УДК: 61:615.1(043.2)
ББК: 5:52.82
А 43
ISBN: 978-985-21-0765-5

Kurzyna P. F., Sztolsztener K.

PROTECTIVE INFLUENCE OF DEXAMETHASONE ON HEPATIC FATTY ACIDS METABOLISM AND TRANSPORT IN HUMAN HEPG2 CELL LINE EXPOSED TO PALMITATE

Scientific supervisors: MD PhD prof. Chabowski A., PhD Konstantynowicz-Nowicka K.
Department of Physiology
Medical University of Białystok, Białystok

Introduction. Dexamethasone (DEX) is a pharmacological synthetic glucocorticoid (GC) with potent anti-inflammatory and immunosuppressive properties, which is used in the treatment of immunological, inflammatory and allergic diseases. Non-alcoholic fatty liver disease (NAFLD) that is described as excessive intracellular triacylglycerols (TAGs) accumulation, when accompanied by inflammation development may progress to non-alcoholic steatohepatitis (NASH).

Aims: the current study sought to determine the influence of dexamethasone in the presence of excessive palmitate bioavailability on hepatic lipid concentration and transport, which play a crucial role in NAFLD development and progression.

Materials and methods. The study was carried out on Human liver hepatocellular carcinoma cells (HepG2) which were incubated with palmitic acid (PA) and/or dexamethasone in two different time expositions (16h and 40h). Intracellular and extracellular lipid concentrations and fatty acid composition were estimated by gas liquid chromatography (GLC). The expression of proteins involved in fatty acid inport and export as well as lipid metabolism were determined by immunoblotting.

Results and discussion. The treatment of HepG2 cells with dexamethasone and palmitate enhanced lipid transport to the cell via increased expression of especially FABPpm and resulted in increased deposition of triacylglycerol (TAG) and diacylglycerol (DAG). Our results revealed that dexamethasone in combination with palmitate altered the fatty acid composition resulting in the elevated activity of n-3 polyunsaturated fatty acid (PUFA) pathway in DAG and TAG fractions and diminished activity of n-6 PUFA pathway in DAG fraction after prolonged exposure.

Conclusions. Prolonged exposure to DEX augmented the secretion of TAG and DAG into the medium, what may be DEX' protective mechanism against NAFLD deterioration in HepG2 cells. Moreover, after prolonged incubation with DEX in high availability of palmitate, observed increase in DAG' and TAG' anti-inflammatory n-3 PUFA as well as the decrease in DAG' proinflammatory n-6 PUFA activity confirmed the inhibitory effect of GCs on the development of inflammation that protected hepatocytes against NAFLD progression to NASH.