# Preface

It is my real pleasure to edit this special issue on Cancer Genetics. Cancer is a multi-stage disease. In each stage there is clonal evolution of specific clone due to accumulation of different genetic/epigenetic changes. Due to successful progression of Human Genome Project, cancer research is now in a new momemtum in identifying the cancer associated genes. Depending on function, these genes can be classified as onco-genes, tumor suppressor genes, predisposing/susceptibility genes, protector genes and replica-tion fidelity genes. The etiological factors that are highly associated with human cancer are a) physical agents like UV/ ionizing radiation, b) chemical agents mainly alcohol, polycyclic aromatic hydrocarbons (PAHs), different nitroso compounds etc, and c) viral agents e.g. HPV, HBV, EBV etc. The physical and chemical agents mainly interact with DNA leading to the accumulation of mutations due to impairment of efficient repair systems. The viral agents code different tumor associated antigens that interact with different cellular proteins that cause dysfunction of the DNA repair system, chromosomal recombination and segregation procedures, and ultimately deregulation of cell cycle. Also, susceptibility differences of different individual to the chemical and viral agents due to genetic polymorphism are assumed to be important factors in the tumor predisposition. Thus, to get an idea about the recent trend in cancer research different aspects of cancer genetics have been discussed in this issue of *International Journal of Human Genetics*.

In the first review, Sengupta et al. described the normal repair system of double strand break (DSB) and discussed how the dysfunction of this repair system leads to the genomic instability and cancer development. They suggested that the genetic polymorphism of the DSB repair pathway genes might be the predisposing factor in the development of cancer. In the subsequent review, Nair et al described how the two HPV associated antigens E6 and E7 interact with the two cell cycle regulatory proteins p53 and RB, and the Pro/Arg polymorphism of p53 affects E6 interaction. They have also suggested that the specific E6 gene variants as well as the genetic polymorphism of some metabolic enzymes may increase the risk of HPV associated uterine cervical cancer. In an another study of Bhattacharya et al, the specific genetic polymorphism of a HLA locus i.e. HLA DQB1*03, has been assumed to be the important factor in determining the susceptibility to HPV or HPV16/18 related cervical cancer in Indian women. On the other hand, it has been shown by Chatterjee et al that the treatment of cervical cancer patients either by surgery or radiation significantly reduces the prevalency of HPV infection. Similar to HPV infection, the polymorphisms in the MHC class II alleles, promoter region of interleukin-10 and mannose binding protein (MBP) are suggested to be associated with the HBV infection (R. Chakraborty). On the other hand Sikdar et al showed that the tobacco users having GSTM1 null genotype are more susceptible to the development of oral cancer. Interestingly, Ray et al have showed that the firefighters having long duration of service are more prone to the development of cancer due to the significantly high incidence of micronucleus in their exfoliated buccal epithelial cells. In undestanding the molecular mechanism of tumorigenesis Bhattacharya et al showed that c-myc oncogene (located in chromosomal 8q24.1 region) activation can be occurred due to the distant 5' or 3' alteration in the pal-1 and mlvi-4 regions like to that have been seen in some hematological malignancies, and this c-myc locus alteration is not associated with the deletion in chromosome 8p. On the other hand, Mukherjee et al showed that enoxacin, a fluoroquinoline antibiotic, inhibits the breast cancer cell line MCF-7 at G2/M phase of the cell cycle indicating the potential importance of this drug in the treatment of cancer.

Thus, it is hoped that the topics discussed in this special issue by various experts may give some idea to the readers about the current trend in the research of cancer genetics and help them to generate new thoughts.

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