therapeutic regimens is the emergence of HIV mutants that are resistant to single or even multiple medications. Such drug-resistance HIV works against the population in two ways. First, the infected individual will eventually run out of treatment options; and second, if the infected individual passes along a virus already resistant to many existing therapeutic agents, the newly infected individual will have a more limited treatment option.

[0024] The HIV-1 replication cycle can be interrupted at many different points. As indicated by the approved medications, viral reverse transcriptase and protease enzymes are good molecular targets, as is the entire process by which the virus fuses to and injects itself into host cells. Thus the recently approved drug T20 (Fuzeon) is the first in a novel class of anti-HIV-1 agents. However, in addition to the drugs already approved for treatment of HIV-1 infection, work continues on the discovery and development of additional treatment modalities. This is necessary because of the propensity of the virus to mutate and thus render ineffective the existing therapies.

[0025] The search for chemotherapeutic interventions that work by novel mechanism(s) of action is particularly important in the search for new medications to combat the spread of the HIV. Several potential areas for intervention that are under consideration or have active programs include 1) blocking the viral envelope glycoprotein gp120, 2) additional mechanisms beyond gp120 to block virus entry, such as blocking the virus receptor CD4 or co-receptors CXCR4 or CCR5, 3) viral assembly and disassembly through targeting the zinc finder domain of the viral nucleocapsid protein 7 (NCp7) and 4) interfering with the functions of the viral integrase protein and interrupting virus specific transcription processes.

[0026] The mechanism by which HIV passes through the mucosal epithelium to infect underlying target cells, in the form of free virus or virus-infected cells, has not been fully defined. In addition, the type of cells infected by the virus could be derived from any one, or more, of a multitude of cell types (Miller, C. J. et al. "Genital Mucosal Transmission of Simian Immunodeficiency Virus: Animal Model for Heterosexual Transmission of Human Immunodeficiency Virus."*J. Virol.* 63:4277-4284 (1989); Phillips, D. M. and Bourinbaiar, A. S. "Mechanism of HIV Spread from Lymphocytes to Epithelia."*Virology* 186, 261-273 (1992); Philips, D. M., Tan X., Pearce-Pratt, R. and Zacharopoulos, V. R., "An Assay for HIV Infection of Cultured Human Cervix-derived Cells."*J. Virol. Methods,* 52, 1-13 (1995); Ho, J. L. et al, "Neutrophils from Human Immunodeficiency virus (HIV)-seronegative Donors Induce HIV Replication from HIV-infected patients Mononuclear Cells and Cell lines. An In Vitro Model of HIV Transmission Facilitated by *Chlamydia Trachomatis." J. Exp. Med.,* 181, 1493-1505 (1995); Braathen, L. R., and Mork, C., in "HIV infection of Skin Langerhans Cells", In: Skin Langerhans (dendritic) cells in virus infections and AIDS (ed Becker, Y.) 131-139, Kluwer Academic Publishers, Boston, (1991)). Such cells include T lymphocytes, monocytes/macrophages and dendritic cells, suggesting that CD4 cell receptors are engaged in the process of virus transmission as is well documented for HIV infection in blood or lymphatic tissues (Parr M. B., and Parr E. L., "Langerhans Cells and T lymphocytes Subsets in the Murine Vagina and Cervix."*Biology and Reproduction* 44,491-498 (1991); Pope, M. et al. "Conjugates of Dendritic Cells and Memory T Lymphocytes from Skin Facilitate Productive Infection With HIV-1."*Cell* 78, 389-398 (1994); and Wira, C. R. and Rossoll, R. M. "Antigen-presenting Cells in the Female Reproductive Tract: Influence of Sex Hormones on Antigen Presentation in the Vagina."*Immunology,* 84, 505-508 (1995)).

[0027] Therefore, the need for efficacious, safe, and inexpensive anti-viral agents to treat or prevent the transmission of HIV (in lieu of a vaccine) is evident.

[0028] Besides HIV, herpes viruses also infect humans ("Herpesviridae; A Brief Introduction", *Virology,* Second Edition, edited by B. N. Fields, Chapter 64, 1787 (1990)) and cause STDs. Some common herpes viruses are described below. However, the list is not meant to be exhaustive, but only illustrative of the problem.

[0029] Herpes Simplex Virus Type 1 (HSV1) is a recurrent viral infection characterized by the appearance on the cutaneous or mucosal surface membranes of single or multiple clusters of small vesicles filled with clear fluid on a slightly raised inflamed base (herpes labialis). In addition, gingivostomatitis may occur as a result of HSV1 infection in infants (Kleymann, G., "New antiviral drugs that target herpes virus helicase primase enzyme." Herpes 10:46-52 (2003); "Herpesviridae; A Brief Introduction", *Virology,* Second Edition, edited by B. N. Fields, Chapter 64, 1787 (1990)).

[0030] Herpes Simplex Virus Type 2 (HSV2) causes genital herpes, and vulvovaginitis may occur as a result of HSV2 infection in infants (Kleymann, G., "New antiviral drugs that target herpes virus helicase primase enzyme." Herpes 10:46-52 (2003)).

[0031] Human Cytomegalovirus (HCMV) infections are a common cause of morbidity and mortality in solid organ and haematopoietic stem cell transplant recipients (Razonable, R. R., and Paya, C. V., "Herpes virus infections in transplant recipients: current challenges in the clinical management of cytomegalovirus and Epstein-Barr virus infections." Herpes 10:60-65 (2003)).

[0032] Varicella-Zoster Virus (VZV) causes varicella (chickenpox) and Zoster (shingles) (Vazquez, M., "Varicella Zoster virus infections in children after introduction of live attenuated varicella vaccine."*Curr. Opin. Pediatr.* 16:80-84 (2004)).

[0033] Epstein-Barr virus (EBV) is the causative agent of infectious mononucleosis and has been associated with Burkett's lymphoma and nasopharyngeal carcinoma. Human Herpes virus 6 (HHV6) is a very common childhood disease causing exanthem subitum (roseola) (Boutolleau, D., et al., "Human herpes virus (HHV)-6 and HHV-7; two closely related viruses with different infection profiles in stem cell transplant recipients", *J. Inf. Dis.* (2003)).

[0034] Herpes Simplex Virus Type 7 (HSV7) is a T-lymphotropic herpes virus and can cause exanthem subitum. The pathogenesis and sequelae of HH7, however, are poorly understood (Dewhurst, S., Skrincosky, D., and van Loon, N. "*Human Herpes virus* 7", Expert Rev Mol. Med. 18:1-10 (1997)).

[0035] Herpes Simplex Virus Type 8 (HSV8) is another herpes virus. The molecular genetics of the human herpes virus 8 (HHV8) has now been characterized, and the virus appears to be important in the pathogenesis of Kaposi's