Hepatic genome-wide expression of lipid metabolism in diet-induced obesity rats treated with cocoa polyphenols

Faisal Ali, Amin Ismail, Norhaizan Mohd Esa, Chong Pei Pei, Sander Kersten

Department of Nutrition and Dietetics, Metabolism and Genomics Group, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 UPM Serdang, Selangor, Malaysia
Laboratory of Molecular Biomedicine, Institute of Bioscience, Universiti Putra Malaysia, Selangor 43400, Malaysia
Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 UPM Serdang, Selangor, Malaysia
Metabolism and Genomics Group, Division of Human Nutrition, Wageningen University, Wageningen, The Netherlands

ARTICLE INFO

Article history:
Received 5 March 2015
Received in revised form 17 June 2015
Accepted 17 June 2015
Available online

ABSTRACT

Cocoa polyphenols (CPs) have been shown to exhibit hypolipidaemic actions, suggesting that CPs offer great potential for ameliorating lipid abnormalities. However, the conceivable molecular mechanisms underlying the pharmacological activity of CPs in obesity-induced liver steatosis have yet to be investigated. This study analysed the hepatic genome-wide expression patterns in high-fat diet (HFD)-induced obese rats using DNA microarray. Rats were fed either a low fat (LFD) or high fat diet (HFD) for 12 weeks. After supplementation, HFD rats were treated with 600 mg/kg bw/day CPs (HFD + CPs) for 4 weeks. As a result, compared to the HFD group, CP treatment significantly lowered lipid in the liver and attenuated the increases in body weight as well as visceral fat accumulation in the CP group. DNA microarray analysis resulted in a differential expression of 862 genes of the 12,282 genes expressed in the liver. The differential expression patterns of selected genes were validated with real-time-PCR. Metabolic pathway analysis via bioinformatic tools showed that genes in lipid catabolism, primarily in fatty acid oxidation, were up-regulated in the CP group, whereas genes in lipid synthesis pathways were down-regulated. Together, these findings provide a novel insight into possible molecular mechanisms behind the pharmacological actions of CPs on the management of the obesity-induced steatosis markers in rats with diet-induced obesity.