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Fatty acid composition in serum correlates with that in the liver and non-alcoholic fatty liver disease activity scores in mice fed a high-fat diet

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ABSTRACT

In this study, we investigated the correlation between the serum fatty acid composition and hepatic steatosis, inflammation, hepatocellular ballooning scores, and liver fatty acids composition in mice fed a high-fat diet. Livers were collected for non-alcoholic fatty liver disease score analysis. Fatty acid compositions were analysed by gas chromatography. Correlations were determined by Pearson correlation coefficient. Exposed to a high-fat diet, mice developed fatty liver disease with varying severity without fibrosis. The serum fatty acid variation became more severe with prolonged exposure to a high-fat diet. This variation also correlated significantly with the variation in livers, with the types of fatty acids corresponding to liver steatosis, inflammation, and hepatocellular ballooning scores. Results of this study lead to the following hypothesis: the extent of serum fatty acid variation may be a preliminary biomarker of fatty liver disease caused by high-fat intake.

1. Introduction

A leading cause of liver dysfunction and non-alcoholic fatty liver disease (NAFLD) worldwide is a clinical metabolic syndrome with fatty accumulation in hepatocytes without alcohol consumption (Angulo, 2002). In recent years, morbidity of NAFLD has grown in both Eastern and Western countries, and diet has become an important health issue (Kojima et al., 2003; Bhala et al., 2013). Nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH) are on the NAFLD spectrum. NASH develops most easily into liver fibrosis, cirrhosis, and even hepatocellular carcinoma (Bugianesi et al., 2002; Jou et al., 2008). Clinically, liver biopsy is the gold standard to assess levels of steatosis, inflammation, and fibrosis, but this method is invasive, NAFLD diagnosis is costly, and the procedures may results in complications (Ratziu et al., 2005). Several kinds of non-invasive biomarkers for evaluating NAFLD have been investigated, but none have performed sufficiently well (Miller et al., 2011). Therefore, discovering a non-invasive method to detect NAFLD is still of great interest.

Fatty acid (FA) accumulation in the liver is considered a characteristic of NAFLD, which results from a FA metabolic disorder not only in the liver but also in the rest of the body (Musso et al., 2009; Fon Tacer and Rozman, 2011). FAs are made available in hepatocytes by importing from the blood and by *de novo* synthesis; meanwhile, they are disposed into the blood by oxidation and triglyceride export through very low-density lipoprotein (VLDL) (Musso et al., 2009). Triglycerides accumulate in the liver when FA import exceeds export (Fabbrini and Magkos, 2014). A recent study suggested that hepatic FA composition is closely associated with the progression of NAFLD (Yamada et al., 2015). FA composition in blood is associated with FA metabolism and correlated with FA composition in the liver. During high-fat diet (HFD) feeding, the FA compositions in the blood and liver are similar (Zhukova et al., 2014). There are few studies focused on

Abbreviations: NAFLD, nonalcoholic fatty liver disease; FA, fatty acid; HFD, high fat diet; CD, Control diet; HE, hematoxylin and eosin; GC, gas chromatography; TFA, total fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids; SFA, saturated fatty acids; UFA, unsaturated fatty acids.

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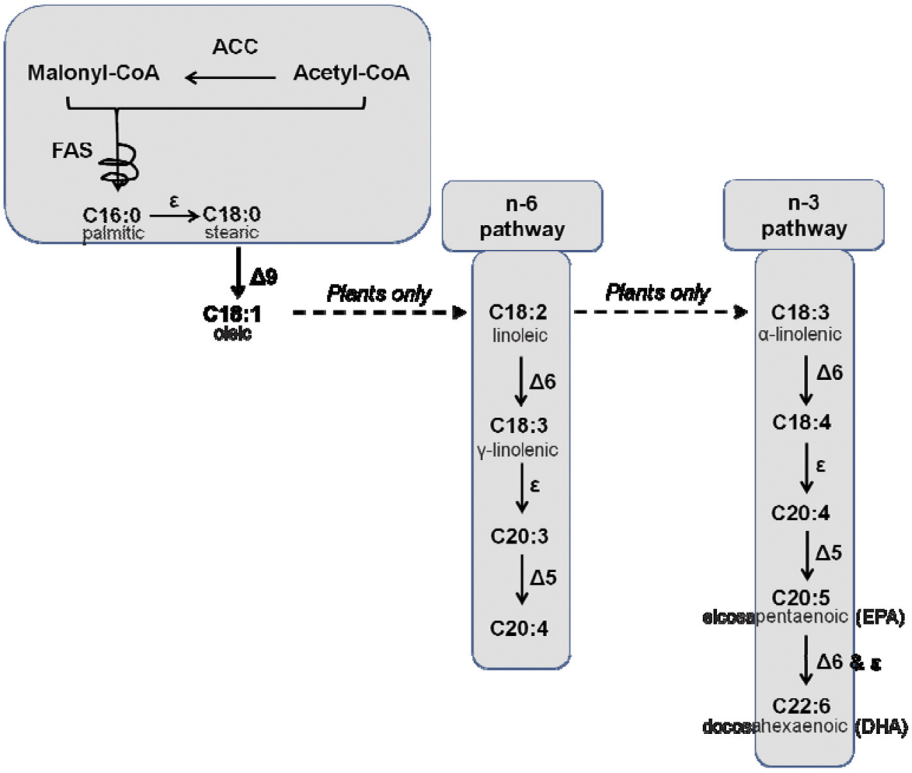

Fig. 1. Fatty acid metabolism in the liver.
This figure shows the biosynthesis and production of different fatty acids in the liver (Guillou et al., 2008). ε represents elongase, which catalyzes the elongation of fatty acids.

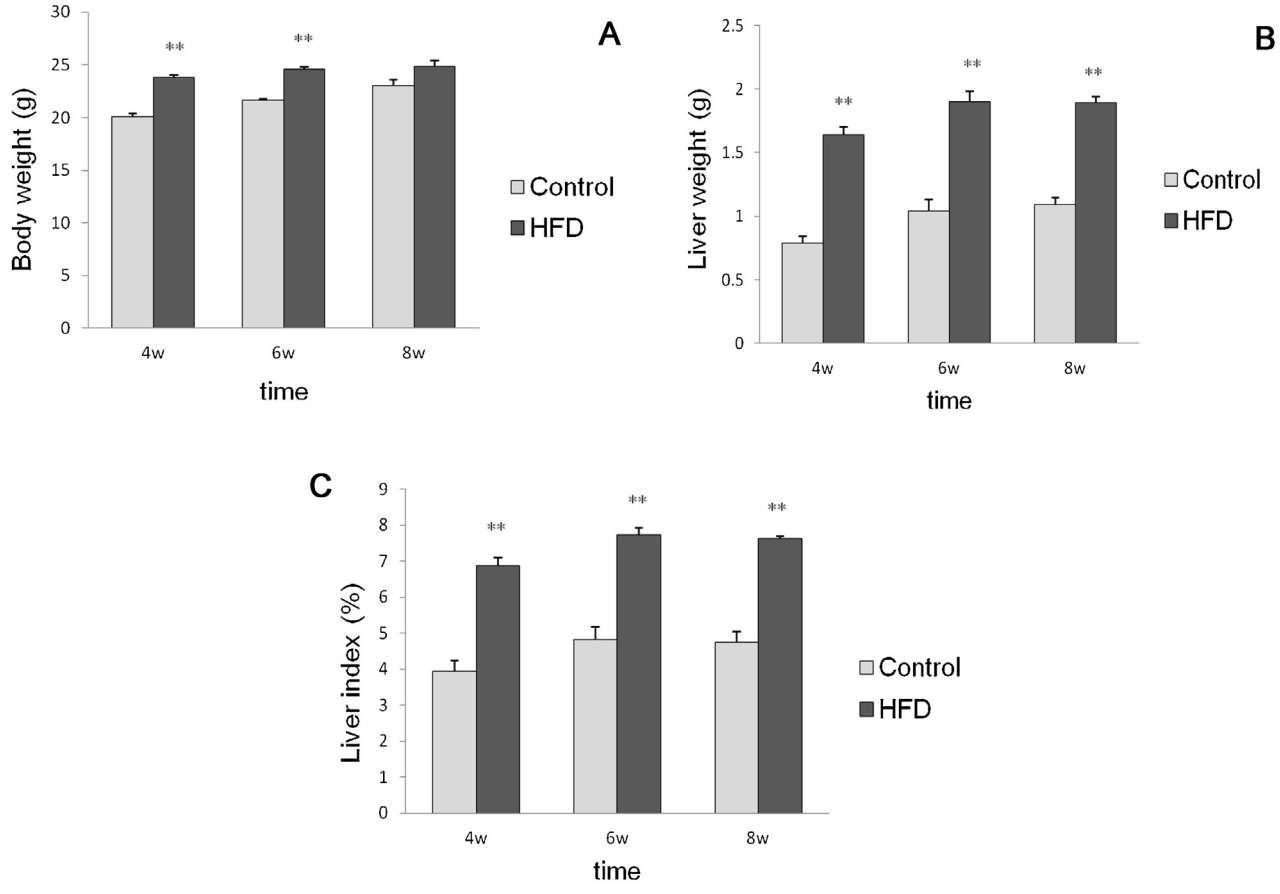

Fig. 2. Body weight, liver weight, and liver index.
(A) Body weights of control and high-fat diet groups. (B) Liver weights of control and high-fat diet groups. (C) The effect of a high-fat diet on the liver index (liver/body weight ratio). Asterisks (*) represent statistical differences between feeding groups; **$P < 0.01$.