Hypertension (HT) is the most prevalent, complex quantitative trait which is characterized by chronic increase in the blood pressure. High blood pressure is one of the known risk factor for stroke, coronary artery disease, congestive heart failure, end-stage renal and cerebrovascular disease. About one billion of the world adult population suffered hypertension in the year 2000; but this data is expected to increase by 29% which is 1.56 billion by the year 2025 (Kearney et al., 2005). HT is directly responsible for 57% of all death due to stroke and 24% of all deaths due to coronary heart disease (Gupta and Gupta, 2009). Treatment of HT at an early stage has been associated with 40% reduction in the risk of stroke and 15% reduction in the risk of myocardial infarction (Whitworth, 2003).

Essential hypertension (EH) is the most common condition which contributes to ~ 95% of cases and has no clear single identifiable cause. In contrast, secondary hypertension affects a small proportion of individuals (2 - 5%) and has an underlying medical cause including renal or adrenal disease (Beevers et al., 2001). Monogenic forms of hypertension, familial studies, twin studies and cross transplantational studies in experimental models have revealed the association of genetic components with essential hypertension. Existing evidences also suggest that the genetic contribution to blood pressure variation is about 30-50% (Marteau et al., 2005). Abdominal obesity, dyslipideamia, glucose intolerance, hyperinsulinemia and hyperuremia aggregates with EH possibly because of the common physiological mechanisms involved (Staessen et al., 2003).
Although, EH has a strong genetic basis, the identification of genes associated with it has been difficult to achieve because of the complexity of regulation of blood pressure, its multifactorial nature and the presence of multiple susceptibility genes that have profound environmental and gene–gene interactions (Bianchi and Cussi, 2000). Genetic dissection of such complex trait has been greatly facilitated by the sequencing of human genome. Several approaches have been developed to identify genes associated with inter-individual variation in blood pressure over a general population. Among the widely used methods, linkage analysis and association studies form the core for locating susceptible genes in humans (Nolte et al., 2010).

Linkage analysis evaluates the co-segregation of traits or genetic markers within family members. It has proved to be very successful in the case of single gene disorders. Risch and Merikangas (1996) showed that linkage analysis has low resolution/limited power to identify genes associated with complex traits like essential hypertension. As a consequence, gene-finding efforts for complex traits mostly rely on association approaches such as candidate gene approach or genome wide association study (GWAS).

The scenario of multiple regulators of blood pressure leads to the prediction that many gene variants have the potential to make individuals either susceptible or resistant to hypertension (Halushka et al., 1999). In this context, large-scale genome-wide association studies (GWAS), wherein thousands of common genetic variants are genotyped, analyzed for their disease association, have shown great success in identifying genes associated with common diseases and traits (McCarthy et al., 2008). Genetic variants may influence blood
pressure variation by modifying the structure of encoded proteins or by altering the gene expression (Levy et al., 2009). Thus, the scope of GWAS lies in its potential to define undiscovered physiological pathways that will offer newer approaches not only to prevent or treat the disorder but also to augment existing therapeutic strategies (Harrap and Petrou, 2001).

Variable gene expression in a population could account for some of the differences in either susceptibility to common diseases or response to drug treatments (Zhang et al., 2008). Genes involved in blood pressure regulation exhibit both ethnic and regional specificity. Kazakh and Uygu r are two ethnic groups from China exhibiting variations in the prevalence and onset of hypertension. Kazakh group had the highest prevalence rate of 17.36% which is also characterized by an early onset, whereas, Uygur group had a prevalence rate of 10.33% which is below the average level in National prevalence rate of hypertension (Zhong et al., 2011).

Genetic variants and mutations studied for decades globally have indicated both positive and negative associations with EH. A classical example being the variants of angiotensiogen genes of the renin-angiotensin-aldosterone system (RAAS pathway), which exhibited both negative (Kato et al., 2000; Rodriguez-Perez et al., 2000) and positive (Hata et al., 1994; Chiang et al., 1997) correlations in different populations/groups. Probably, this is because of the effects of variants under study might be effectively masked by effects of unknown variants that affect the phenotype (Templeton, 2000; Moore and Williams, 2002). Therefore, cumulative effects of multiple candidate variations may provide more information in exploring hypertension susceptibility genes.
Currently available antihypertensive drugs are effective only in about 40 - 60% of hypertensive patients. Such variability in the drug efficacy may precipitate in as poor BP control (Materson, 2004) implying that hypertension management is not optimal. Evaluation of genetic determinants of BP, identification of susceptible loci, analyzing their functional role and pharmacokinetics of the drugs would guide the choice of the most promising antihypertensive drug as a personalized medicine (Johnson et al., 2009).

To some extent susceptible loci for hypertension although have been mapped, their association is found to affect only a small section of the total variation in SBP (1mm Hg per allele) or DBP (0.5mm Hg per allele) individually. But the conjoint effect of the potential risk alleles identified would be good enough to increase the cardiovascular risk (Cheh et al., 2009). When such risk alleles culminate at a physiological point, it becomes a prime target for therapeutic intervention.

Recent developments in the field of genetics of hypertension have exposed many novel genes but their physiological role in the development of EH still remains to be understood. Genes encoding ion channel transporters, immunomodulators, adhesion and fusion proteins, solute carrier proteins etc., have shown significant association with EH in various populations. The present study is an effort to analyze some of those genetic variants which could be directly or indirectly linked to EH. Knowledge about the polymorphic markers of these genes could pinpoint novel avenues to target identification and development of effective drugs for the everincreasing hypertensive population worldwide.