Biochemistry 2018: Effects of Lipoic Acid Supplementation on Activities of Cyclooxygenases and Levels of Prostaglandins E 2 and F 2α Metabolites, in the Offspring of Rats With Streptozotocin-Induced Diabetes - Hisham Y Al-Matubsi - University of Petra, Jordan

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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. We aimed to estimate the protective effect of lipoic acid (LA) on the fetal outcome and to elucidate changes that may be involved in the mechanisms of implicit diabetic fetopathy.

Methods: Female rats were rendered hyperglycemic using streptozotocin and then mated with a 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 the morphological inspection, 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 originate to meaningfully ($p<0.05$) reduced resorption rates in diabetic rats and increased mean fetal weight compared to the 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 were 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. Drug-induced diabetes is when the use of a specific medication has lead to the development of diabetes. In some cases, the development of diabetes may be reversible if the use of the medication is discontinued, but in other cases, drug-induced diabetes may be permanent. Hypoglycaemic drugs were also reduced by 50% in all patients when given supplemental chromium.

These data demonstrate that corticosteroid treatment increases chromium losses and that steroid-induced diabetes can be reversed by chromium supplementation. Is steroid-induced diabetes permanent? High blood glucose levels whilst taking steroids may subside after you stop taking steroids, however, some people may develop type 2 diabetes which will need to be managed for life. Generally, blood sugar levels should return to their previous levels 1–2 days after finishing steroid treatment. However, some people may develop type 2 diabetes as a result and will need appropriate follow-up treatment with oral medication or insulin therapy. Steroid-induced diabetes, then, is a sign that your pancreas is not entirely normal, that it may be fine when not challenged by steroids, but is limited when you do have to take the steroids But the good news is that when the dose of steroids is reduced or they are stopped altogether, diabetes may well go away. Prednisone and other steroids can cause a spike in blood sugar levels by making the liver resistant to insulin. The pancreas produces insulin to control blood sugar levels. Diabetes can result from a fault in the way that the body reacts to insulin or a problem with the production of insulin in the pancreas. One of the side effects of oral corticosteroids is that they can increase blood glucose levels and increase insulin resistance, which can lead to type 2 diabetes. Being on steroids for a longer period, over 3 months, may also increase your risk of type 2 diabetes. Treatment with a faster-acting diabetes medication such as insulin, or a sulfonylurea such as glipizide, will control the elevated blood glucose during steroid treatment and may have worked better for you. How is steroid-induced diabetes treated? The treatment for diabetes you are put on may depend on the extent of insulin resistance and how high your blood glucose levels are. It may be possible to treat your diabetes with diet and physical activity but you may need oral anti-diabetic medication or insulin. This is why an increase in the stress hormone increases the body's stores of glucose. Long-

term prednisone use can cause diabetes in someone who tends to be diabetic. Moreover, the higher the dose of prednisone, the greater the likelihood that the blood glucose (sugar) level will rise. However, prednisone produces side effects that can make users more susceptible to type 2 diabetes. Common side effects of prednisone include increased insulin production, high blood sugar, weight gain, and high blood pressure. Normal blood sugar levels are less than 100 mg/dL after not eating (fasting) for at least eight hours. And they're less than 140 mg/dL two hours after eating. During the day, levels tend to be at their lowest just before meals.