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Letter to the Editor

Comment on "The influence of palatable high-energy diet in diet-induced obesity pregnant rats on offspring oxidative stress in liver"

Dear Editor,

We read with great interest the article by Jiang et al[1] on the effect of high-energy diet (HED) on oxidative stress in filial rat liver and their offspring. The authors evaluated both the expressions of malondialdehyde (MDA), reduced glutathione (GSH) and antioxidative enzyme activities and the expressions of inflammatory factors, tumor necrosis factor-α (TNF-α), IL-1β, mRNA level of heme oxygenase-1 (HO-1), COX-2 and nuclear factor kappa B (NF-κB) expressions. They found in offspring rats of HED group, significant reductions in the activities of a number of antioxidant enzymes, such as glutathione S-transferase (GST), superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GSH-Px), and a significantly higher level of MDA. Moreover HO-1 mRNA, the expression of COX-2 and p-NF-κB-p65 in HED group offspring rat liver, is significantly higher than in the control group offspring rat liver, with slight structure deformation and misalignment of the liver.

Also Liu et al[2] studied the effect of high-fat diet (HFD) on five-month-male spontaneously hypertensive rat which undergo acute vasoconstriction-stress and severe multi-organ damage. Encephalopathy, heart dysfunctions, hepatic and renal dysfunctions, and blood system disorders were testified through the detections of some parameters covering hepatic and renal function, blood glucose and lipid levels, myocardial enzymes and energy metabolisms, blood coagulative and anti-coagulative system, oxidative stress (OS) and anti-inflammatory cytokines. Furthermore, some studies[3] reveal how high consumption of red and/or processed meat was independently associated with higher odds of non-alcoholic fatty liver disease (NAFLD) and insulin resistance.

Xia et al[4] analyzed the effect of antihyperlipidemic and antioxidant activities of niga-ichigoside F1 (NI) on male C57BL/6J mice fed a standard diet or a HFD with or without 40 mg kg^{-1} NI for 12 weeks. NI alleviated hepatic steatosis, interacting with HFD to regulate lipid metabolism genes. The authors demonstrated the important effects of antioxidant agents on the natural history of the disease, like in similar articles[5,6].

It is known that hepatic lipid accumulation, advanced glycosylation end products (AGEs)[7], and OS lead to NAFLD. Although the etiologies of dyslipidemias and NAFLD are complex and multifactorial, the pathogenesis of these diseases contains an important genetic component that is strongly influenced by environmental factors and life habits[8], especially the dietary intake profile.

NAFLD is becoming one of the most global heath problems in both developed and developing countries. It is a circumstance that may progress towards liver cirrhosis[9], HCC and its complications[10,11]. For these reasons, it would be interesting to see if these factors can also further the neoangiogenesis, certainly implicated in the development of liver disease[12].

Conflict of interest

The authors declare no conflicts of interest.

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