Does Switching to Norvir-Boosted Reyataz Spare the Heart?

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New research suggests that the protease inhibitor (PI) Reyataz (atazanavir), when boosted by low-dose Norvir (ritonavir), might not improve cardiovascular disease (CVD) risk factors as much as some had hoped. A small study published online December 29 in *AIDS* finds that people with elevated lipids—cholesterol and triglycerides—who switch to Norvir-boosted Reyataz from another PI combination do see improvements in their lipids, but not several other markers of CVD risk.

CVD has become an increasing concern for people living with HIV, particularly as more HIV-positive people are living into their 50s and 60s. A number of recent studies have demonstrated that people with HIV appear to be at higher risk for CVD—which can cause heart attacks and strokes—than their HIV-negative counterparts. Studies have shown that PIs and HIV itself can increase CVD risk, in addition to traditional CVD risk factors such as smoking. Researchers hoped that Reyataz, given that it tends to have a more neutral effect on lipids than other protease inhibitors, might help mitigate the increased risk found in some studies in HIV-positive people.

To explore this possibility, Robert Murphy, MD, from Northwestern University in Chicago, and his colleagues randomized 50 people to either remain on their current PI regiment or switch to Norvir-boosted Reyataz. All of the participants had HIV fully suppressed for at least 12 weeks and all had elevated lipids. Twenty-four remained on their current PI, and 26 made the switch.

People with uncontrolled diabetes or high blood pressure and those who smoked more than one pack of cigarettes per day were excluded from the study. The average age of the participants was 43, and roughly 85 percent were male. About 60 percent of the study volunteers were white, and just less than half were smokers. Most had been on Kaletra (lopinavir plus ritonavir).

Twenty-four weeks into the study, people who switched to Norvir-boosted Reyataz saw significant reductions in total cholesterol and triglycerides compared with those who remained on their old PI regimen. But when Murphy's team measured blood vessel function, called endothelial function, and looked at levels of inflammatory proteins—both markers of CVD risk—there was no difference between the two groups.

The authors acknowledge the small size of their study, and that there might have been changes in endothelial function and inflammatory markers after switching to the Reyataz-containing regimen if the participants, on average, had started the study with greater lipid elevations.

“In this setting, the impact of switching to [Reyataz] on CVD risk is limited to the favorable changes observed in the lipid profile,” Murphy and his colleagues conclude.
