Impact of Oxidative Stress in Diabetic Pregnant Rats Strain Wistar Treated by Hesperidin

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Abstract: The objective of this study aims to evaluate the effect of a free radicals-scavenging antioxidant, hesperidin, on the susceptibility of the diabetic rats to the diabeto-teratological complications during pregnancy. Indeed, the intraperitoneal administration of Streptozotocin (SZT) at a rate of 60 mg/Kg of body weight induces an experimental diabetes mellitus in 2-4 days. Work was centered on the one hand, by a hyperglycemia-associated considerable reduction in the maternal hepatic and maternal cardiac of the reduced glutathione (GSH) and on the other hand, by an increase in the maternal cardiac glutathione S-transferase (GST) activity, biomarkers of an oxidative stress. The treatment of the pregnant diabetic rats by hesperidin restored the maternal cardiac of GSH, attenuated hyperglycemia and dyslipidemias in the dams rats. This suggests a prophylactic effect of hesperidin opposite the embryonic malformations occurring at the time of badly followed -diabetes type 1.

Key words: Hesperidin • Rats • Diabetes Type1 • Oxidative Stress

INTRODUCTION

Diabetes is a serious disease that is linked to the development of a multitude of neuro-immune and metabolic complications including oxidative stress. Location researchers involved in most human diseases (cancer, immune deficiencies, neurodegenerative diseases...).

To investigate the etiology of the disease and because of the Gravitee its many metabolic and neurodegenerative effects in 1974 Portha [1] established diabetes in rats clinically by administration of streptozotocin, a substance that has selective toxicity on ß cells of the islets of Langerhans in the endocrine pancreas thereby inducing insulin-dependent diabetes [2].

Flavonoids are powerful antioxidants that may inhibit the formation of free radicals and to oppose the oxidation of macromolecules reporting to the efficacité several medicinal plants as natural anti-diabetic [3]. In this context our study is to explore the involvement of oxidative stress in diabetic pathology rated the antioxidant power hesperidin on oxidative stress in pregnant rats apply.

MATERIALS AND METHODS

Animals: The biological material base that we have chosen is the rat Rattus rattus of the Wistar strain from Pasteur Institute in Algiers. The rats are nocturnal mammals of the order of rodents. Upon their arrival, the rats weighed an average of 180 grams and at the time of the experiment, they weighed on average 250 ± 20 grams. The rats were acclimated under standardized conditions of natural photoperiod, an average temperature of 22 ± 4 °C and humidity of 50-70%. After an adaptation period of three weeks, we have selected 25 females based on weight which we separated into five experimental groups each include five rats control group T, vehicle control CV lot, lot control treated hesperidin CH Lot diabetic vehicle DV lot diabetic treated hesperidin DH.

Treatment of Animals

Administration of Streptozotocin: Streptozotocin (STZ) is a chemical commonly used in animal models for the study of diabetes [4]. Diabetes was induced in rats by intraperitoneal injection of STZ (Sigma Lowis ST, Mo) at a dose of 60 mg / kg body weight [5] dissolved in a 0.1M sodium citrate buffer pH 4.5.
Administration of Hesperidin: Administration is by gastric gavages of rats to a high dose of 100 mg / kg body weight. Treatment with vehicle or the antioxidant Nacl for controls begin 72 hours after the induction of diabetes and it is for three days (15th, 17th and 18th day gestational).

Determination of Reduced Glutathione (GSH): The assay of GSH is based on the colorimetric method of Ellman [6]. The principle is based on the oxidation reaction of GSH with acid 5,5'-dithiobis - 2 nitrobenzoic acids (DTNB) and releasing the absorbent at 412 nm thionitrobenzoic acid (TNB). For this assay, one gram of organ was homogenized in three volumes of 5 % TCA using a mill and then centrifuged at 1000 revs / min. 50 μl of supernatant are diluted in 10 ml of phosphate buffer (0.1 M, pH 8). To 3 ml of the dilution mixture, 20 μl DTNB (0.01 M) are added, the absorbance measurement is obtained at a wavelength of 412 nm.

Determination of Glutathione S-Transferase (GST): The measurement of the overall GST activity is to provide to the various isoenzymes CDNB chlorodinitrobenzene. The CDNB readily reacts with GSH to form an enzymatically light at 340 nm absorbing conjugate. The value of the optical density is proportional to the bound GST activity [7]. The procedure is to mix 840 μl phosphate buffer (100 mM, pH 6.5), 50 μl CDNB, 10 μl sample then add 100 μl GSH. The measurement of the enzymatic activity was performed for 5 minutes.

The concentration of proteins in each organ was determined by the method of Bradford M [8] by using a calibration curve previously made by means of bovine serum albumin (BSA).

Statistical Analysis of Results: Results are presented as mean ± SEM and shown in histograms. A comparison test was used medium. The test T of Student with the MINITAB program for comparing two averages.

RESULTS

Cardiac Content of Reduced Glutathione (GSH) in Pregnant Rats: The results showed a very significant (P < 0.01) in glutathione content in diabetic rats vehicle and significant increase (P < 0.05) contribution to the controls

(DV: 24±2.64 vs T: 59±8.91) (DH: 35,6±4,92 vs T: 59±8,91) (DH: 35,6±4,92 vs DV: 24±2,64).

Measuring the Cardiac Activity of Glutathione S-Transferase (GST) in Pregnant Rats: The results indicate a very significant increase (P < 0.01) in GST concentration in diabetic rats treated with vehicle and hesperidin by contribution to controls


Fig. 1: Variation of the concentration of cardiac GSH nmol / mg protein

Ns.: non significant difference P > 0.05 ; *P < 0.05 ; **P < 0.01; ***P < 0.001
Our experimental study focused on properties that potentiate hesperidin fight against neuro behavioral alterations in rats of Wistar diabetic pregnant.

The relationship between DHC complications and pro status / maternal antioxidant in recent years was a journey of extensive research [9]. Our results have showed a highly significant decrease in cardiac reduced glutathione (GSH) in diabetic pregnant rats. It is ubiquitous tripeptide core network of the cellular antioxidant which includes a series of enzymatic systems scavengers, such as that of glutathione S-transferase (GST) [10]. The increase in total activity of cardiac GST in the untreated diabetic lot reflects the mobilization of anti-radical defenses dependent GSH in response to intense oxidative stress, indicating that chronic hyperglycemia generalize the production of free radicals in the body. Supplementation of hesperidin partially restores the cardiac GSH levels while simultaneously attenuates hyperactivity S-transferase in the heart.

What prompts us to consider our results as positive for the administration of flavonoids was largely due to a decrease in oxidative phenomena via mechanisms involving the interference of these polyphenols with GSH-dependent enzyme system [11]. In this sense, the antioxidant power of hesperidin has been evaluated in several scientific contexts in which it protected the liver mesenchymal against oxidative stress induced by toxic molecules [12].

This crucial issue to which we are interested, is summed up in the fact that the induction of experimental diabetes mellitus in pre-pregnant female mice causes 72h after a disturbance of the biochemical metabolism and causes a state of oxidative stress that is shown, on the one hand, by increasing the activity of glutathione-S-transferase (GST) with a strong cellular uptake of reduced glutathione (GSH ) in the Heart of maternal bodies. hesperidin appears to prevent the complications of diabetes through an effective antioxidant effect.

REFERENCES


