

Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome (HIV/AIDS) is a major public health problem, socio-economic burden and a serious threat to development. Entry of human immunodeficiency virus type I (HIV-1) into target cells requires the binding of the external envelope glycoprotein gp 120 to both the CD4 molecule and one of several chemokine receptors, that function as co-receptors. T-cell line tropic HIV-1 strains utilize the α -chemokine receptor CXCR4, whereas the β -chemokine receptor 5 (CCR5), which is expressed on monocytes/macrophages, T cells and granulocyte precursors, is the key co-factor for macrophage-tropic HIV-1 strains, which predominate during the asymptomatic phase of infection. A thirty two-base pair (bp) deletion mutation (A 32) within the second extra cellular loop-encoding region of the CCR5 gene, which results in a truncated, non functional protein, has been associated with relative resistance to HIV -1 infection and slower progression to acquired immunodeficiency syndrome (AIDS). Specifically, A32/ Δ 32 homozygotes are protected against acquisition of HIV-1 by the mucosal route despite high risk exposure, whereas disease progression among CCR5/ Δ 32 heterozygote occurs more slowly. In this study, the status of the CCR5 gene polymorphism in the sampled population from Kenya was investigated in an attempt to explain the differences in HIV prevalence in different parts of the country. To determine this, 200 samples were collected from the 8 provinces of Kenya, that is, 25 samples per province, some of which were positive for HIV-1. Twentyfive samples were randomly selected from a batch of 250 per province, that is, every tenth sample. The samples were collected from HIV screening centres, district and provincial hospitals. Peripheral blood mononuclear cells (PBMC) were extracted from whole blood. Genomic deoxyribonucleic acid (DNA) was then extracted from PBMC. A targeted region of the CCR5 gene flanking the 32bp deletion was amplified by polymerase chain reaction (PCR) using CCR5 specific primers. All the PCR amplicons were then analyzed by gel electrophoresis. The results showed that CCR5- Δ 32 mutations do not exist in the sampled population from Kenya. Samples were then randomly selected, 4 samples per province and sequenced. This was done to determine the genotype of the PCR products that were amplified. After ClustalW analysis of the sequences generated, it was seen that CCR5 gene is not highly conserved in the sampled population from Kenya, as there were amino acid differences between the sequences analyzed suggesting that CCR5 gene in the sampled population from Kenya is highly polymorphic. Based on the samples analysed, it was evidenced that CCR5- Δ 32 mutations does not exist in the sampled population from Kenya. The disparity in HIV distribution observed in different parts of Kenya may not be attributed to CCR5- Δ 32 mutation. The differences in prevalence of HIV in different parts of the country may be due to cultural practices, religious backgrounds, socio-economic status and other intrinsic genetic factors.