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Editorial

Phylogenetic and Genotypic Analytic Insights of HIV-1

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In vivo, human immunodeficiency virus type 1 (HIV-1) is typically an unusually high degree of variability. Three main groups of HIV-1 are M (major), N (new) and O (outlier). M group of HIV-1 has been subdivided into subtypes A-J by genetically clusters of HIV-1 env genes. "Unclassifiable" or "uncertain" (category U) variants of HIV-1 has been reported that may represent a recombinant sequence or a new subtype. The env region of HIV-1 is principally targeted for HIV-1 subtyping to identify new and old variants. Analysis of the C2-V3 segment of env is the most common procedure used for HIV-1 subtyping. An analysis of larger env sequences has demonstrated that limited V3 region sequences reliably determine HIV-1 subtypes whereas most polymerase chain reaction (PCR)-derived sequences contain a suboptimal length for phylogenetic analysis. These shorter fragments of genome still remain the primary technique used to monitor HIV-1 genetic diversity around the world. Globally, different sets of primers are urgently needed to optimize the efficiency of PCR amplification and sequencing due to the broad heterogeneity within the C2-V3 domain of HIV-1 group M viruses and the constant changes of nucleotides in this region over time. Reverse transcriptase (RT) and/or protease (PR) genes are particularly significant when there is presence of genetic variability in HIV. High genetic variability of HIV-1 indicates high error rates of the viral RT, high replicative rate of the virus, high circulating recombinant forms (CFRs), and viral plasticity under selective host and drug pressure. There are transmission of a single monophyletic variant to most newly-infected individuals (> 95%) although HIV continually adapts in infected humans. Distinct HIV subtype and quasispecies have also been identified in HIV-1-infected persons due to high degree of HIV genetic variability. HBV or HCV co-infection in HIV-infected patients previously has been related to patient deaths.

Currently, there is still no information of HIV RT and PR genetic variability that is indicators of HIV-drug resistance in HIV-infected patients co-infected with Hepatitis B (HBV) or Hepatitis C (HCV) Virus. A previous study revealed that the most frequent amino acid substitutions in RT were L214F (67.6%), I135T (55.9%) and in PR was V15I (41.2%). In a previous study in Australia, phylogenetic research has revealed that individuals with recent HIV infection drive transmission clustering among Men-having-Sex with Men (MSM) and intravenous drug users (IDU), nevertheless, approximately represents 10% to 65% of the estimates. A previous study demonstrated that the M184V mutation conferred HIV resistance to Lamivudine (3TC) and Emtricitabine (FTC) that is often the first to emerge in the aftermath of HIV treatment failure.

In conclusion, phylogenetic analysis can trace the association of viral variants at a population-level, providing a molecular epidemiological surveillance for HIV transmission dynamics and can map the geographic distribution and domestic expansion of viral subtypes in different areas around the world.

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