

Crestor Bests Pravachol for Elevated Lipids

October 27, 2007 By [Tim Horn](#) and David Evans

Crestor (rosuvastatin) is superior to Pravachol (pravastatin) in HIV-positive people on antiretroviral therapy who experience LDL (“bad”) cholesterol and triglyceride elevations, according to new data presented this week at the 11th European AIDS Conference (EACS) in Madrid.

Approximately 50 percent of people starting a [protease inhibitor](#)-based combination will develop [hyperlipidemia](#)—increased cholesterol and triglycerides—within ten months, according to Elisabeth Aslangul, MD, of the Hopital Hôtel Dieu Paris and her fellow study presenters. In clinical trials involving HIV-negative volunteers, Crestor has been shown to be superior to Pravachol—branded as Vasten in the European Union—in terms of lowering LDL cholesterol levels.

Among HIV-positive patients with hyperlipidemia, Crestor and Pravachol have long been the favored lipid-lowering “statins,” as both have minimal interactions with protease inhibitors and other HIV/AIDS drugs. Yet little is known about how the two drugs compare in terms of reducing lipid levels in people living with HIV.

To answer this question, Dr. Aslangul and her colleagues conducted a 45-day clinical trial comparing 10 mg Crestor to 40 mg Pravachol in 83 HIV-positive patients on a protease inhibitor-based regimen with elevated LDL cholesterol and triglyceride levels. Forty-one patients were randomized to receive Crestor and 42 patients were randomized to receive Pravachol. Patients in the study had been receiving antiretroviral therapy for approximately nine years.

At study entry, the average total cholesterol was 292 mg/dL (7.48 mmol/L). LDL cholesterol averaged 192 mg/dL (4.93 mmol/L) and “good” HDL cholesterol averaged 50 mg/dL (1.27 mmol/L). Average triglycerides at baseline were 204 mg/dL (2.29 mmol/L).

After 45 days of statin treatment, LDL was reduced by 37 percent in the Crestor group, compared with 19 percent in the Pravachol group. And compared with a 19 percent drop in triglycerides among those in the Crestor group, a smaller 7 percent reduction was noted in the Pravachol group. These differences were statistically significant, meaning that they weren’t due to chance.

Reducing LDL to below 160 mg/dL (less than 4.1 mmol) was a primary goal of the study, as levels higher than this are associated with an increased risk of cardiovascular disease. This primary endpoint was met by 88 percent of those receiving Crestor, compared with 60 percent of those

receiving Pravachol.

No significant HDL changes, or differences between the two groups, were reported.

During the question-and-answer period following Dr. Aslangul's presentation, it was noted that Norvir (ritonavir)—frequently used to boost the efficacy of other protease inhibitors—can slightly increase Crestor levels, and slightly decrease Pravachol levels, in the blood. This, in turn, may have affected the study results. Dr. Aslangul countered that such effects of Norvir are “relatively small” and that the differences between the two groups were unlikely to be due solely to these interactions.

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