Correspondence

Letter by Vítek et al Regarding Article, “Niacin Inhibits Vascular Inflammation via the Induction of Heme Oxygenase-1”

To the Editor:

We read with interest a recent article by Dr Wu and colleagues on the anti-inflammatory effects of niacin, mediated by induction of heme oxygenase-1. The authors performed detailed studies on a rabbit model of vascular inflammation, clearly demonstrating that niacin inhibits inflammatory processes in the vascular wall mainly via induction of heme oxygenase-1, an important cytoprotective protein responsible for bilirubin production in the human body. In fact, the authors demonstrated substantial elevation of serum bilirubin levels in response to niacin treatment. In our opinion, 2 aspects of the study deserve additional explanation. First, the concentrations of bilirubin in the rabbit sera were extremely low, almost 3 orders of magnitude lower compared with data by others and below the sensitivity of the majority of analytical methods, including that used by the authors. Second, bilirubin is not the major bile pigment in rabbits. Because rabbits exhibit only <0.4% of the rat hepatic biliverdin reductase activity, the major biliary bile pigment is biliverdin. Moreover, bilirubin infusion to rabbits leads to increased biliary secretion of biliverdin, indicating that bilirubin in rabbits is rapidly oxidized back to biliverdin. Thus, it is our opinion that both pigments in lapine sera, ie, biliverdin plus bilirubin, should be considered when assessing the effects of heme oxygenase-1 induction in rabbits.

Disclosures

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