

# Norvir-Boosted Protease Regimen Bests Other Regimens

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People taking an antiretroviral (ARV) treatment regimen that includes a [protease inhibitor](#) (PI) boosted with low-dose [Norvir](#) (ritonavir) were less likely to develop drug resistance than people taking other types of regimens, according to the authors of a [study published](#) in the July 1 issue of the *Journal of Infectious Diseases*.

Current federal HIV treatment guidelines recommend regimens containing either a Norvir-boosted PI or a [non-nucleoside reverse transcriptase inhibitor](#) (NNRTI) for people starting antiretroviral (ARV) treatment for the first time. If people can't tolerate a boosted PI or an NNRTI, they may use a PI that isn't boosted with low-dose Norvir.

To determine which regimens are likely to have the most prolonged benefit—and the least likely to be limited by the development of drug-resistance mutations in HIV—Viviane Lima, PhD, from the British Columbia Centre for Excellence in HIV/AIDS in Vancouver, and her colleagues compared the medical records of a cohort of 2,350 people living with HIV taking NNRTI-, boosted-PI-, or unboosted-PI-based regimens between August 1996 and November 2004.

Un-boosted PIs were most likely to be used in the late 1990s, with people often taking [Crixivan](#) (indinavir) or [Viracept](#) (nelfinavir). NNRTI use gained popularity around 1999, with many patients initially using [Viramune](#) (nevirapine). Boosted PI regimens gained favor beginning in 2002 and most commonly included [Kaletra](#) (lopinavir/ritonavir), followed by [Reyataz](#) (atazanavir).

Lima's team found that the development of resistance occurred at the same rate in people who started treatment with an un-boosted PI as in those who started treatment with an NNRTI. However, people who started treatment with a boosted PI were nearly two and a half times less likely to develop drug resistance than people on an NNRTI or an un-boosted PI.

The year treatment was started also made a difference. Those starting treatment after 2002—regardless of the type of treatment regimen—were about 60 percent less likely to develop resistance than people who started treatment before 2002.