Mol Cell Biochem. 2011 Apr;350(1-2):149-54. doi: 10.1007/s11010-010-0689-x. Epub 2011 Jan 4.

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

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PMID: 21203802 DOI: 10.1007/s11010-010-0689-x

Abstract

Hepatic encephalopathy is an important cause of morbidity and mortality in patients with severe hepatic failure. This disease is clinically characterized by a large variety of symptoms including motor symptoms, cognitive deficits, as well as changes in the level of alertness up to hepatic coma. 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. In this context, the authors evaluated the activities of mitochondrial respiratory chain complexes in the brain of rats submitted to acute administration of acetaminophen and treated with the combination of N-acetylcysteine (NAC) plus deferoxamine (DFX) or taurine. These results showed that acetaminophen administration inhibited the activities of complexes I and IV in cerebral cortex and that the treatment with NAC plus DFX or taurine was not able to reverse this inhibition. The authors did not observe any effect of acetaminophen administration on complexes II and III activities in any of the structures studied. The participation of oxidative stress has been postulated in the hepatic encephalopathy and it is well known that the electron transport chain itself is vulnerable to damage by reactive oxygen species. Since there was no effect of NAC + DFX, the effect of acetaminophen was likely to be due to something else than oxidative stress.

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