Clonal Analysis of the gp120 V3 Loop from Clinical Isolates Displaying Phenotypic Resistance to Vicriviroc

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Abstract

Background: Understanding the molecular mechanisms of resistance to antiviral agents is essential for the development of novel therapeutic options. Resistance to the HIV-1 entry inhibitor vicriviroc (VCV) is mediated in part by mutations in the gp120 V3 loop.

Objectives: To investigate the relationship between VCV susceptibility and mutations in the gp120 V3 loop from clinical isolates with reduced susceptibility to VCV.

Methods: A panel of clinical isolates with reduced susceptibility to VCV was characterized for mutations in the gp120 V3 loop. VCV susceptibility was determined by an assay referenced to the u03672 strain and compared to the Susceptibility Test Reference Virus (JrCSF) parallel, controls for day-to-day fluctuations in the assay.

Results: The data presented in Table 1 illustrate the susceptibility phenotypes of the clinical isolates analyzed. A significant correlation was observed between the clinical strain susceptibility data and the presence of specific mutations in the gp120 V3 loop. In general, isolates with reduced susceptibility to VCV were associated with specific gp120 mutations, particularly in the V3 loop. These findings support the hypothesis that VCV resistance is mediated by mutations in the gp120 V3 loop.

Conclusions: The results of this study provide evidence that mutations in the gp120 V3 loop are associated with reduced susceptibility to VCV. These findings support the development of future research aimed at understanding the molecular mechanisms of VCV resistance and identifying new therapeutic strategies.