Dietary methionine imbalance in pregnant mice induces lipid peroxidation in the offspring liver

Tarsila Daysy Ursula Hermogenes Gomes¹*; Alexandre Ferro Aissa²; Lívia Cristina Hernandes¹; Vinícius de Paula Venâncio¹; Mara Ribeiro de Almeida¹; Joana D’Arc Castania Darin¹; Maria de Lourdes Pires Bianchi¹, Lusânia Maria Greggi Antunes¹.

¹Faculdade de Ciências Farmacêuticas de Ribeirão Preto, Universidade de São Paulo; ²Faculdade de Medicina de Ribeirão Preto, Universidade de São Paulo. *tarsilagomes@yahoo.com.br

Introduction: Several studies have demonstrated that a nutritional imbalance in maternal diet can affect organs metabolism and can cause risk of disease in the offspring life. One of the important nutrient present in the diet is the amino acid methionine (Met). It is required not only for protein synthesis, but also for methyl donation in the DNA methylation process. It is metabolized by liver and its byproduct, homocysteine, may increase reactive oxygen species (ROS) levels. ROS can damage biological structures, such as lipid membrane and DNA, and trigger the lipid peroxidation. The lipid peroxidation products can be evaluated by the thiobarbituric acid reactive substances (TBARS) quantification. Therefore, it is important to assess how different Met intakes may affect the lipid peroxidation. Objective: The aim of this study was to evaluate the Met imbalance in the hepatic lipid peroxidation of mice offspring treated with different Met diets, by evaluation of TBARS, an important biomarker related to oxidative stress. Methods: Male mice offspring of dams, previously treated with either a control (0,3% DL- Met) or a supplemented (2,0% DL- Met) diet, were treated with the same Met diets ad libitum during 18 weeks. Another offspring group was treated with a deficient (0% DL-Met) diet. Then, the animals were euthanized and the liver was collected to quantify TBARS, by a spectrophotometric assay. Results: The offspring of supplemented dams, which fed either the supplemented or the control Met diet, presented a reduction (p<0,05) of the lipid peroxidation, when compared to the control offspring group. On the other hand, the deficient offspring of the supplemented dams presented an increase (p<0,05) of the lipid peroxidation level. Both the Met supplementation and deficiency had no effects on the lipid peroxidation level of offspring of control dams. Conclusions: The results demonstrate that a previous treatment in the gestational period may influence the hepatic lipid peroxidation of offspring, reducing or increasing it. Further studies have been performed to evaluate other biomarkers and other possible alterations in liver induced by Met dietary imbalance.

Keywords: methionine, supplementation, deficiency, pregnancy, mice, liver, lipid peroxidation.

Financial support: FAPESP, CNPq, CAPES.