Sleep deprivation predisposes liver to oxidative stress and phospholipid damage: a quantitative molecular imaging study

麥富德

Chang HM;Mai FD;Chen BJ;Huang YL;Lan CT;Ling YC

摘要

Abstract

Sleep disorders are associated with an increased rate of various metabolic disturbances, which may be related to oxidative stress and consequent lipid peroxidation. Since hepatic phosphatidylcholine plays an important role in metabolic regulation, the aim of the present study was to determine phosphatidylcholine expression in the liver following total sleep deprivation. To determine the effects of total sleep deprivation, we used adult rats implanted for polygraphic recording. Phosphatidylcholine expression was examined molecularly by the use of time-of-flight secondary ion mass spectrometry, along with biochemical solid-phase extraction. The parameters of oxidative stress were investigated by evaluating the hepatic malondialdehyde levels as well as heat shock protein 25 immunoblotting and immunohistochemistry. In normal rats, the time-of-flight secondary ion mass spectrometry spectra revealed specific peaks (m/z 184 and 224) that could be identified as molecular ions for phosphatidylcholine. However, following total sleep deprivation, the signals for phosphatidylcholine were significantly reduced to nearly one-third of the normal values. The results of solid-phase extraction also revealed that the phosphatidylcholine concentration was noticeably decreased, from 15.7 micromol g-1 to 9.4 micromol g-1, after total sleep deprivation. By contrast, the biomarkers for oxidative stress were drastically up-regulated in the total sleep deprivation-treated rats as compared with the normal ones (4.03 vs. 1.58 nmol mg-1 for malondialdehyde levels, and 17.1 vs. 6.7 as well as 1.8 vs. 0.7 for heat shock protein 25 immunoblotting and immunoreactivity, respectively). Given that phosphatidylcholine is the most prominent component of all plasma lipoproteins, decreased expression of hepatic phosphatidylcholine following total sleep deprivation may be attributed to the enhanced oxidative stress and the subsequent lipid peroxidation, which would play an important role in the formation or progression of total sleep deprivation-induced metabolic diseases.