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HIV: Current Research

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Perspective

Global Health Priority over HIV Research

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DESCRIPTION

As it is well known, the population dynamics of infectious diseases have been extensively studied in recent years. HIV (Human Immunodeficiency Virus) in particular has been extensively studied in and has become a global problem. HIV infection is divided into three stages: primary infection, clinically asymptomatic stage (chronic infection), and Acquired Immunodeficiency Syndrome (AIDS) or drug therapy.

During primary infection, viral load in the peripheral blood increases significantly to a peak level, then declines to a steady state, known as the viral set point. During primary infection, extremely high viral loads activate T cells, which are known as Cytotoxic T cells (CTL) capable of suppressing viral replication.

The decline from the peak is due to immune cell control and/or limited target cell availability. Clinical research in conjunction with mathematical modelling has advanced our understanding of HIV-1 infection.

This is due to the fact that mathematical models can be used to study the dynamics of viral load *in vivo* and are very useful in understanding the interaction between virus and host cell.

In practice, however, there may be a lag between the time virus particles contact target cells and the time the contacted cells become actively affected, implying that the contacting virions enter cells.

This is explained by the virus's initial (or eclipse) phase, which includes all stages from viral attachment until the host cell contains infectious viral particles in its cytoplasm. When compared to models without delays, models of HIV-1 infection with intracellular delays are more accurate representations of the biology and change the estimated values of kinetic parameters, according to research. As a result, we should include time delays in the model foundation to make it more realistic. Many authors have studied HIV-infection models with time delays in the last decade, and time delays of one kind or another have been incorporated into biological models by many authors. Here, we include both intracellular and immune delays. Some models include an intracellular delay, and some authors believe that time delays cannot be ignored in immune response models.

To find out whether HIV-1-specific T-cell immunity elicited by this vaccine may offer prophylaxis from HIV-1 infection or at least would reduce plasma viral loads after infection, Merck and the National Institutes of Health launched the Trials Network (HVTN) 502, popularly known as the "STEP" trial. The HIV-1 clade B Gag, Pol, and Nef-expressing rAd5 vectors used to create the vaccine candidate were mixed in a trivalent fashion.

After challenging rhesus monkeys with the chimeric Simian-Human Immunodeficiency Virus (SHIV-) 89.6P, preclinical and phase I trial results demonstrated this vaccine's strong immunogenicity and ability to lower viral loads. However, the STEP trial was abruptly ended on September 18, 2007. Both infection and early plasma virus levels in those who received the vaccine were not able to be reduced.

CONCLUSION

Since its discovery in 1983, HIV/AIDS has posed humans with unprecedented scientific, medical, and ethical difficulties. Even though there have been numerous failures and a barrier in the way of an HIV vaccine, there has been substantial advancement in recent years. We believe we will find the solution and ultimately defeat HIV/AIDS and put an end to the pandemic by analyzing what had happened in the past and determining what is the key issue before us.

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