Decelerating HIV-1Viral Escape by Inhibiting Entry into CD4 T Cells

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Viral escape is a major challenge for lymphocytes to develop an immune response for neutralizing HIV viruses. HIV mutates rapidly where immune cells cannot recognize the modified epitopes of HIV viruses. Decelerating the viral escape of HIV-1 could serve to provide more time for a therapeutic vaccine to produce an immune response for neutralizing the virus. This review attempts to advocate for an inclusion of antiviral methods in a therapeutic vaccine design that can prolong the viral escape of HIV-1. Delaying viral escape can enhance the effectiveness of HIV-1 therapeutic vaccination. The gene editing of T cells through CCR5 disruption can lower the frequency of infection. A lesser rate of infection due to CCR5 disruption can reduce the rate of viral escape when HIV-1 is prevented from entering and infecting naive HIV-resistant CD4 T cells. An antibody-like CD4 T cell entry inhibitor called eCD4-Ig can trap the HIV virus by mimicking the CD4 T cell receptor and then lock in the virus. HIV cannot quickly escape from eCD4-Ig because HIV mutations to evade eCD4-Ig would need to be produced at a higher fitness cost. This delay may offer more time for antivirals and a therapeutic vaccine induced immune response to recognize and neutralize the virus.

The time of the initial HIV-1 infection plays a major role in the efficacy of antivirals and determines the kinetics of HIV-1 viral escape. Combining gene disruption of CCR5 expression on CD4 T cell surfaces and an eCD4-Ig HIV-1 entry inhibitor with a therapeutic vaccine may delay viral escape and control HIV-1 replication, which can possibly provide a functional cure for HIV.

Biography:

Tatiana Hillman is an independent researcher for The LAB INC., in Los Angeles, California. The LAB INC. is a private biotechnology company where she conducts microbiology research with an emphasis on synthetic biology and genetic engineering. She has designed projects such as inhibiting Gram-negative bacterial metabolic genes, required for fatty acid synthesis and lipid membrane assembly, to decrease quorum sense signaling, reduce bacterial growth, and to lower the antibiotic resistance of Escherichia coli cells. She believes many infectious diseases such as multi-drug resistance of bacteria and HIV have a pathogenicity that is gene-based and may require a gene-based solution. For this reason, she hopes to further study methods of recombinant DNA technology and synthetic biology for treating infectious diseases.