[The authors respond:]

As Pankaj Madan points out, adiponectin is an insulin-sensitizing hormone secreted by adipose tissue; its levels are lower among people with central obesity, which is a common characteristic of patients with NAFLD. Our review[1] incorrectly stated that leptin and adiponectin promote liver fibrogenesis in animal models, where it should have stated that leptin and adiponectin affect fibrogenesis in animal models.

Adiponectin may protect against NAFLD by multiple mechanisms. In animal models of NAFLD, adiponectin supplementation improves hepatic steatosis by increasing hepatic free fatty acid oxidation.[2] Correspondingly, in human cross-sectional studies, serum adiponectin is inversely correlated with degree of hepatic steatosis.[3,4] Furthermore, experimental liver injury and fibrosis, which are enhanced in adiponectin knock-out mice, are ameliorated by adiponectin supplementation.[5–7] Consistent with this observation is the finding that the hepatic expression of adiponectin is lower among patients with nonalcoholic steatohepatitis than among those with steatosis.[8] However, serum adiponectin levels do not appear to correlate with fibrosis, nor are they consistently associated with degree of necroinflammation.[3,4] Thus although an increasing body of evidence from in-vitro and animal studies supports the "hepatoprotective" effect of adiponectin, the exact role in the pathogenesis of human NAFLD requires further investigation and is eagerly awaited.

Diana Mager and Eve Roberts correctly point out that NAFLD is being increasingly recognized among children. As stated in our article, the prevalence of this condition among school children (4–12 years of age), as detected by ultrasonography, is 2.6%,[9] increasing to 22.5% among obese children. Unfortunately, space limitations prevented us from discussing this important issue further in the original review. The significance of this common liver condition among children remains to be determined, and thus the need for specific treatment aimed at preventing progression to cirrhosis is unknown, particularly as we are unable to accurately identify those who will experience liver-related complications. Intuitively, we might expect a greater risk of advanced hepatic fibrosis with a longer duration of "hepatic fat exposure" from childhood, which would place these subjects at risk for liver disease later in life. Clearly, further research on this important topic is needed. It is important, however, to recognize that the metabolic conditions frequently accompanying pediatric (and adult) NAFLD place these patients at risk of complications such as cardiovascular disease (and perhaps cirrhosis), and intervention is therefore essential.

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Corrections

An error in the recent obituary of Dr. William Tatlow[1] was pointed out to us, as follows, by Dr. Joseph Stratford, a long-time friend and colleague of the late Dr. Tatlow. "[A]ll is fine until you come to the point where it says 'out for his morning *job* when he fell.' [I]t is true that Bill had continued working, reading EEGs, and he was a consultant for certain pharmaceutical companies, so in a sense he did have a 'job,' but that morning, out at Hudson, he was out for his morning *jog* when he fell." We apologize for our mistake.

Reference

1. Deaths. *CMAJ* 2005;173(3):323.

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The following information was mistakenly omitted from a recent article.[1] Steven Grover has conducted research supported by Pfizer, Bristol-Myers Squibb, Aventis and AstraZeneca. He has received consulting fees from Pfizer and AstraZeneca and speaker fees from Pfizer, Oryx and Aventis. He owns stock in Pfizer, Merck, Bristol-Myers Squibb and Kos.

Reference

1. Hemmelgarn BR, Grover S, Feldman RD, for the Canadian Hypertension Education Program. Applying the 2005 Canadian Hypertension Education Program recommendations: 2. Assessing and reducing global atherosclerotic risk among hypertensive patients. *CMAJ* 2005;173(6):593-5.

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