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Editorial

An Overview on Designing Potential Antiretrovirals for HIV

Uddhav Jordan*

Research Institute of Virology, Tashkent, Uzbekistan

EDITORIAL

Recently acquired knowledge about the HIV entry process points to new strategies to block viral entry. For most HIV strains, the successful infection of their target cells is mainly dependent on the presence of the CD4 surface molecule, which serves as the primary virus receptor [1]. The attachment of the viral envelope to this cellular CD4receptor can be considered as an ideal target with multiple windows of opportunity for therapeutic intervention. Therefore, drugs that interfere with the CD4 receptor, and thus inhibit viral entry, may be promising agents for the treatment of AIDS. The CD4-targeted HIV entry inhibitors Cyclotriaza disulfonamides represent a novel class of small molecule antiviral agents with a unique mode of action [2,3]. The lead compound, CADA, specifically interacts with the cellularCD4receptor and is active against a wide variety of HIV strains. CADA may also act synergistically in combination with other anti-HIV drugs. This work includes study of interaction between CADA and CD4, mode of inhibition of CD4, designing a better drug against HIV also with antibacterial activity adding value to the entry inhibitor.

Acquired immunodeficiency syndrome (AIDS) is a caused by the human immunodeficiency virus (HIV). HIV belongs to a subset of retroviruses called lenti viruses (or slow viruses) meaning that there is an interval-sometimes years-between the initial infection and the onset of symptoms [4-6]. HIV is transmitted through direct contact of a mucous membrane or the blood stream with a bodily fluid containing HIV. Although treatments for AIDS and HIV exist to decelerate the virus' progression, there is currently no known cure. The different anti-HIV drugs available today are of three main types: NRTIs, NNRTIs and PIs. How-ever, HIV Entry Inhibitors are being a major attention.

In medicine, biotechnology and pharmacology, drug discovery is the process by which drugs are discovered and/or designed. The process of drug discovery involves the identification of candidates, synthesis, characterization, screening, and assays for therapeutic efficacy based on their biological targets. The structure of the drug molecule can be modeled using computational tools [7]. Acquired immunodeficiency syndrome (AIDS) is a caused by the human immunodeficiency virus (HIV). HIV belongs to a subset of retroviruses called lenti viruses (or slow viruses) meaning that there is an interval-sometimes years-between the initial infection and the onset of symptoms. HIV is transmitted through direct contact of a mucous membrane or the blood stream with a bodily fluid containing HIV [8]. Although treatments for AIDS and HIV exist to decelerate the virus' progression, there is currently no known cure. The different anti-HIV drugs available today are of three main types: NRTIs, NNRTIs and PIs. How-ever, HIV Entry Inhibitors are being a major attention.

References

1. Chan DC, Kim PS (1998) HIV entry and its inhibition. Cell 93: 681-4.
2. Chesebro B, Buller R, Portis J, Wehrly K (1990) Failure of human immuno-deficiency virus entry and infection in CD4-positive human brain and skincells. J Virol 64: 215-21.
3. Kabat D, Kozak SL, Wehrly K, Chesebro B (1994) Differences in CD4dependence for infectivity of laboratory-adapted and primary patient iso-lates of human immunodeficiency virus type 1. J Virol 68: 2570-7.
4. Kozak SL, Platt EJ, Madani N, Ferro FE Jr, Peden K (1997) CD4, CXCR-4,and CCR-5 dependencies for infections by primary patient and laboratory-adapted isolates of human immunodeficiency virus type 1. J Virol 71: 873-82.
5. Kwong PD, Wyatt R, Sattentau QJ, Sodroski J, Hendrickson WA (2000)Oligomeric modeling and electrostatic analysis of the gp120 envelope gly-coprotein of human immunodeficiency virus. J Virol 74: 1961-72.
6. Layne SP, Merges MJ, Dembo M, Spouge JL, Nara PL (1990) HIV requiresmultiple gp120 molecules for CD4-mediated infection. Nature 346: 277-9.
7. Platt EJ, Madani N, Kozak SL, Kabat D (1997) Infectious properties ofhuman immunodeficiency virus type 1 mutants with distinct affinities forthe CD4 receptor. J Virol 71: 883-90.
8. Platt EJ, Wehrly K, Kuhmann SE, Chesebro B, Kabat D (1998) Effects ofCCR5 and CD4 cell su rface concentrations on infections bymacrophagetropic isolates of human immunodeficiency virus type 1. J Virol 72: 2855-64.

Correspondence to: Uddhav Jordan, Research Institute of Virology, Tashkent, Uzbekistan; E-mail: uddhavjol@bk.ru

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