Metabolic interactions among dietary cholesterol, copper, and fructose

Leslie M. Klevay
Departments of Internal Medicine and of Pharmacology, Physiology, and Therapeutics, University of North Dakota, School of Medicine and Health Sciences, Grand Forks, North Dakota

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).

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 copper. Perhaps Basciano et al. (3) will examine possible interactions among copper, cholesterol, and fructose by repeating their experiments with a substantial increase in copper.

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 copper.

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 copper.

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 copper.

REFERENCES


