Prenatal Hypoxia Influence on Liver Tissue Homeostasis

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Background: Oxygen deficiency of the fetus can be followed by impairment of liver functioning. Prenatal hypoxia induces mitochondrial dysfunction in the fetal liver cells in experimental animals. Oxidative stress can be generated by several conditions, such as, prenatal hypoxia, maternal under-and over nutrition, and excessive glucocorticoid exposure. By linking oxidative stress with dysregulation of specific liver functions and tissue growing, we may be able to develop therapeutic strategies that protect against organ dysfunction. Methodology: In experimental research was performed artificial prenatal hypoxia on white laboratory rats. After prenatal hypoxia modeling was examined as newborn, as well as 4-weeks of age animals and detected that their body weight, ultimate liver weight and relative liver weight (in adult animals) decrease. Results: Investigations shown, that artificial prenatal hypoxia cause decrease DNA-synthetic activity of hepatocytes and intensify free-radical oxidation in the liver of newborn animals, inducing nucleolus number decrease in hepatocytes. In adult animals detect, also, reducing of hepatocytes size and their nucleolus area, increase number of binuclear hepatocytes. Thus, we can conclude, that prenatal hypoxia cause liver tissue homeostasis failure in both postnatal as well as in adult period.

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