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Lipogenic diet-induced alteration of microRNAs in hepatic stress signaling: Implications for hepatic lipoprotein metabolism and insulin resistance

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Emerging evidence has demonstrated the important role of microRNAs in energy metabolism and their participation in excessive caloric intake-induced metabolic syndrome. The consequences of the altered microRNAs, however, have not been well explored. The present study sought to advance our understanding of the potential association between reduced expression of microRNAs induced by high-fructose diet in the initiation of cellular stress signaling and their contribution to the onset of dyslipidemia and hepatic insulin resistance. By applying nutrition manipulated animal models, the high-fructose fed-rat and hamster models, and human apolipoprotein B100 (apoB100) transgenic mice, we were able to demonstrate that high-fructose diet significantly reduced expression of 13 hepatic microRNAs that are associated with genes involved in inflammatory cell differentiation and lipid signaling, including microRNA-15 and microRNA-378/378*. These phenotypes were closely associated with disruption of mitochondrial membrane integrity, induction of oxidative stress and activation of mitochondrial unfolding protein response (UPR). *In vitro*, delivering the microRNAs isolated from the livers of fructose fed-hamsters into McA-7777 cells, a rat hepatoma cell line, induced activation of mitochondrial UPR. Further investigating the lipid and lipoprotein metabolic profiles of the fructose-fed rats, we noticed that the reduced microRNA expression was associated with overproduction of hepatic apoB100, increased secretion of hepatic VLDL-apoB, and the development of hepatic steatosis and insulin resistance. Our finding unveils a novel role for microRNAs in maintaining metabolic homeostasis of intracellular stress-responding machinery (e.g. mitochondria). It also provides evidence for exploring microRNAs as pharmaceutical targets for the prevention and treatment of metabolic syndrome derived from nutrient–surplus.

Biography

Qiaozhu Su received her PhD from McGill University in 2007 and completed her post-doctoral fellowship at the University of Toronto, Canada. She is an assistant professor in the Department of Nutrition and Health Sciences at the University of Nebraska-Lincoln. Qiaozhu has published more than 20 papers in reputed journals and served as an editorial board member for several professional journals. Her research interests include lipid and lipoprotein metabolism, mechanism of metabolic syndrome, with a particular emphasis on the role of microRNAs, ER and mitochondrial stress in obesity, fatty liver disease and insulin resistance.

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