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The role of hepatic rho-kinase in regulating glucose and lipid homeostasis

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Non-alcoholic fatty liver disease (NAFLD) is a common disease associated with obesity and insulin resistance and ultimately leads to cirrhosis and other complications. However, the pathogenesis of this disease has not yet been fully understood. In our studies, mice with liver-specific Rho-kinase 1 (ROCK1) deletion were used to examine role of ROCK1 in regulating hepatic glucose homeostasis and lipid metabolism. Our results show that hepatic ROCK1-deficiency leads to reduced body weight, improved insulin sensitivity and hepatic steatosis in diet-induced obese mice. These changes are associated with a decreased lipogenic rate in the ROCK1-deficient primary hepatocytes compared to control hepatocytes using 14C-lactate as a lipogenic substrate. These data are further supported by the experimental evidence that ROCK1 deletion in the liver causes a significant suppression in expression of key lipogenic enzymes, including FAS, SCD1, Elovl2 and SREBP-1c. In addition, the up-regulation of hepatic ROCK1 expression and activity and hepatic steatosis induced by a high-fat diet are greatly reduced after metformin treatment. Consistent with the *in vivo* data, treating hepatocytes with metformin markedly reduces ROCK1 expression and activity in a dose-dependent manner and decreases expression of lipogenic enzymes and the rate of *de novo* lipogenesis. Collectively, these data identify hepatic ROCK1 as a key regulator of fat metabolism and further demonstrate that inhibition of hepatic ROCK1 could be a novel therapeutic approach for the treatment of NAFLD.

Biography

Hu Huang has completed his PhD from University of Tsukuba, Japan and postdoctoral studies from Beth Israel Deaconess Medical Center and Harvard Medical School. He has published more than 15 papers in reputed journals and serving as an editorial board member of repute. He is assistant professor at East Carolina Diabetes and Obesity Institute & East Carolina University. His laboratory seeks to understand basic molecular mechanisms underlying the metabolic syndrome, including obesity, insulin resistance, impaired glucose metabolism and non-alcoholic fatty liver disease.

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