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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