Abstract
Nonalcoholic fatty liver disease (NAFLD) is the most important cause of chronic liver disease and is considered the hepatic manifestation of the metabolic syndrome associated with diabetes mellitus type 2. The prevalence of NAFLD in the general population reaches 15-20%. It is also estimated that nonalcoholic steatohepatitis (NASH) affects 3% of the population. NAFLD refers to a wide spectrum of liver damage, which ranges from simple steatosis or intracellular triglyceride accumulation, to inflammation (NASH), fibrosis and cirrhosis. The mechanisms involved in the accumulation of triglycerides in the liver and subsequent hepatocellular damage are multifactorial and are not completely understood. However, metabolic changes such as insulin resistance (IR) are developed, being a common factor in the retention of fatty acids (FA) within the hepatocytes with oxidation and production of free radicals at the mitochondrial level, which are capable of causing lipid peroxidation, cytokine production, and necrosis. In addition, there are alterations in the hepatic bioavailability of long chain n-3 polyunsaturated fatty acids, conditions that alter the expression of a series of transcriptional factors involved in lipolytic and lipogenic processes in the liver. A greater knowledge of the etiopathogenic mechanisms of NAFLD is fundamental for the development of future effective therapeutic strategies. The pathophysiological fundamentals of liver steatosis are analyzed in this study.

Keywords
Fatty liver, Obesity, PPAR-alfa, SREBP-1c, n-3 polyunsaturated fatty acids.