Effects of the Interaction of Diabetes and Iron Supplemented on Liver PPAR-alpha Expressions

Silva, M.¹; Oliveira, R.P.²; Silva, M.E.³; Pedrosa, M.L.¹

¹Dep de Ciências Biológicas, ²Dep de Biodiversidade, Evolução e Meio Ambiente, ³Dep de Alimentos, Núcleo de Pesquisas em Ciências Biológicas (NUPEB), Universidade Federal de Ouro Preto (UFOP), Ouro Preto, MG

Increased iron stores have been found to predict diabetes development, but the exact mechanism is still unknown. On the other hand, diabetes can modulate PPARs through increased inflammatory cytokines and oxidative stress. Thus it is possible to suppose that the interaction between iron and diabetes might modulate the PPARα expression in the liver. This study evaluated the effects of the interaction of diabetes and a carbonyl iron supplemented on liver oxidative stress markers and PPAR-alpha expressions. Hamsters were divided into four groups: Control (C), which received a standard AIN 93 diet; Control Iron (CI), composed of control animals that received a diet with 0.83% carbonyl iron; Diabetic (D), composed of animals that received a injection of streptozotocin (STZ, 50 mg/Kg) on day 35; and Diabetic Iron (DI) composed of streptozotocin treated animals that received a diet supplemented with carbonyl iron. Diabetes decreased mRNA levels of PPAR alpha. Iron attenuated the diabetes induced down regulation of PPAR alpha mRNA. Moreover, diabetes increased carbonyl protein in 62% and decreased glutathione levels in 14% and catalase activity in 27%, while iron attenuated the increase in levels of carbonyl protein and attenuated the decrease in those of glutathione level and catalase activity. The results show that iron does not aggravated liver oxidant/antioxidant status and PPAR alpha expression in diabetic hamsters.

Word Keys: Hamsters, carbonyl iron, diabetes, oxidative stress, PPAR alpha
Supported by: FAPEMIG, CNPq and CAPES