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Research Article

Influence of Flavonoid-Rich Fruit and Vegetable Intake on Diabetics-Related Biomarkers

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ABSTRACT

Our problem is to evaluate the behavioral effects of infection and combination diabetes - infection in pregnant rats and the protective effect of an antioxidant on neurobehavioral alterations and complications. Diabetes was induced by a single intraperitoneal injection of streptozotocin (STZ) at a dose of 60 mg / kg.

The administration of stresozotocine which is revealed, on one hand, the effects of of a single STZ i.p. injection on anxiety- and depression-like behaviors, brain oxidative status and immune response in adult wistar rates, and the other hand the protective role of antioxidant (Hesperidin) on STZ-induced disorders.

The hesperidins, a natural flavonoid was administered orally (gavages) at a dose of 100 mg / kg. This administration after WAS oxidative brain status showed an increase in glutathione-S-transferase (GST) activity and a decrease in reduced glutathione (GSH).

Key word: Diabetes, GHS, GST, Hesperidin, 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 β 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 efficacitée 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 stress control CSV lot , lot control stress treated hesperidin CSH ,Lot diabetic stress vehicle DSV lot diabetic stress treated hesperidin DSH .

Treatment of Animals

Administration of streptozotocin

Streptozotocin (STZ) is a chemical commonly used in animal models for the study of diabetes[4]. Diabetes was

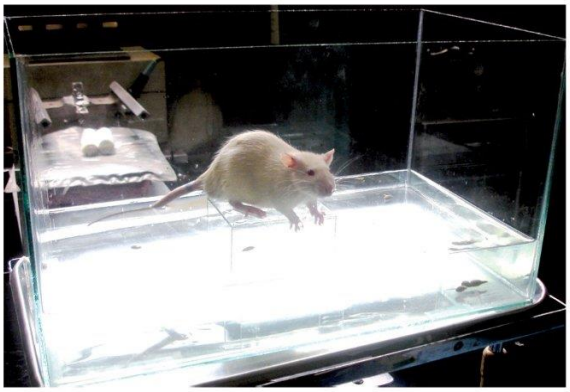

Figure 1 : WAS conditions. A mouse was placed on a glass platform located in the middle of a plastic container filled with water up to 1 cm below the level of the platform. The mouse on the platform cannot escape even if it jumps.

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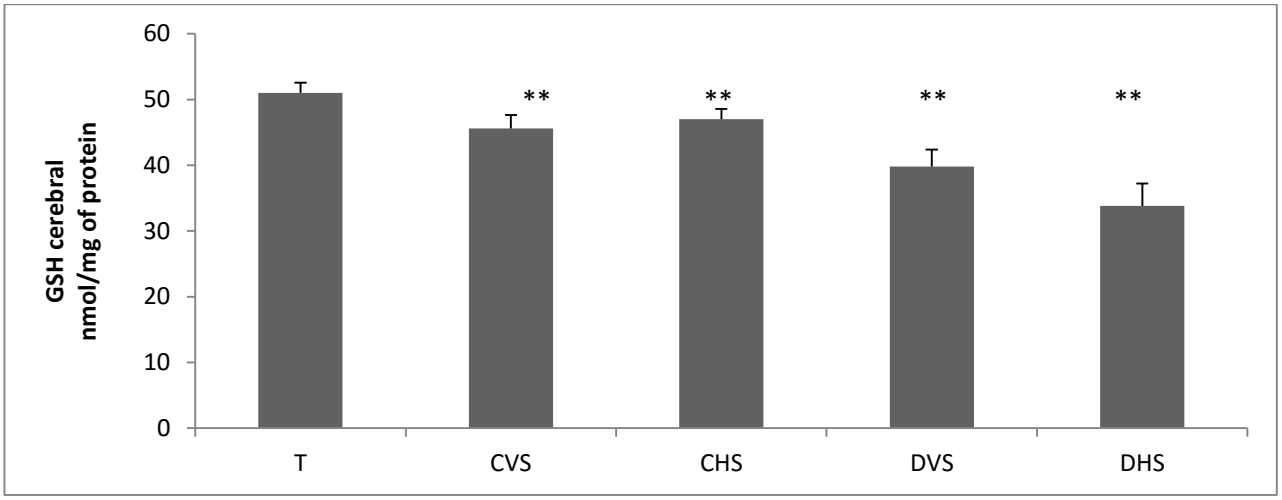

Figure 2: Variation of the concentration of cerebral GSH nmol / mg protein
Ns. : non significant difference P > 0.05 ; *P < 0.05 ; **P < 0.01; ***P < 0.001

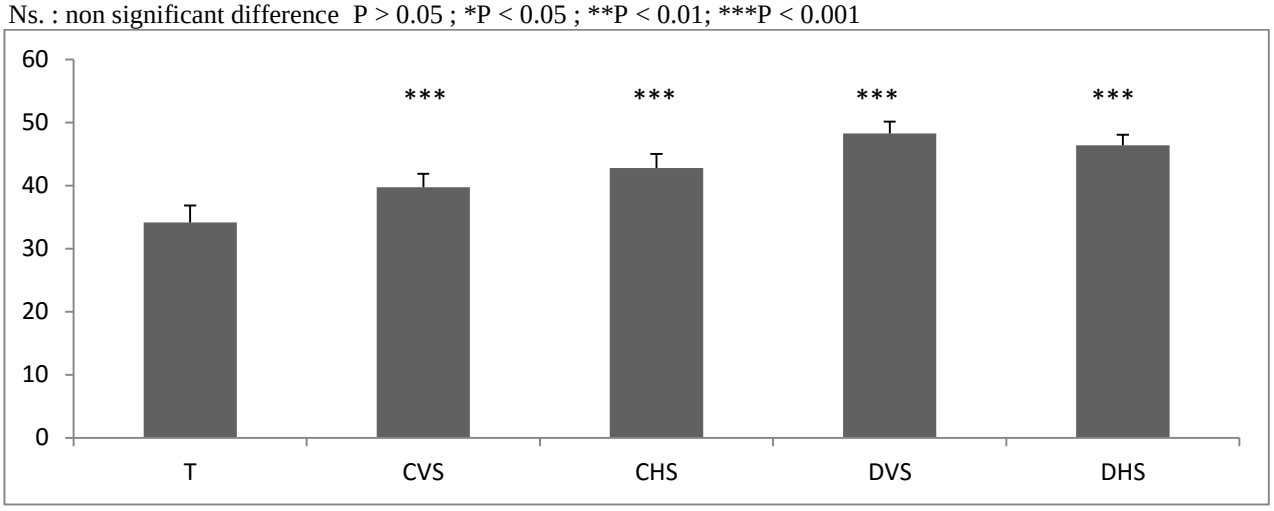

Figure 3: cerebral activity variation of GST in µmol/min/mg of proteins
Ns: Non significant difference P > 0.05 ; *P < 0.05 ; **P < 0.01; ***P < 0.001

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).

The Stress Model (WAS Protocol)

WAS as reported by Santos et al (2000) [6]was chosen as the model of psychological stress. WAS consisted of placing a mouse for 1 h on a glass platform (height from floor, 11 cm; diameter, 7cm) located in the middle of a plastic container (60_58cm) filled with sterile water up to 1 cm below the level of the platform .

Determination of reduced glutathione (GSH)

The assay of GSH is based on the colorimetric method of Ellman[7]. 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 mu.l of supernatant are diluted in 10 ml of phosphate buffer (0.1 M, pH 8). To 3 ml of the dilution mixture, 20 .mu.l 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 acivité 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[8]. son procedure is to mix 840 mu.l phosphate buffer (100 mM , pH 6.5), 50 mu.l CDNB , 10 .mu.l sample then add 100 µ 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[9] 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

Cerebral Content Of Reduced Glutathione (Gsh) In Pregnant Rats

The results showed a very significant ($P < 0.01$) in glutathione content in diabetic rats vehicle stress and significant increase ($P < 0.01$) contribution to the controls stress.(Fig. 2)

Measuring the cardiac activity of glutathione S-transferase (GST) in pregnant rats

The results indicate a very significant increase ($P < 0.001$) in GST concentration in diabetic rats stress treated with vehicle and hesperidin by contribution to controls stress. (Fig.3)

DISCUSSION

Our experimental study focused on properties that potentiate hesperidin fight against neuro behavioral alterations in rats of Wistar diabetic pregnant.

The relationship between complications and pro status / maternal antioxidant in recent years was a journey of extensive research[10]. Our results have showed a highly significant decrease in cerebral 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)[11]. The increase in total activity of cerebral 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 cerebral organs.

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[12]. 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[13].

CONCLUSION

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 cerebral organs of maternal bodies. hesperidin appears to prevent the complications of diabetes through an effective antioxidant effect.

REFERENCES

1. Portha, B.,1974. In: noninsulin-dependent diabetes mellitus. Dr Fatalis CR. Raynal AFP. Paris
2. Omari, N., Y. Dahmani-aït akli, F Labrousse and F.Hadj bekkouche , 2011.Influence of the streptozotocin on the corticotrope axis of the Wistar rat (Rattus norvegicus)., 80,907- 938
3. Toumi, M.L., S. Merzoug, A Boutefnouchet, A. Tahraoui, K Ouali and M.A.Guellati, 2009. Hesperidine, a natural citrus flavanone, alleviates hyperglycaemic state and attenuates embryopathies in pregnant diabetic mice. Journal of Medicinal Plans Research Vol. 3(11), pp. 862-869
4. Frode, T.S and Y. S. Medeiros, 2008. Animal models o test drugs with potential antidiabetic activity Journal of Ethnopharmacology., 155, 173-183.
5. Akbarzadeh, D., M. Norouzian, R. Mehrabi, Sh, Jamshidi., A. Farhangi, A. Allah Verdi, S. M. A. Mofidian and B. Lame Rad, 2007 . Induction of diabetes by Streptozotocin in rats Indian J Clin Biochem., 22(2), 60–64.
6. Santos J, Yang PC, So¨derholm JD, Benjamin M, Perdue MH (2001). Role of mast cells in chronic stress induced colonic epithelial barrier dysfunction in the rat. Gut 48: 630–636.
7. Ellman, G. L.,1959. Plasma antioxidants. Arch. Biochemistry and Biophyisics., 82, 70-77.
8. Habig, W H., M. J. Pabs and W. B. Jacobi, 1974. The first enzymatic step in mercapturic acid formation J Biol Chem., 249, 137-143
9. Sabu M C, Smitha K, Ramadasan K (2002). Anti-diabetic activity of green tea polyphenols and their role in reducing oxidative stress in experimental diabetes *Journal of Ethnopharmacology* 83: 109-116
10. Valko, M., D. Leibfritz, J. Moncola, M.T. D Cronin, M Mazura and J. Telser, 2007. Free radicals and antioxidant in normal physiological functions and human disease The International Journal of Biochemistry & Cell Biology., 39, 44-84
11. Abidi, P., F. Aaqq, J M. Arif, M Lohani and Q. Rahman, 1999. Chrysotile-mediated imbalance in the glutathione redox system in the developement of pulmonary injury Toxicol Lett., 106, 31-39
12. Dastmalchi, K., H. J. D. Dormana, P.P. Oinonena, Y Darwis, I Laakso and R . Hiltunen, 2008. Chemical composition and in vitro antioxidative activity of lemon balm (Melissa officinalis L) extract LWT., 41, 391-400
13. Tirkey, N., S. Pilkhwal, A Kuhad and K. Chopra, 2005. Hesperidin, a citrus bioflavonoid, decreases the oxidative stress produced by carbon tetrachloride in rat liver and kidney BMC Pharmacol., 5(1), 2