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VIRAL RESERVOIRS AND ONGOING VIRUS REPLICATION IN PATIENTS ON HAART: IMPLICATIONS FOR CLINICAL MANAGEMENT

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Recent evidence suggests that virus production continues at low levels in HIV-1-infected patients who have responded well to highly active antiretroviral therapy (HAART). The source and clinical significance of this low-level viremia are unclear. It is critical to determine whether low-level viremia results from or leads to the initial evolution of drug resistance. One likely source of this low-level viremia is that latent reservoir for HIV-1. It has been well established that HIV-1 can persist in a latent form in resting CD4+ T cells for very long periods of time. This talk will review recent developments in the study of viral reservoirs that allow persistence of HIV-1 in patients on HAART, focusing on the mechanism by which the latent reservoir in resting CD4+ T cells is established and the mechanisms that are responsible for the extraordinary stability of this reservoir. The relationship between ongoing virus production and the latent reservoir for HIV-1 will be considered. Using a recently developed method for genotypic analysis on patients with <50 copies/ml of plasma HIV-1 RNA, we have shown that there is continued release, for as long as 45 months, of archival drug-sensitive viruses devoid of new resistance mutations selected under HAART. Resistance mutations that were observed reflected prior therapy with non-suppressive one or two drug regimens. Thus, low-level viremia in patients on HAART with <50 copies/ml of HIV-1 RNA results primarily from release of archival, pre-HAART viruses rather than new, HAART-selected, partially resistant mutants. The results indicate that long-term suppression on HAART may be possible. However, the continued release of archival, drug-resistant viruses selected by prior non-suppressive regimens may limit the effectiveness of the "recycling" of antiretroviral drugs.

Keywords: AEGIS, Antiretroviral Therapy, Highly Active, Virus Replication, HIV-1, Viremia, HIV-1

Reverse Transcriptase, Antigens, CD4, Human, virology, AIDS

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