
Vitamin A deficiency in patients with hepatitis C virus-related chronic liver disease.

Peres WA, Chaves GV, Gonçalves JC, Ramalho A, Coelho HS.

Department of Nutrition and Dietetics, Institute of Nutrition, Federal University of Rio de Janeiro

Abstract

Hepatitis C virus (HCV) infection is associated with oxidative stress and vitamin A possesses antioxidant activity. The objective of the present study was to investigate vitamin A nutritional status in chronic hepatitis, liver cirrhosis and hepatocellular carcinoma (HCC), according to biochemical, functional and dietetic indicators correlating these findings with liver function, liver damage and death. A total of 140 patients with HCV-related liver disease were enrolled.


Inhibition of mitochondrial respiratory chain in the brain of rats after hepatic failure induced by acetaminophen.


Universidade do Extremo Sul Catarinense, Criciúma, S.Catarina,

Abstract

Hepatic encephalopathy is an important cause of morbidity and mortality in patients with severe hepatic failure. Acetaminophen is frequently used in animals to produce an experimental model to study the mechanisms involved in the progression of hepatic disease. The brain is highly
dependent on ATP and most cell energy is obtained through oxidative phosphorylation, a process requiring the action of various respiratory enzyme complexes located in a special structure of the inner mitochondrial membrane.


A thioacetamide-induced hepatic encephalopathy model in C57BL/6 mice: a behavioral and neurochemical study.


Laboratory of Immunopharmacology, Department of Biochemistry and Immunology, Institute of Biological Sciences, Federal University of Minas Gerais, Belo Horizonte.

Abstract

Hepatic encephalopathy (HE) is a neuropsychiatric syndrome resulting from liver failure. In the present study, we aimed to standardize an animal model of HE induced by thioacetamide (TAA) in C57BL/6 mice evaluating behavioral symptoms in association with liver damage and alterations in neurotransmitter release. HE was induced by an intraperitoneal single dose of TAA (200 mg/kg, 600 mg/kg or 1,200 mg/kg).


Detoxification of ammonia in mouse cortical GABAergic cell cultures increases neuronal oxidative metabolism and reveals an emerging role for release of glucose-derived alanine.


Department of Biochemistry, ICBS, Federal University of Rio Grande do Sul, Porto Alegre.
Abstract

Cerebral hyperammonemia is believed to play a pivotal role in the development of hepatic encephalopathy (HE), a debilitating condition arising due to acute or chronic liver disease. In the brain, ammonia is thought to be detoxified via the activity of glutamine synthetase, an astrocytic enzyme. Moreover, it has been suggested that cerebral tricarboxylic acid (TCA) cycle metabolism is inhibited and glycolysis enhanced during hyperammonemia. The aim of this study was to characterize the ammonia-detoxifying mechanisms as well as the effects of ammonia on energy-generating metabolic pathways in a mouse neuronal-astrocytic co-culture model of the GABAergic system.


5) Ann Med. 2011 May 23

The Finnish Diabetes Risk Score (FINDRISC) as a screening tool for hepatic steatosis.

Carvalho JA, Barengo NC, Tuomilehto J, Conceição RD, Santos RD.

Preventive Medicine Center Hospital Israelita Albert Einstein, Sao Paulo, Brazil.

Abstract

Hepatic steatosis due to non-alcoholic fatty liver disease is associated with obesity, dyslipidemia, insulin resistance, and type 2 diabetes. The Finnish Diabetes Risk Score (FINDRISC) is a prognostic screening tool to detect people at risk for type 2 diabetes without the use of any blood test. The objective of this study was to evaluate whether FINDRISC can also be used to screen for the presence of hepatic steatosis. Steatosis was determined by ultrasound. The study sample consisted of 821 non-diabetic subjects without previous hepatic disease.

6) J Nutr Biochem. 2011 May 2

Maternal high-fat feeding through pregnancy and lactation predisposes mouse offspring to molecular insulin resistance and fatty liver.

Ashino NG, Saito KN, Souza FD, Nakutz FS, Roman EA, Velloso LA, Torsoni AS, Torsoni MA.

Universidade Braz Cubas, Mogi das Cruzes, São Paulo.

Abstract

The exposure to an increased supply of nutrients before birth may contribute to offspring obesity. Offspring from obese dams that chronically consume a high-fat diet present clinical features of metabolic syndrome, liver lipid accumulation and activation of c-Jun N-terminal kinases (JNK) consistent with the development of nonalcoholic fatty liver disease (NAFLD).


Absence of melatonin induces night-time hepatic insulin resistance and increased gluconeogenesis due to stimulation of nocturnal unfolded protein response.


Department of Pharmacology, Institute of Biomedical Sciences, University of Sao Paulo, Sao Paulo.

Abstract

It is known that the circadian rhythm in hepatic phosphoenolpyruvate carboxykinase expression (a limiting catalytic step of gluconeogenesis) and hepatic glucose production is maintained by both daily oscillation in autonomic inputs to the liver and night feeding behavior. However, increased glycemia and reduced melatonin (Mel) levels have been recently shown to coexist in diabetic patients at the end of the night period. In parallel, pinealectomy (PINX) is known to cause glucose intolerance with increased basal glycemia exclusively at the end of the night. The
mechanisms that underlie this metabolic feature are not completely understood.