Atherosclerosis and Nitric Oxide Production

To the Editor:

We read with interest 2 recent reports published in Circulation. We believe that the work of Qian et al1 and Ridker et al2 has an important connection in light of our recent findings.3 Qian and coworkers1 showed that in cholesterol-fed rabbits, increased nitric oxide production, via nitric oxide synthase gene transfer, produced rapid amelioration of several markers of atherosclerosis. Ridker et al2 reported that pravastatin reduced levels of C-reactive protein, a sensitive marker identifying those at risk of future atherosclerotic cardiovascular events.4,5 Furthermore, this effect was not related to the magnitude of lipid alterations.

Our study3 demonstrated that pravastatin activates endothelial nitric oxide synthase independently of its effects on lipids. In view of our data and those presented by Qian et al,1 we believe that increased nitric oxide production caused by pravastatin is part of the nonlipid mechanism of benefit in reducing C-reactive protein levels seen in the study of Ridker et al.2 Furthermore, we have shown that L-arginine potentiates nitric oxide production by pravastatin.3 As a result, we believe the beneficial effects of pravastatin on C-reactive protein seen by Ridker and others2 may be enhanced by the administration of pravastatin in combination with L-arginine.

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