

Cholic-acid rich diet induces the development of fatty liver and decreases the concentration of plasma adiponectin in rats

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Introduction: As one of the onset factors of metabolic disease, there is a diet-induced obesity by excessive energy intake. The cause of the excessive energy intake is increase of high-fat diet. Secretion amount of primary bile acid (BA) contributing to lipid absorption, increases by the long-term intake of high-fat diet. Some reports show that ingestion of a diet added with cholic acid (CA), a primary BA, has anti-obesity effect. However, we detected an enormous amount of conjugated BAs in the feces in similar experimental conditions with those reports, suggesting that a high dose of CA disturbs BA metabolism. Usually, conjugated BAs is not detected. Here, we found a moderate concentration of CA supplementation that enable normal BA conversion in the intestinal contents.

Aim: We investigate some parameters related to metabolic syndrome on this condition.

Methods: WKAH/HkmSlc male rats were fed an AIN-93G-based diet with or without the 0.05% CA supplementation for 13 weeks. We determined the parameter involved in a metabolic disease of the abdominal aorta of plasma, and determined triglyceride and total cholesterol of the liver tissue as a marker of lipid metabolism. In addition, we analyzed the concentration of total adiponectin secreted from fatty acid in the blood and the concentration of BA in feces and blood.

Results and Discussion: Ingestion of the CA diet increased liver weight accompanied by accumulation of triglyceride and total cholesterol despite no difference in adipose tissue weight. Elevations in some plasma parameters, such as cholesterol, non-esterified fatty acid (NEFA), aspartate aminotransferase (AST), and alanine aminotransferase (ALT), were observed in the CA-fed rats. Moreover, we observed increased serum transaminase, liver damaged markers, and development of fatty liver. Then we checked the concentration of plasma adiponectin involved in fatty liver. Adiponectin is protein secreted from adipocyte and decreased synthesis by the hypertrophy of adipocyte. In the result, the concentration of plasma adiponectin significantly decreased in CA-fed rats. On the other hand, no difference was observed in the expression of adiponectin in mesenteric adipose tissue between the groups. These results indicate that the reduction of adiponectin in the CA-fed rats does not necessarily depend on the gene expression in the mesenteric adipose tissue.

Key Words: bile acid, fatty liver

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