INTRODUCTION

Nonalcohlic fatty liver disease (NAFLD) is a clinicopathological syndrome characterized by hepatic steatosis with or without active inflammation in patients with a negligible alcohol intake **(Sheth et al., 1997)**. There is a growing concern about NAFLD, not only because this is a common liver disorder with a worldwide distribution, but also because it is recognized as one of the leading causes of chronic liver disease **(Yoon et al., 2005)**. In addition, a study has revealed that patients with nonalcoholic steatohepatitis (NASH) may progress to liver fibrosis, and approximately 20% progress to liver cirrhosis **(Diehl, 2002)**. Although NAFLD may occur in non-obese patients **(Sheth et al., 1997)**, most cases of NAFLD are associated with obesity, type 2 diabetes mellitus **(Marceau et al., 1999)** and hyperlipidemia **(Kelly et al., 2003)**. Weight reduction alone can improve liver function in obese patients with fatty liver **(Sears & Patel, 2005)**. Moreover, insulin resistance is suggested to underlie most cases of NAFLD, with a resultant increase in the circulating insulin levels **(Yoon et al., 2005)**.

Adiponectin is a protein secreted by adipose tissue, which displays several anti-atherogenic, anti-diabetogenic and anti-inflammatory effects **(Stejskal et al., 2005)**. In target tissues, it is an antagonist of tumor necrosis factor alpha **(Li, 2003)**. Adiponectin inhibits the production of glucose in the liver, enhances lipoprotein clearance and increases beta-oxidation of fatty acids **(Berg et al., 2002)**.

Experimental and clinical studies have repeatedly confirmed that adiponectin concentration shows a positive correlation with insulin sensitivity and a negative correlation with amount of adipose tissue

(Stejskal et al., 2005). Low adiponectin values have been associated with a high basal and reduced insulin-induced phosphorylation of tyroxin receptor for tyrosine kinase in muscle, resulting in progression of insulin resistance **(Stefan et al., 2002)**.

Low adiponectin values typically occur in obese individuals, type 2 diabetic patients, persons with metabolic syndrome and persons with coronary artery disease (CAD), while high adiponectin values are associated with good insulin sensitivity, lower frequency of type 2 diabetes mellitus and CAD **(Stejskal et al., 2005)**.

Insulin resistance is a state in which a given concentration of insulin produces a less than expected biological effect and also it has been defined as the requirement of 200 or more units of insulin per day to attain glycemic control and prevent ketosis **(Olatunbosun & Dagogo-Jack, 2004)**. And according to the homeostasis model assessment (HOMA) which is calculated as: fasting glucose (in mg/dl) / 18 **x** insulin (in micro unit/ml) /22.5, patient is considered to have insulin resistance if the result of the equation is more than 2.14 **(Haffiner et al., 1997).** Insulin resistance causes alterations in the uptake, synthesis, degradation, and secretion of hepatic lipids, which is suggested to be the metabolic cause of NAFLD **(Angulo, 2002)**. But an important question is still: which one of the insulin resistance and NAFLD is the cause of the other **(Bloomgarden, 2005)**.

Since low adiponectin is associated with insulin resistance, which seems to be closely correlated with NAFLD, it is hypothesized that there is a relationship between NAFLD, adiponectin and insulin resistance **(Yoon et al., 2005)**.