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Commentary

The Development of Curative Therapy for Elimination of HIV

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DESCRIPTION

After years of antiretroviral medication, Human Immunodeficiency Virus type 1 (HIV) infection remains Antiretroviral Therapy (ART). Approaches to eliminate or cure HIV infection are required in order to remove the stigma and burden of chronic infection. Efforts to supplement ART with medicines that reverse viral latency, in combination with immunotherapies to remove infection, have progressed to the clinic, although the field is still in its infancy. Future medicines that remove HIV infection, in conjunction with breakthroughs in ART administration and HIV preventive efforts, may relieve society of the burden of the HIV epidemic.

Human immunodeficiency virus type 1 has killed approximately 50 million people and caused widespread misery. As the pandemic spread, a remarkable response by clinicians, researchers, social activists, the pharmaceutical industry, and government officials resulted in the development and implementation of potent ART, capable of arresting disease, restoring health, and reducing the spread of new infection. Long-acting antivirals and engineered antibodies are now in advanced clinical studies, with the prospect of replacing daily tablets for both treatment and prevention with only a few doses per year. Although subsequent efforts to recreate the success of RV144 have recently failed with the early closure of HVTN 702, sequential prime and boost vaccinations may speed the generation of broadly neutralizing Antibodies (bnAbs) that might lower the incidence of new infection throughout the world.

If these possible breakthroughs are applied properly over the world, the impact of the HIV epidemic will be considerably lessened. Millions, however, will continue to be plagued by decades of chronic medical therapy and the stigma of HIV-1 infection, with the consequent cost on global health systems. Treatment that may result in a cure, or short of viral eradication, allow for long-term and strict immunological control without the need for medication ("functional cure"), would be a game changer for the millions of people living with HIV. The main impediment to HIV treatment is an infected population of long-lived cells with persistent and latent viral genomes that cannot be recognized or removed by host defences. Earlier decades of research discovered various molecular pathways that generate and maintain this retrovirus's post-integration latency. Many analogous attempts have now been launched across the world.

The last decade of study has resulted in a better knowledge of the molecular and cellular underpinnings of HIV latency, the development of innovative assays to increase the capacity to assess the latent reservoir, and investigations in HIV latency animal models. While other efforts have sought to develop cellular or gene therapies to control or clear infection, strategies to permanently silence viral genomes or induce apoptotic death in infected cells, or strategies to induce viral remission in the absence of viral eradication, this overview will focus more narrowly on efforts to target and eliminate HIV infection's persistent reservoirs in order to develop curative therapy. Although human pilot efforts to cure HIV latency and diminish the reservoir of persistent infection have begun, there is still much to learn and much to accomplish.

A chosen handful of the innumerable infection events that occur within an untreated HIV-infected individual culminate in the integration of a completely intact, functioning provirus that produces persistent infection with low viral gene expression. The majority of viral genomes that can be evaluated are faulty due to mistakes in viral reverse transcription that result in tiny deletions or mutations, or substantial deletions produced by the host APOBEC3 proteins. The few complete viral genomes that have survived remain in cellular reservoirs. These latent proviruses can, by definition, return to a productive form *in vivo*, but during latency they are unaffected by ART and cannot be recognized by the host immune response. This condition of proviral latency has been quantified in HIV-infected people peripheral blood and lymphoid tissues, and a variety of molecular pathways that allow the formation and maintenance of persistent, latent HIV infection have been characterized. The fact that cellular components are necessary to sustain quiescence suggests that proviral latency is an unstable state of HIV infection that is susceptible to therapeutic intervention.

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