 2008; Yiannikouris et al., 2010). Whether hypertension is causally related to insulin resistance and/or hyperinsulinemia is a matter of debate. The probable reasons by which insulin resistance and/or hyperinsulinemia may increase blood pressure include an antinatriuretic effect of insulin, increased sympathetic nervous system activity, augmented responses to endogenous vasoconstrictors, altered vascular membrane cation transport, impaired endothelium-dependent vasodilatation, and stimulation of vascular smooth muscle growth by insulin (Kotchen, 2010). The putative role of insulin resistance in childhood obesity assumes significance in view of the fact that Indian children exhibit higher blood pressures in comparison to their Western counterparts (Raj et al., 2010a,b).

The classification of weight status into dichotomous categories of “obese” or “non-obese” is clinically useful for characterizing the overall risk of hypertension from obesity. In fact, the risk of hypertension in children increases across the entire range of BMI values and is not defined by a simple threshold effect. Sorof et al. (2002) found an increased prevalence of systolic hypertension as BMI percentiles increased from the 5th to 95th percentile. Among all demographic and clinical factors analyzed, BMI was most strongly associated with hypertension.

1.4.2.2. Lifestyle risk factors: A lifestyle is a characteristic bundle of behaviors that makes sense to both others and oneself in a given time and place, including social relations, consumption, entertainment and dress. The behaviors and practices within lifestyles are a mixture of habits, conventional ways of doing things, and reasoned actions that together constitute the mode of living of an individual or group. In the present study diet, physical activity, exercise, screen time, sleeping time, smoking and alcohol have been included.

1.4.2.2.1. Diet: Previously, non-vegetarian diet was thought to be superior over the vegetarian diet, as it was believed to contain more energetic ingredients, but, this concept has changed over the time. With advancements in medical sciences, vegetarian diet has been found to be more scientific for human body. Non-vegetarian diet contains
cholesterol and fatty acids which are important cause for hypertension. Nearly 60% of non-vegetarian diet is useless for human body and the remaining 40% is composed of harmful and toxic products including hydrochloric acid. In addition, non-vegetarian diet lacks fibers which are useful in lowering the incidence of hypertension and obesity.

Zhao et al. (2011) has studied the 27 dietary factors, 17 have been proposed to have protective effects against hypertension, six were proposed to be risk factors for hypertension, and the association between blood pressure and the remaining factors were considered inconclusive. Using milk formula rather than mother’s milk during infancy is the starting diet risk factor for hypertension. Significant adverse changes have occurred in older children’s food consumption (French et al., 2001). A reduction in regular breakfast consumption, an increase in consumption of foods prepared away from the home, an increase in the percentage of total calories from snacks, an increase in consumption of fried and nutrient-poor foods, a significant increase in portion size at each meal, and an increase in consumption of sweetened beverages, whereas dairy product consumption has decreased, and a shift away from high-fiber fruits and vegetables as well as a general decline in fruit and vegetable consumption other than potatoes (Siega-Riz et al., 1998; Cavadini et al., 2000; French et al., 2001; Nielsen et al., 2002; Nielsen and Popkin, 2003). Fried potatoes make up a substantial portion of the vegetable intake (Cavadini et al., 2000). Sugar consumption has increased, particularly in preschool children (Kranz et al., 2005). With regard to micronutrients, the shift in dietary patterns has resulted in median intakes, below recommended values of many important nutrients during adolescence (Wright et al., 2003). Sodium intake is far in excess of recommended levels, whereas, calcium and potassium intakes are below recommended levels (Wright et al., 2003; Ervin et al., 2004). Sodium intake is positively associated with SBP and risk for pre-hypertension and hypertension among US children and adolescents, and this association may be stronger among those who are overweight/obese (Yang et al., 2012).

1.4.2.3. Physical activity: Besides unhealthy food habit, the sedentary lifestyle is also of major concern in adolescents. A negative association between the amount of physical activity and hypertension, leading to mortality in the both developed and developing countries, has been well established (Divakaran et al., 2010; Tsioufis et al., 2010).
Physical activity is any bodily movement produced by skeletal muscles that result in energy expenditure. Exercise is a structured activity having fitness as its goal. All types of physical activities produce significant health benefits (Atkinson et al., 2009; Rodriguez-Rodriguez et al., 2009). According to Bonow et al. (2006), sedentary lifestyle is among the five major risk factors, along with high blood pressure, abnormal values of blood lipids, smoking and obesity, contributing to CVD (Pate et al., 1995; Jousilahti et al., 1996; Manson et al., 1999; Willett et al., 1999; Blair et al., 2001; Wannamethee and Shaper, 2001; Dubbert et al., 2002; Fang et al., 2003; Kreatsoulas and Anand, 2009; Divakaran et al., 2010). Declining levels of physical activity among adolescents are thought to be partly responsible for hypertension (Andersen et al., 1998; Goran et al., 1999; Molnar and Livingstone, 2000). Less physical activity result in sedentary lifestyle like watching TV, playing video games and unhealthy food consumption which again result in hypertension (Kelishadi et al., 2007b). Gidding et al. (2006a) studied a cohort comprised 663 youths, of whom 623 completed the 3 year visit and observed for every 100 estimated-matabolic-equivalent hours of physical activity. There was a decrease of 1.15 mmHg of systolic blood pressure. Many other studies also report the lower levels of SBP with higher levels of physical activity (Brage et al., 2004; Anderson et al., 2006; Leary et al., 2008).

Regular physical activity, fitness and exercise are of critical importance for the health and well being of people of all ages. Interventional studies (Austin et al., 2004; Campos-Rodrigueg et al., 2007; Pouliou et al., 2012) have demonstrated that increased physical activity reduces blood pressure in hypertensive and normotensive individuals independently from weight loss. Therefore, physical activity should be practiced at a moderate intensity levels in order to reduce systolic and diastolic blood pressure. Physical activity should be considered as an important measure for the prevention and treatment of hypertension in adulthood. Even though for adults, the effect of physical activity on blood pressure is well established, but the literature data are still limited regarding young adolescents. Physical activity is a key component of the therapeutic life style changes recommended for preventing and treating hypertension in children and in youth (Gu et al., 1998). The American Heart Association recommends that children and youth should participate in atleast 60 minutes of moderate-to-vigorous physical activity daily for
cardiovascular health (Robinson et al., 2005; Topouchian et al., 2006; Pouliou et al., 2012).

1.4.2.4. Exercise: As many as 2,50,000 deaths per year in the United States are attributable to a lack of regular exercise (Bonow et al., 2006). Exercise helps by reducing weight and lowering blood pressure. It also reduces the LDL (bad cholesterol) levels and total cholesterol and raise the HDL (good cholesterol) levels in blood. Pouliou et al. (2012) found that death rates reduced by 20-25% in heart attack patients who participated in formal exercise programs. In children and adolescents, several intervention studies have been undertaken to assess the effects of exercise, on resting systolic and diastolic blood pressure. Kelley et al. (2003) performed a meta analysis of studies that randomized children to an intervention of at least 8 weeks of exercise; the pooled estimates suggested the reduction of 1% (SBP) and 3% (DBP) in those participants in exercise, although the confidence intervals around these estimates were wide.

1.4.2.5. Screen time: The studies aiming to understand associations between sedentary behavior and weight gain and hypertension have almost exclusively focused on adolescents’ television use (Eisenmann et al., 2008; Fulton et al., 2009; Steffen et al., 2009), or the broader concept of screen time, which incorporates other technology-based activities such as computer and video game use (Must et al., 2007; Li et al., 2010; Sisson et al., 2010). In children and adolescents, several studies have analyzed to show significant association between TV viewing and individual CVD risk factors such as abdominal fat (Ortega et al., 2007a,b), lipid abnormalities (Wong et al., 1992 and Guillaume et al., 1997), hypertension (Pardee et al., 2007; Martinez-Gomez et al., 2009), and insulin resistance (Margeirsdottir et al., 2007). Excessive TV viewing seems to be related to an unfavorable CVD risk factors profile in adolescence (Martinez-Gomez et al., 2010). In obese children, the amount of time spent watching TV is associated with both hypertension and the severity of obesity. Thus, TV viewing is a potential target for addressing hypertension in obese children (Hancox et al., 2004; Pardee et al., 2007).

1.4.2.6. Sleeping time: Physiologically, average sleep duration decreases with progression from infancy, childhood to adolescence (Iglowstein et al., 2003) due to increasing demand from school and work, as well as changes in leisure activities such as
television watching and computer games. Sleep deprivation is now increasingly recognized as a lifestyle factor contributing to the global epidemic of childhood obesity and a novel, potentially reversible cardiovascular risk factor. Both laboratory and epidemiological studies suggested the associations of obesity, insulin resistance, diabetes and cardiovascular disease with sleep debt in children, adolescents and adults (Van Cauter et al., 2007; Van Cauter and Knutson, 2008). Reports of usual sleep duration are inversely linked to resting blood pressure and hypertension risk in adults (Cappuccio et al., 2007; Kim and Jo, 2010). A study is consistent with the hypothesis that the cardiovascular consequences of short sleep may also seen in adolescence (Mezick et al., 2012). Adolescents who don’t sleep well or long enough may have a higher risk of elevated blood pressure that could lead to cardiovascular diseases later in life (Lusardi et al., 1999). Poor sleep quality is associated with prehypertension in healthy adolescents (Javaheri et al., 2008).

1.4.2.7. Smoking: Another life style factors is cigarette smoking. Smoking in youth is an overwhelming public health problem for the following three reasons. First of all, direct health consequences as the development of premature atherosclerotic lesions have been shown in youth (Strong et al., 1994; Berenson et al., 1998). Secondly, the onset of this habit leads to deleterious health effects in later adulthood (Dagenais et al., 1990; Kannel and Higgins, 1990). Finally, smoking in teenagers is often associated with unhealthy lifestyle (Freedman et al., 1986; Raitakari et al., 1995; Burke et al., 1997). Due to these reasons, smoking uptake during adolescence entails harmful consequences on the current as well as on future population health. Tobacco use continues to be the single leading preventable cause of death and is responsible for 4 million annual deaths worldwide (Mokdad et al., 2004; CDC, 2011a,b). Because 80% of established adult smokers begin smoking before 18 years of age (SAMHSA, 2009) and in view of the unequivocal evidence linking tobacco use, particularly cigarette use, and adverse health and developmental outcomes (CDC, 2006; 2011a,b). Therefore, the American Academy of Pediatrics (AAP) Committee on environmental health called attention to the health hazards of environmental tobacco smoke in 1997 with evidence based recommendations for pediatricians and child care providers (AAP, 1997). Environmental tobacco smoke in childhood has been linked to cardiovascular diseases and many other complications in adulthood (CDC, 2006).
In India, tobacco consumption is found among 27.5% of males and 11.6% of females (Singh et al., 2007). Cigarette smoking causes reduced circulation by narrowing the blood vessels and puts smokers at risk of developing peripheral vascular disease and abdominal aortic aneurysm. Certain carcinogens, such as carbon monoxide found in tobacco, form carboxyhemoglobin when it combines with hemoglobin. Carboxyhemoglobin decreases the ability of the blood to carry oxygen, narrowing of arteries, blockage of blood vessels and high cholesterol. There is high smokeless tobacco usage in India because of the local belief that it cures minor ailments like toothache. Also, there is less awareness regarding its health hazards (Gupta and Ray, 2003; Kaur et al., 2011). It is more common in rural population as compared to urban. World Health Organization (WHO) research estimates that over 20% of CVD is due to smoking (Guilbert, 2003).

1.4.2.8. **Alcohol consumption:** Alcohol abuse in adolescents has been a source of increasing concern for parents and the public authorities who are in charge of controlling alcoholic consumption by teenagers (Bailey and Valley, 1993; Klatsky, 1995). Alcohol consumption in adolescents seems to have increased in many folds where several surveys indicate that alcohol use/abuse by college students is a serious and growing problem (Huselid and Cooper, 1992; Gross, 1993). Many factors may be involved in this problem, including family attitudes toward alcohol, association with alcohol-using peers (Barnes and Welte, 1986). Similarly, the level of alcohol consumption at a young age has been regarded as a predictor of alcoholism at older ages (Andreasonn et al., 1993). On the other hand, an association between alcohol consumption and high blood pressure has been reported in several studies (Jerez and Coviello, 1998; Soudarissanane et al., 2006; Yin et al., 2007; Wakabayashi, 2009). The prevalence of systolic hypertension was greater in moderate and heavy drinkers than in nondrinkers. Approximately half of the students had drunk an alcoholic drink at 13–14 years old for the first time and only 10% started before the age of 10 years. No association was observed between the age of onset of alcohol drinking and a higher frequency in consumption. This would suggest that drinking patterns are acquired with age and that a strong social influence may be claimed. Most studies performed to analyze the relationship between alcohol consumption and arterial blood pressure values show there is a correlation with increased consumption and
increased systolic blood pressure, but not with increased diastolic blood pressure (Xin et al., 2001; Klatsky, 2008). If heavy alcohol intake is considered to be one of the risk factor for hypertension then one should easily realize that it is so reversible (O’Keefe et al., 2007; Sugathan et al., 2008).

1.4.2.9. Socio-economic risk factors: It is likely that when different ethnic groups are compared within the same environment, non-conventional risk factors appear more explanatory, whereas the conventional risk factors are more explanatory when the same gene pool confronts different environments, as in rural-urban and migrant–nonmigrant comparisons (Badaruddoza and Brar, 2006). Indians appear to manifest high risk of hypertension due to some undefined factors that contribute to ethnic susceptibility which are triggered or magnified by an environment that promotes unhealthy living habits and superimposes conventional risk factors.

Socio-economic status (SES) is defined as the relative position of a family or individual on a hierarchical social structure, based on their access to or control over wealth, prestige and power (Mueller and Parcel, 1981). Socio-economic status (SES) is usually measured by determining education, income, occupation, or a composite of these dimensions. Although education is the most commonly used measure of SES in epidemiological studies (van den Berg et al., 2013; Rasiah et al., 2013) Socioeconomic status is the important predictor of living standard and health status as it influences social security in terms of accessibility, affordability, acceptability and actual utilization of various health facilities. Therefore, several attempts have been made to develop a uniform scale for socio-economic classification of population on income with scientific basis to apply with ease in each section of the population.

Hypertension in developing countries is characterized by early age of onset and greater mortality. Worldwide data reveal that while more than 70% of deaths in high income countries occur after the age of 70 years, in low and middle income countries more than 70% deaths occur below this age. Hypertension occur at least ten years earlier in South Asians as compared to other ethnic groups and the average age of stroke is much lower in western countries (Gupta et al., 2009; Kar et al., 2010; Boparai et al., 2011). India is one of the fastest growing economies of the world. Rapid economic growth, globalization, urbanization, rural-urban migration and aggressive marketing are all leading to a dramatic
shift in diet and living behaviors of individuals, families and communities. Consequently, adverse dietary changes in the population, sedentary activity, increasing tobacco use with consequent changes in the hypertension risk factors are accruing at great speed and at earlier stages than other countries (Hawkes, 2005; Popkin et al., 2005; Reddy et al., 2006, Badaruddoza and Kumar, 2009; Badaruddoza et al., 2011b; Badaruddoza and Kaur, 2012).

Of further concern is the fact that Indians are surrendering CVDs five to ten years earlier than their Western counterparts (Mohan et al., 2007; Teoh et al., 2007), in their most productive years. Unfortunately, scientific data also show that socio-economically disadvantaged sections of the population are now the dominant victims of hypertension and other CVD risk factors (Gupta et al., 1991; Pais et al., 1996; Reddy et al., 1996; Rastogi et al., 2004a,b; Reddy et al., 2007; Badaruddoza and Kumar, 2009). There is also preliminary evidence that the burden of hypertension in rural areas is increasing (Joshi et al., 2006; Badaruddoza et al., 2011b).

The relationship between SES and health has been observed from many centuries. But still much remain to understand about how low socio-economic status increase hypertension and mortality risk. It has been shown consistently that socio-economic status is inversely associated with cardiovascular mobility and mortality. In developed countries, the declining trend in hypertension has been more pronounced among individuals in higher socio-economic groups, and the difference in the occurrence of hypertension between higher and lower socio-economic group is widening. Regardless of which measure of SES is used, there is abundant evidence for the existence of an inverse relationship between SES and hypertension in developed and developing countries, with only few exceptions (Kaplan and Keil, 1993; Kumar and Badaruddoza, 2010; Badaruddoza et al., 2011b). Elevated blood pressure and current smoking tended to decrease with an increment in SES, and total cholesterol level, body mass index, and fasting blood glucose levels showed somewhat inconsistent patterns (Song et al., 2006; Kumar and Badaruddoza, 2010).

Many investigators have pointed out social and economic status differences, indicated either by the type of school attended (i.e. public vs private), by the region of residence (i.e. rural vs urban), or by the level of parental education (primary school vs higher
education) as important determinants that appear to influence the prevalence of hypertension risk factors in both developed and developing world (Kaplan and Keil, 1993; Bunker et al., 1996; Winkleby et al., 1999). Cross-sectional studies have indicated that various socio-economic factors are inversely associated with CVD morbidity and mortality in developed countries (Winkleby et al., 1992; Kaplan and Keil, 1993), while in developing countries, existing data on socio-economic conditions and the prevalence of hypertension are inconsistent (Sorlie and Garcia-Palmieri, 1990; Gupta et al., 1994; Zhijie et al., 2002). Socio-economic position of the family and parents’ education are very important risk factors for adolescent hypertension. Various investigators found various levels of associations (Manios et al., 2004; Kocaoglu et al., 2005; Amin et al., 2008; Chen et al., 2011). A study by Zaman et al. (2012) has shown that those of lower socio-economic levels had more adverse levels of behavioral risk factors of smoking, alcohol and low fruit intake, while those of higher socio-economic levels had more adverse levels of overweight, diabetes and sedentary lifestyles. However those from lower levels had poorer knowledge of risk factors of hypertension and were less likely to have received risk factor screening and are therefore, particularly ill-prepared to manage the consequences of a projected epidemic.

1.4.2.10. Diabetes: Diabetes is a disorder of glucose metabolism with complex interplays between genetic, lifestyle and environmental factors. Historically, type 2 diabetes is much less common in children and adolescents compared to autoimmune type 1 diabetes. However, with increasing prevalence of obesity worldwide, type 2 diabetes in children and adolescents is increasing at an alarming pace (Jones, 2008), which is also a risk factor for hypertension, as resulted in many studies (Sarnblad et al., 2003; Kong and Chan, 2010).

1.4.3.11. Dyslipidemia: Typically dyslipidemia in children and adults with obesity and insulin resistance include increased triglyceride and decreased high-density lipoprotein (HDL) cholesterol levels. Low density lipoprotein (LDL) cholesterol remains to be the primary target of lipid control to prevent cardiovascular events in adults (Grundy, 2002). Hence, majority of randomized controlled trials carried out in pediatric populations have also focused on the use of statins in youth with elevated LDL cholesterol levels. There is
Introduction

general consensus that statin should be initiated, in combination with diet and lifestyle modification. if LDL cholesterol level > 4.1 mmol/l (i.e. 160mg/dl) in at-risk youth (McCrindle et al., 2007; Mcneal and Wilson, 2008). Fibrates and niacin are lipid lowering drugs targeted to treat high triglyceride and low HDL cholesterol in adults, but neither drugs is approved for use by U.S. Food and Drug Administration (FDA) in the pediatric population.

Remarks

In conclusion, hypertension is an increasing world health problem. In view of the substantial tracking of its risk factors from adolescents to adulthood, there is an urgent need to intervene early with efficacious strategies to identify and treat the youth with cardiovascular risk factors. The traditional hypertension risk factors, namely overweight/obesity, family history diabetes and dyslipidemia do not account for all cardiovascular deaths and novel factors, including lifestyle, socio-economic and environmental, as well as the consequences and interactions related to these traditional and novel risk factors appear to be important, accounting for the dramatic recent changes in prevalence and would be of public health concern. Moreover, more intensive program for lifestyle modification and aggressive approach of pharmacological treatment should be considered in the youth at-risk of cardiovascular events.

There are very few systematic and population based studies related to blood pressure and anthropometric measurements available in adolescent population. However, it is reported that developments of cardiovascular diseases have their origin in adolescence time (Soudarssanane, 2008). Therefore, it is clear that many traditional risk factors such as higher blood pressure, obesity and related metabolic determinants are significantly prevalent among adolescent age group. It is assumed that high risk adolescents are likely to become high risk adult (Dietz, 1998a,b). Therefore, the study of the present project related to identify the trends of blood pressure in adolescence which is the marker of future population burden of cardiovascular disease among Punjabi adolescent population. Therefore, the present study enables the complete understanding of the dynamics of blood pressure with respect to many quantitative traits.
Hence, the major objectives of the present study are as follows:

(i) to examine the prevalence of pre-hypertension and hypertension and to provide overall essential base-line data for cardiovascular disease among the study population

(ii) to analyze the trend of blood pressure variations with respect to different anthropometric, physiometric and socio-economic lifestyle risk factors to track the cardiovascular diseases in different adolescent age groups in the urban population in Punjab

(iii) to clarify that which of the anthropometric and socio-economic lifestyle variables are the best predictors for cardiovascular diseases and also to determine the magnitude of association with gender in adolescent population in Punjab.

**Hypothesis to be tested (H₀)**

(i) No association of rich battery of relevant anthropometric and socio-economic lifestyle variables with SBP and DBP in a multivariate system in adolescent population.

(ii) None of the anthropometric and socio-economic lifestyle risk factors are the best predictor for cardiovascular diseases among adolescent population.

**Alternative hypothesis (H₁)**

(i) Rejection of first hypothesis confirms impact of complex mode of anthropometric and socio-economic lifestyle variables on variation of SBP and DBP.

(ii) Rejection of second hypothesis implies any one or combined anthropometric and socio-economic lifestyle risk factors would be the best predictor of cardiovascular diseases in adolescent population.