Fructose Likely Does Have a Role in Hypertension

To the Editor:

Ha et al recently performed a meta-analysis and concluded that fructose intake is unlikely to have a role in hypertension. This conclusion is unjustified. Less than half of the studies were randomly assigned, and blood pressure (BP) was the primary outcome in only 3 of 15 studies. BP measurements were not postprandial, at which time the acute effects of fructose on BP are best shown. Ambulatory BP, which would detect these changes, was not used. Most studies examined BP after an overnight fast long after fructose was metabolized. None of the studies controlled for fructose absorption, which can also vary remarkably in individuals. The analysis excluded trials in which fructose was administered as sucrose or high-fructose corn syrup, both the largest contributors of fructose intake. Studies including fructose from natural fruits, such as our study, are not equivalent with studies using fructose from added sugars, because fruits have substances (vitamin C and flavonols) that counter fructose effects. It would be similar to making conclusions about the effect of salt on BP by including studies in which a high-salt diet was administered with a diuretic. In our study, both the low-fructose diet group and the low-fructose diet plus natural fruit supplements showed dramatic BP decreases beyond that expected for weight loss.

In contrast, the physiological evidence for fructose in hypertension is mounting. Fructose intake from added sugars, as well as added sugars themselves, are epidemiologically associated with elevated BP. Fructose raises BP acutely in humans, which is not seen with glucose. The mechanism relates in part to the ability of fructose to raise intracellular and serum uric acid. Over time, fructose also increases de novo uric acid synthesis, which likely accounts for why large doses of fructose eventually raise fasting uric acid levels and fasting BPs. Allopurinol treatment blocks fructose-increased BP in humans. Epidemiological studies also show that reducing sugar intake reduces BP. Finally, the administration of fructose to animals results in changes in the renal microvasculature that are predicted to result in salt-sensitive hypertension and that are likely to potentiate and maintain salt-sensitive hypertension. Further studies are needed, but it is important to recognize that “evidenced-based medicine” that is not based on physiology can lead to serious misinterpretations.

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Disclosures

Dr Johnson discloses he has patent applications related to blocking fructose metabolism or lowering uric acid as a means for blocking features of the metabolic syndrome. He is also author of The Sugar Fix (Rodale, 2008) which makes the argument that excessive fructose intake may have a role in obesity and metabolic syndrome.

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