Advances in Biomolecular Medicine

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Fatty liver in fasted FABP4/5 null mice is not followed by liver function deterioration

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ABSTRACT: We have recently found that deletion of Fatty Acid-Binding Protein-4 and -5 (FABP4/5) resulted in a marked perturbation of metabolism in response to fasting, including fatty liver. The purpose of our study was to investigate liver function in FABP4/5 null mice (DKO mice) with fatty liver after prolonged fasting. Wild-Type (WT) and DKO mice were fed and fasted 24 hours and 48 hours. Liver was collected and preserved for Hematoxylin and Eosin (HE) and Masson’s Trichrome (MT) staining. To analyze liver function, serum was collected to determine Aspartate Transaminase (AST) and Alanine Transaminase (ALT) levels. After 48 hours of fasting, there was no increase in fibrosis in the liver of DKO mice. The serum level of AST was lower in DKO mice while that of ALT was comparable after 48 hours of fasting, suggesting that liver damage in DKO mice was modest. In conclusion, the study shows that massive lipid accumulation in fasted DKO mice is not accompanied by liver function deterioration.

Keywords: FABP4, FABP5, fasting, liver function, fatty liver

1 INTRODUCTION

The emerging problem of fatty liver has become a major concern in research. The prevalence of fatty liver is 10 to 24% in general population and the number increases from 57 to 74% in obese people (Angulo, 2002). Fatty liver is associated with metabolic disorder, including hypertriglyceridemia and insulin resistance. The pathophysiology of fatty liver into inflammatory stage, Non-Alcoholic Steatohepatitis (NASH), has been proposed in “two hits” hypothesis (Tessari et al., 2009). The first hit is insulin resistance leading to fatty liver disease, followed by the second hit, the condition which involved oxidative stress, determining lipid peroxidation, increased cytokine production, and inflammation leading to NASH. It is well known that the susceptibility of advanced fibrosis or cirrhosis leads to death in patients with fatty liver (Tessari et al., 2009, George and Liddle, 2008).

Fasting can promote triglyceride accumulation in the liver, suggested as an adaptive mechanism in response to the lack of energy source (Teusink