Metabolic interactions among dietary cholesterol, copper, and fructose

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TO THE EDITOR: Basciano et al. (3) found glucose intolerance, hypercholesterolemia, and hypertriglyceridemia plus numerous other changes related to the development of diabetes mellitus and the metabolic syndrome in hamsters fed diets high in fructose and enriched with cholesterol; they mentioned nutrient-nutrient interactions. Interactions with copper should be considered in explaining their findings, as both cholesterol and fructose disrupt the utilization of dietary copper and because copper deficiency can produce glucose intolerance, hypercholesterolemia, and hypertriglyceridemia.

That cholesterol feeding can induce copper deficiency has been confirmed several times (22–24, 28, 29, 31) since the phenomenon was reported two decades ago (16). Similarly, the disruption of copper utilization by fructose is well established (8–11, 26).

Glucose intolerance from copper deficiency was first found by Keil and Nelson (14) and has been confirmed (4–6, 13, 21, 27). Metabolism of cholesterol and copper were linked in 1973 (17); hypercholesterolemia from deficiency has been confirmed many times (7, 12, 20, 27, 30). Hypertriglyceridemia also occurs (1, 26, 27).

Basciano et al. (3) suggest that their experimental diet resembles the Western diet, which, inter alia, is high in simple sugars and fat and is often low in copper (18, 25). For example, 62% and 36% of diets of 80 randomly selected adults in Baltimore, MD, were below the recommended dietary allowance (0.9 mg/day) and the estimated average requirement for adults (0.7 mg/day), respectively (2).

Copper deficiency is the only nutritional insult that elevates cholesterol, blood pressure, triglycerides, and uric acid, impairs glucose tolerance, promotes oxidative damage, and induces numerous other anatomic, chemical, and physiological changes that characterize the atherosclerotic process (20). Perhaps Basciano et al. (3) will examine possible interactions among copper, cholesterol, and fructose by repeating their experiments with a substantial increase in dietary copper to determine which of their findings depends on adequate copper. This method has been useful in revealing adverse dietary effects previously unrecognized as being from inadequate copper status (15, 19, 22–24).

REFERENCES


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