

Functional Vaccines and Therapy Aimed at Limiting HIV Disease

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DESCRIPTION

There are two forms of Human Immunodeficiency Virus (HIV) viruses: HIV-1, which is pandemic and aggressive, and HIV-2, which is mostly found in West Africa and is less harmful. Despite the fact that Acquired immunodeficiency syndrome (AIDS) was discovered about 40 years ago, there is currently no treatment or vaccine against it. As a result, functional vaccinations and therapies that attempt to restrict HIV disease progression and transmission through permanent viral replication control without life-long therapy have been proposed as more practical approaches for controlling the HIV pandemic. To uncover virus-host processes that may be targeted for functional cure development, researchers have concentrated on a tiny subset of HIV-1 infected people who regulate their infection on their own, known as elite controllers. Nevertheless, these attempts have not been successful in elucidating the key processes of infection control. With HIV-2 infection, the proportion of long-term viral control is higher than in HIV-1 infection. As a result, adopted HIV-2 as a paradigm for developing a functioning HIV cure. Knowing the main distinctions between HIV-1 and HIV-2 infections, as well as the cross-reactive effects in HIV-1/HIV-2 dual infection, might lead to innovative insights in the development of functional HIV treatments and vaccines.

Despite the fact that HIV-2 has been detected in various countries of Africa, Europe, India, and the United States, West Africa has continuously had the highest prevalence of HIV-2. The initial HIV-2 research revealed a lower incidence of disease progression compared to HIV-1 among female sex workers in Senegal in 1994. In Guinea-Bissau, HIV-2 infected people had double the death rate as HIV negative people, according to a 1997 study. Further investigations in Guinea-Bissau found that death rates in HIV-2 infected people were two to five times higher than in HIV-negative people. Additional research from The Gambia and France examined HIV-1 with HIV-2 infection and found that HIV-2 infected people had a slower drop in CD4 + T-cells. As a result, HIV-2 infected people have longer asymptomatic phases than HIV-1 infected people. Yet, when it comes to AIDS, HIV-1 and HIV-2 have a similar clinical spectrum, with the exception of HIV-2 infected people having a reduced prevalence of Kaposi's sarcoma. Interestingly, studies have found that HIV-1 and HIV-2 infected people with identical

baseline viral loads and CD4+ T-cell counts had similar prognoses. This might imply that illness prognosis is decided early on in both forms of HIV infections.

The viral set-point in HIV-2 is thought to be 10-28 times lower, with lower levels of viraemia lasting into clinical stages of illness. Moreover, AIDS seems to occur at a lower viral load level in HIV-2 infection compared to HIV-1 infection, despite the fact that the CD4 count is generally greater in HIV-2-infected people when AIDS-defining symptoms manifest. The concurrent rise in HIV-1 and fall in HIV-2 transmission rates in West Africa between 1990 and 2010 emphasizes the lower transmission rates of HIV-2. According to several studies, only about 15-25% of HIV-2 infected people will get AIDS if the illness runs its natural course. Nevertheless, these assumptions were based on data from HIV-2 infected people without information on infection date. On the one hand, a lack of infection date will invariably select for those who advance at a slower pace than the norm. When measuring genuine illness progression rates, these biases will generate a paradox that will be difficult to correct effectively.

CONCLUSION

Data from people with an estimated date of infection in 2018 revealed that the illness trajectory was almost equal between HIV-1 and HIV-2 infections, but at around half the incidence among HIV-2 infected individuals. Significantly, study demonstrated that without antiretroviral therapy (ART), the majority of HIV-2 infected people will get AIDS (ART).

Nonetheless, while no such indication was found in the study, the existence of a subset of HIV-2-infected subjects who maintain long-term viral control and have a normal life expectancy cannot be completely ruled out because this would necessitate a complete follow-up of all study participants to the terminal (AIDS or death). Nevertheless, the duration to AIDS in such a subgroup would be longer than the projected human lifetime, implying that the age of HIV-2 infection would be a determining factor for the group size. In reality, the HIV-2 infected group's median age upon infection was 38 years. This, together with the paucity of information on the timing of infection, might explain prior findings of a large proportion of HIV-2 infected people not acquiring HIV-related illness.

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