

# Powerful HIV drugs inhibit retrovirus linked to prostate cancer, chronic fatigue syndrome

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Certain drugs used to combat HIV also inhibit a retrovirus recently linked to prostate cancer and chronic fatigue syndrome (CFS), a new study from University of Utah and Emory University/Veterans Affairs Medical Center (VA) researchers shows.

The finding means that if the retrovirus XMRV (Xenotropic murine leukemia virus-related virus) is proved to cause [prostate cancer](#) or CFS, it's possible those two illnesses might one day be treatable with drugs aimed at HIV, the researchers write in a study published April 1, 2010, in [PLoS One](#), an open-access journal published by the Public Library of Science (PLoS).

"These results offer hope to infected persons, but we are still at the early stages of our understanding of the potential link between XMRV and these diseases," said Ila R. Singh, M.D., Ph.D., associate professor of pathology at the University of Utah School of Medicine. "Not all studies that have looked for XMRV have been able to detect it in prostate cancers or in samples from [chronic fatigue syndrome](#). Even if XMRV is established to be the cause of prostate cancer or chronic fatigue syndrome, we will need to see the results of clinical trials before these drugs can be used in a clinical setting. We, along with other investigators, are working as hard as we possibly can to get to that point, but it is important to caution patients that we are not there yet."

Singh, and Raymond F. Schinazi, Ph.D., D.Sc., professor of pediatrics and chemistry and an investigator with the Center for AIDS Research at

the Emory University School of Medicine and the Atlanta VA, and colleagues tested 45 compounds used to treat HIV and other viral infections to see how effectively they worked against XMRV in cultured human [breast cancer](#) and prostate cancer cells. The most potent drug at inhibiting XMRV was raltegravir, made and marketed to treat HIV by Merck & Co., which inhibited XMRV replication in cultured cells at concentrations known to inhibit HIV in humans. Three other drugs, L-00870812, Zidovudine (ZDV or AZT), and tenofovir disoproxil fumarate (TDF), also effectively prevented virus replication. This study was not supported by Merck or any other pharmaceutical company that markets antiviral drugs.

"Our study showed that these drugs inhibited XMRV at lower concentrations when two of them were used together, suggesting that possible highly potent 'cocktail' therapies might inhibit the virus from replicating and spreading," said Schinazi. "This combination of therapies might also have the added benefit of delaying or even preventing the virus from mutating into forms that are drug-resistant." Singh and Schinazi are currently investigating the development of viral resistance to raltegravir and other active drugs.

XMRV, a [retrovirus](#) discovered in 2006 by researchers at the University of California, San Francisco, and at the Cleveland Clinic, is one of three retroviruses known to infect people. Other retroviruses that are closely related to XMRV are known to cause leukemia and sarcomas in animals. Last fall, Singh led a study that demonstrated the presence of XMRV in malignant human prostate cancer cells. That study, published in the Proceedings of the National Academy of Sciences, found XMRV in 27 percent of prostate cancers examined, with the virus more likely to be present in the most-aggressive tumors.

More recently, scientists at the Whittemore Peterson Institute in Reno, Nev., identified XMRV in blood cells of patients with Chronic Fatigue

Syndrome.

Provided by University of Utah

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