Acyclovir Slows HIV Disease Progression, But Fails to Reduce HIV Transmission

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Acyclovir doesn't prevent HIV-positive people from transmitting the virus to their uninfected partners, but it does appear to slow HIV disease progression, according to a study presented at the Fifth International AIDS Society (IAS) Conference on HIV Pathogenesis, Treatment and Prevention in Cape Town.

Acyclovir is an antiviral drug active against herpes simplex viruses (HSV), both HSV-1—the cause of oral herpes—and HSV-2—the cause of genital herpes. It is a commonly prescribed treatment for both viruses to help shorten and prevent outbreaks of sores.

In terms of HIV prevention, it has been hoped that transmission rates might be lowered by controlling the formation of herpes sores, which can increase the chance of both transmission and acquisition of HIV. An earlier study suggested that it wasn't effective for this purpose, but further studies were needed to confirm this.

In terms of HIV treatment, several studies have hinted that acyclovir could perhaps lower HIV levels somewhat, thus helping to avoid HIV disease progression. To test these two theories, Jairam Lingappa, MD, PhD, and Connie Celum, MD, MPH, both from the University of Washington at Seattle, conducted two separate studies in Africa.

In the first, an HIV prevention study, 3,408 African couples participated. In each couple, one was HIV positive and the other HIV negative. All of the HIV-positive partners also had HSV-2. None, however, were on antiretroviral (ARV) drugs at the study's initiation. Sixty-seven percent of the HIV-positive partners were female.

Celum's team found that acyclovir significantly reduced HSV-2 outbreaks by 73 percent. It also lowered HIV levels somewhat. It did not, however, reduce the risk of HIV transmission from the HIV-positive to the HIV-negative partner. The authors conclude that larger reductions in HIV would be needed to lower the transmission rate.

In the other study, Lingappa's group recruited 3,381 HIV-positive men and women who were also infected with HSV-2. All of the participants had a CD4 count over 250, and none were taking

antiretroviral therapy at the start of the study. The aim was to prevent three things: CD4 levels from dropping to 200; having to start ARV therapy; and death. These outcomes were considered study endpoints. Roughly half of the participants received 400 mg of acyclovir twice a day, and half received a placebo.

Lingappa and his colleagues found 284 of those receiving acyclovir reached one or more of the study endpoints, compared with 325 in the placebo arm. The difference held true no matter which of the three endpoints were used as a comparison, and the overall difference, 17 percent, was too large to have occurred by chance. The researchers, nevertheless, are recommending further studies to determine whether a 17 percent reduction in disease progression is large enough to warrant treating all people coinfected with both HIV and HSV-2 with acyclovir.
