Speed of HIV Replication Affects Disease Progression

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The replication speed of the first strain of HIV an individual acquires has a strong correlation to the rate of immune system decline. Publishing their findings in the Proceedings of the National Academy of Sciences, researchers in the Zambia-Emory HIV Research Project drew samples of the virus from 127 untreated people in Zambia who had recently become HIV positive, an average of 46 days before.

The investigators determined each viral strain's ability to reproduce in a laboratory setting. They found that a greater replication speed was tied to faster decline in CD4 cells in the study participants. Those infected with HIV that replicated more slowly developed a low CD4 count more than two years after those with fast-replicating viruses. In addition, the study found that the relative effects of slow- versus fast-replicating viruses were set in motion quite soon after infection, and that these effects were independent of people's initial viral loads and whether they had certain variations in immune genes known as HLAs that have been shown to yield a greater immune response to the virus.

Those who had fast-replicating viruses had more signs of acute inflammation during the first few months of infection. What's more, their CD4 cells showed more signs of "exhaustion," a scenario that can also lead to faster progression of HIV disease.

"The effect of viral replicative capacity is just as big as, and independent from, the effect of well-studied protective HLA alleles, which influence whether someone is likely to become an 'elite controller,'" Daniel T. Claiborne, PhD, lead author of the study and a postdoctoral fellow at Emory University, said in a press release. "This suggests lowering viral replicative capacity and the resulting immune activation might have benefits, in terms of morbidity and mortality, for individuals whether they remain treatment naive or go on antiretroviral therapy.

"This may have important implications for cure strategies aimed at eliminating the viral reservoir, as individuals infected with low replicative capacity viruses may have smaller latent viral pools that may be easier to eradicate," he continued. "The implication is that if a vaccine enters the fight against HIV, its contribution doesn't have to be a knockout blow. A vaccine that targets qualities of the virus responsible for replicative capacity could have an important impact on disease progression and secondary transmission."

To read the press release, click here.

To read the study abstract, click here.
