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Effects of lipoic acid supplementation on activities of cyclooxygenases and levels of prostaglandins E2 and F2 alpha metabolites, in the offspring of rats with Streptozotocin-induced diabetes

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Background & Aim: Uncontrolled diabetes mellitus (DM) is an etiological factor for recurrent pregnancy loss and major congenital malformations in the offspring. Antioxidant therapy has been advocated to overcome the oxidant-antioxidant disequilibrium inherent in diabetes. Our aims were to evaluate the protective effect of lipoic acid (LA) on fetal outcome and to elucidate changes that may be involved in the mechanisms implicit diabetic fetopathy.

Methods: Female rats were rendered hyperglycemic using streptozotocin and then mated with normal male rat. Pregnant non-diabetic (group-1; n=9 and group-2; n=7) or pregnant diabetic (group-3; n=10 and group-4; n=8) rats were treated daily with either LA (30 mg/kg body weight; groups-2 and 4) or vehicle (groups-1 and 3) between gestational days 0 and 15. On day 15 of gestation, the rats were sacrificed and the fetuses, placentas and membranes dissected out of the uterine horns. Following morphological examination, the fetuses, placentas and membranes were homogenized and used to measure cyclooxygenase (COX) activities and metabolisms of prostaglandin (PG) E2 (PGEM) and PGF2α (PGFM) levels. Maternal liver and plasma and in the fetuses of all groups..

Results: Supplementation of diabetic rats with LA was found to significantly ($p<0.05$) reduced resorption rates in diabetic rats and increased mean fetal weight compared to vehicle-treated diabetic (V-TD) group. Treatment of diabetic rats with LA (LA-TD) leads to a significant ($p<0.05$) increase in liver and plasma total glutathione, in comparison with V-TD rats. Decreased levels of PGEM and elevated levels of PGFM in the fetuses, placentas and membranes were characteristic of experimental diabetic gestation associated with malformation. LA treatment to diabetic mothers failed to normalize levels of PGEM to the vehicle-treated control rats. However, the levels of PGEM in malformed fetuses from LA-TD mothers was significantly ($p<0.05$) higher than those in malformed fetuses from V-TD rats.

Conclusions: LA can reduce congenital malformations in the offspring of diabetic rats at day 15 of gestation. Thus, LA treatment did not completely prevent the occurrence of malformations, other factors such as arachidonic acid deficiency and altered prostaglandin metabolism may be involved in the pathogenesis of the diabetes-induced congenital malformations.

Biography

Hisham Al-Matubsi has completed his PhD from Victoria University-Australia and worked at different academic levels in different reputable academic organizations such as Victoria University-Victoria, Australia, Cincinnati University-Ohio, USA and now at The University of Petra-Amman, Jordan. Dr Al-Matubsi's professional interests are in the area of reproductive physiology research, with a specific emphasis on ovarian hormones and changes that may be involved in the mechanism(s) underlying diabetic fetopathy. He has published more than 20 papers in reputed journals and is serving as an editorial board member of repute journal Diabetes and related Disorders.

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