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Drug Resistance in HIV through Anti-retroviral Therapy

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INTRODUCTION

HIV drug resistance has a negative influence on how well Antiretroviral (ARV) medications work to prevent and treat HIV infection. Antiretroviral Treatment (ART) programme expenditures, new HIV infections, and mortality related to HIV/AIDS all rise as a result of HIV medication resistance. Therefore, meeting the 95-95-95 objectives and eliminating AIDS as a public health issue by 2030 depend on the surveillance, prevention, and control of HIV medication resistance. The World Health Organization (WHO) advises monitoring HIV medication resistance in people starting and restarting ART in order to choose appropriate prophylactic regimens and first-line ART treatments that are efficacious. In Latin America and the Caribbean, the frequency of HIV medication resistance among people starting and restarting ART has increased: The likelihood of HIV medication resistance to Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTI) increased by 11% year from 2007 to 2016. In addition, persons who have previously used ARV drugs have been found to have considerably greater rates of NNRTI resistance than adults who have never used ARV drugs. For people starting or restarting ART, some low-and middle-income countries have reported a nationally representative prevalence of HIV drug resistance to Efavirenz (EFV) or Nevirapine (NVP) exceeding 10%, the WHO-recommended cutoff point to encourage switching to a non-NNRTI-containing regimen.

DESCRIPTION

In Uruguay, persons with HIV who were starting or restarting antiretroviral medication participated in the first nationally representative cross-sectional HIV Drug Resistance (HIVDR) study in 2018-2019. Sequencing of HIV-1's protease, reverse transcriptase, and integrase genes was done. A total of 206 people signed up for the survey; 63.2% of them were men, 85.7% were older than 25, and 35.6% admitted to having used Antiretroviral (ARV) medications in the past. Individuals with prior ARV medication exposure had a considerably greater rate of HIVDR to efavirenz or nevirapine than adults without prior ARV drug exposure. HIVDR to any inhibitors of nucleoside reverse transcriptase was 10.3%. HIVDR to ritonavir-boosted protease inhibitors was 1.5%, resistance to ritonavir-boosted darunavir was 0.9% (0.4%-2.1%), and it was not noted in people who had previously used ARV medications. HIVDR to dolutegravir, bictegravir, and cabotegravir was not detected, despite HIV resistance to integrase inhibitors being 12.7%. The high rate (>10%) of HIVDR to efavirenz emphasises the necessity of hastening the switch to the dolutegravir-based ART that the WHO advises. People who report prior ARV medication exposure should have priority access to dolutegravir-based ART.

You may think of the glycoproteins on the surfaces of enveloped viruses like HIV as a special target for antiviral treatment. Different Carbohydrate-Binding Substances (CBAs) target certain viral glycoproteins of enveloped viruses that include glycans. Long-term CBA pressure *in vitro* has been demonstrated to produce mutant HIV-1 isolates with a number of N-linked glycan deletions on gp120. These analyses showed that the majority of the deleted glycans are of the high-mannose type. It's noteworthy to note that despite lengthy CBA exposure, N241, N262, and N356 on gp120 have never been reported to be impacted. In this article, we examine the mutation and (cross) resistance characteristics of eleven distinct, CBA-resistant HIV-1 strains.

The produced CBA-resistant HIV-1 clade B isolates showed that the broad-neutralizing anti-carbohydrate binding mAb 2G12 entirely lost its ability to inhibit them. Furthermore, every CBA mentioned in this review with the exception of NICTABA prevented the 2G12 mAb from binding to gp120 produced on HIV-1-infected T cells. Even among different classes of CBAs with various binding moieties or sugar specificities, the

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cross-resistance profiles of mutant HIV-1 strains range from increased susceptibility to extremely high resistance levels. Recent studies highlighted the potential of non-topical formulations for prevention (microbicides) and antiviral therapy by demonstrating promising results when administered non-topically (e.g., intranasally or subcutaneously).

CONCLUSION

Human Immunodeficiency Virus (HIV), the causative agent of acquired immune deficiency syndrome, targets $CD4^+$ immune cells as its primary target cells (AIDS). When the biological CD4 receptors on T helper cells (Th cells), Dendritic Cells (DCs), monocytes, and macrophages connect with the viral envelope glycoproteins gp120, infection begins. To achieve subsequent binding with the cellular chemokine receptors CCR5 or CXCR4 for, respectively, CCR5-trin vivoopic (or R5) and CXCR4-tropic (or X4) HIV-1 strains, the first gp120/CD4 contact causes a first series of conformational changes inside gp120. Following these coreceptor contacts, a second series of conformational modifications leads to the exposure of gp41, which together with the endocytic machinery of the host cell triggers membrane fusion by forming the six-helix-bundle.