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High-sensitivity C-reactive protein levels do not decrease with the use of statins in all persons with HIV infection

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We read with interest the study by Aslangul *et al.* [1] on the effects of pravastatin and rosuvastatin on inflammatory biomarkers. It is interesting that they found a lowering of high-sensitivity C-reactive protein (hs-CRP) with short-term use of pravastatin and rosuvastatin. It is not clear from the data whether the hs-CRP was significantly lowered in each group at day 45 or only when the groups were combined [−20%, interquartile range (IQR) −44 to 17, $P=0.001$]. It would be helpful to know since the IQR bounds positive values in both the pravastatin and rosuvastatin groups, suggesting that the data may have been skewed with some extreme values in some patients. The authors also asserted that the fall in short-term hs-CRP could be accentuated with longer-term use as was observed in the JUPITER study (37% reduction after 1.9 years of treatment, on average). We have examined this question in a previously published study of 74 individuals who were part of ACTG 5087, a randomized trial of pravastatin, fenofibrate or their combination to treat combined hyperlipidemia in persons with HIV infection [2]. Our participants were also receiving a variety of antiretroviral regimens and most (78%) had HIV viral loads less than 50 copies/ml. In contrast the findings of Aslangul *et al.* [1], hs-CRP levels did not significantly change from baseline to weeks 12 or 48 despite similar changes in both studies in total cholesterol and LDL-C. In 37 patients receiving pravastatin 40 mg daily, the baseline median hs-CRP was 3.5 mg/l (IQR 2.1–6.0) and, at week 12, the median value was 3.35 mg/l (IQR 1.95–5.95). The median change was −0.2 mg/l (IQR −1.0 to 1.1, $P=0.76$). Furthermore, in 58 patients treated with pravastatin and micronized fenofibrate 160 mg daily, there was no significant change in hs-CRP from baseline to week 48. The median hs-CRP at baseline was 2.7 mg/l (IQR 1.5–4.5) and, at week 48, was 3.2 mg/l (IQR 1.25–7.10). The median change from baseline to week 48 was −0.05 (IQR −0.80 to 2.70, $P=0.56$). We also found no significant changes, short or long-term, in plasminogen-activator inhibitor 1 or P-selectin. It is possible that the timing of these evaluations may explain differences in our observations (day 45 versus day 84 and day 336). Alternatively, participants the study by Aslangul *et al.* [1] were enrolled between 2005 and 2007, whereas our participants were enrolled between 2000 and 2001. There may have been differences in the types of antiretroviral regimens, though 87% of our patients were on a protease inhibitor-based regimen. Thus, we would be cautious in concluding that pravastatin or rosuvastatin will have a longer-term effect in

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Conflicts of interest

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persons with HIV infection similar to that observed in seronegative persons in the JUPITER study. We believe that further long-term studies of statins using a variety of inflammatory biomarkers are warranted in persons with HIV infection.

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