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Interrupting Infection by HIV

Jyoti Prakash Singh*

Sido Kanhu Murmu University, Jharkhand, 814101, India

Abstract

Over the past more than two, but not a lot of years there has been intense activity directed at the possibility of accomplishing or gaining with effort temporarily free of disease or destruction/permanent removal of HIV infection. Current tests to be tested for the measurement of hidden/covered up HIV are not enough to show or prove complete clearance of answer to do something very good HIV. Therefore, the final test/toughest test for testing/evaluating whether act of asking questions and trying to find the truth about something al actions that help bad situations have resulted in HIV temporarily free of disease or destruction/permanent removal is to interrupt standard drugs in a carefully controlled scientific fact-finding experiment setting. These procedures, known as related to careful studying or deep thinking treatment interruptions ATIs, raise important scientific and questions of right and wrong. The lack of definite tests/things to be tested for measuring viral holding tanks or areas not only makes research on HIV temporarily free of disease or cure challenging; it also affects the ability to test/evaluate risks from ATIs themselves. In spite of these challenges, basic honest and right judging requirements can be met with careful study design and close watching/supervising. In this brief report we outline standards for HIV cure research involving ATIs. These judging requirements should be revisited as the science changes. The HIV cycle presents some opportunities for interruption by virus-killing. Since 1986 a good deal of progress has been made; however, much of the information that comes out/becomes visible from studies highlights that must be overcome before effective prophylactic or medically helpful(actions that help bad situations are able to be done.

Keywords: Infection, HIV, DNA, *In Vitro*.

The earliest event in the establishment of HIV infection is the binding of the virus particle through its envelope glycoprotein (gp120) to a specific receptor on the host cell's surface. This CD4 cell receptor is found on the surface of certain members of the T lymphoid and macrophage-monocyte cell lineages. The range of cells that are susceptible to HIV-1 infection, both in vitro and in vivo, appears to parallel those that display the CD4 surface receptor. HIV-2 also uses the CD4 molecule as its receptor in the initiation of infection. The region of the gp120 molecule that interacts with the CD4 molecule recently has been defined; the identification of the corresponding binding domain of CD4 is being actively pursued. Potential strategies to inhibit the gp120-CD4 interaction, and thus HIV infection, include vaccination to elicit antibodies that recognize and bind to the critical receptor-binding domain of the HIV envelope and the fear/stopping of behavior of cell surface binding through competition with appropriate coming from the outside of something ly added pieces of the CD4 protein. Recent studies have shown the ability to actually be done of the last thing just mentioned approach by successfully stoping HIV infection *in vitro* with an able to be dissolved in something form of the CD4 molecule produced through recombinant DNA methods.

After HIV binds to the CD4 receptor, it appears to enter the host target cell by direct fusion of the viral and cellular plasma membranes. It is thought that this process requires a hydrophobic domain on the HIV gp41 that assumes an active fusogenic conformation following cleavage of the gp160 precursor molecule. One important manifestation of the cytopathic consequence of HIV infection in vitro involves a specific interaction between the HIV envelope glycoprotein complex and the CD4 molecule that results in the fusion and subsequent death of cells that have CD4 receptors. This process, which is known as syncytia formation, may also involve the fusogenic domain of gp41. The development of approaches to inhibit envelope-mediated membrane fusion may lead to novel ways of preventing HIV infection and its consequences. The process inhibiting HIV replication. The necessary proteolytic cleavage of the gag-pol polyprotein precursor is one possible target, and its mechanism is currently undergoing active analysis. Likewise, the proteolytic processing of the envelope polyprotein gp160, which is necessary for HIV infectivity, is a susceptible stage for inhibition. The glycosylation of the HIV envelope protein also appears to be necessary for viral infectivity, and drugs that interfere with this process have demonstrated antiviral activity *in vitro*.

***Address for Correspondence:** Jyoti Prakash Singh, Sido Kanhu Murmu University, 814101, India, Email: singh.jyoti@gmail.com*

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