Herpes drug inhibits HIV replication, but with a price

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The anti-herpes drug acyclovir can also directly slow down HIV infection by targeting the reverse transcriptase (RT) enzyme, researchers report in this week's *JBC*. This beneficial effect does pose a risk though, as HIV-infected cells treated with acyclovir promote the emergence of multi-drug resistant HIV variants.

HIV and herpes (HSV) are two of the most common sexually transmitted diseases worldwide, and individuals frequently become co-infected with both. In such cases, the two viruses interact with each other; the presence of HIV often results in more frequent HSV lesion outbreaks, while HSV can speed up the progression of HIV to AIDS.

Considering their interaction, recent studies showing that acyclovir treatment could reduce HIV viral load in co-infected patients were not surprising, and attributed to an indirect effect of HSV suppression. However, Moira McMahon and colleagues at Johns Hopkins decided to look whether the effects on HIV might be direct.

They used a sensitive infection assay of white blood cells and found that acyclovir can directly inhibit HIV replication. The drug specifically targeted RT, the key HIV enzyme that converts the virus' RNA into DNA so it can be replicated. However, acyclovir treatment had some unexpected results; as early as five days after initial infection, a mutant version of HIV (V75I) appeared in the cells, and within 94 days spread to comprise over 90% of the viral population. The V75I strain is part of the resistance pathway to many drugs, including the commonly used RT

inhibitors.

What this means, the authors note, is that acyclovir could be a great model for designing future HIV treatments, but also could be a risky drug if given to HSV patients co-infected with HIV by potentially promoting cross-resistance to current treatments.

Source: American Society for Biochemistry and Molecular Biology

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