Oral HIV Drug Tenofovir Does Not Affect Herpes Virus

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People taking the antiretroviral (ARV) drug tenofovir (found in Viread, Truvada and Atripla) as part of their HIV treatment regimen had no better control of their herpes virus activity than people not on a tenofovir-containing regimen. These data, published in the January 1 issue of *AIDS*, run counter to a recent trial, which found that a tenofovir vaginal gel could cut acquisition of herpes by roughly 50 percent.

Tenofovir has always been developed as a drug to treat HIV. No one, however, expected that tenofovir could also fight the herpes simplex virus type 2 (HSV-2), the virus that causes genital herpes. Thus, it was a surprise to many researchers when a recent study comparing a vaginal gel version of tenofovir was found to protect HIV-negative women from both HIV and HSV-2. This led researchers to reconsider whether oral tenofovir might also protect against herpes infection or control herpes in people already living with the virus.

In one of the first trials designed to specifically explore this matter, Darrell Tan, MD, and his colleagues from the University of Toronto studied the activity of herpes viruses in 40 HIV-positive people who were also infected with either HSV-2 or herpes simplex virus type 1 (HSV-1), the strain responsible for most cases of oral herpes (cold sores, for example). Tan's team looked at herpes activity by asking the participants to swab their mouths, genitals and anuses every day for 28 days.

All of the study participants were taking ARVs, and all had undetectable HIV levels. To be included in the study, all the participants had to have a documented history of herpes infection but could not have had an outbreak any time during the four months before entering the study. In all, 32 had HSV-1, 30 had HSV-2, and 22 had both viruses. Twenty-two were taking a regimen containing tenofovir, and 18 were not.

Tan and his colleagues found that tenofovir had no effect on herpes shedding—a sign that the virus is actively reproducing. Overall, only 7 percent of all samples had detectable HSV-2 shedding, and there was no difference in the frequency of shedding between those on tenofovir and those not on tenofovir.

The authors caution against drawing definitive conclusions from their study: It was small, and since shedding can occur for as little as one hour per day, it is possible that they did not fully

capture HSV shedding in both groups.

Tan’s team concludes that because there would be clinical benefit if tenofovir therapy could fight both herpes and HIV, and because their study size was so small, further trials are warranted.

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