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BIOCHEMICAL EFFECTS OF TAURINE ON EXPERIMENTALLY INDUCED HYPERGALACTOSAEMIA IN RATS

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A B S T R A C T

In recent years, there has been a great demand for natural products that have possible preventive action against diabetes and its secondary complications. Current diabetes research makes use of hypergalactosemic rat as experimental models of type I diabetes mellitus. Keeping this in mind, our study was undertaken to investigate the influence of taurine on galactose and glucose levels, oxidative stress markers and lipid profile in galactose-induced hypergalactosemic rats. 70 weaning albino male rats divided into four groups were used in this study. Rats treated daily with 30% galactose (6 g/kg) orally for 80 days (group C; n=25) to induce hypergalactosaemia. Control rats received normal chow diet (group A; negative control group, n=10). Rats treated with 4% taurine (0.8 g for each rat daily) (group B; positive control group, n=10). The fourth set of rats received 30% galactose and 4% taurine (group D; n=25). Taurine administration started after 80 days from the beginning of the experiment and continue for 21 days. Galactose and glucose levels as well as body weights were estimated weekly to monitor hypergalactosaemia and hyperglycemia induced by galactose feeding. Serum were collected at the end of the experiment to determine insulin, lipid profile, total protein content, antioxidant enzymes (superoxide dismutase (SOD), catalase(CAT) and glutathione peroxidase (GSH-Px) activities), and reduced glutathione (GSH) levels, while glycogen estimated in liver tissues of rats. In addition HbA1c was determined in whole blood of rats. The results of this study imply that treatment of rats by taurine can control, to some extent, galactose-induced hypergalactosaemia, hyperglycemia and its attendant complications.

KEY WORDS: galactose, experimental hypergalactosaemia, taurine, diabetes mellitus, antioxidants.

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1. I N T R O D U C T I O N

Galactosaemia considered as one of the most mysterious of the heavily-researched metabolic diseases. It is among the most common carbohydrate metabolism disorders and can be a life-threatening illness during the newborn period [1]. It is a group of diseases marked by high levels of blood galactose resulting from the inability to use galactose to produce energy [2]. This inability of the body to metabolize galactose causes the accumulation of galactose-1-phosphate in the body and this causes damage to the liver (cirrhosis), kidneys (Fanconi syndrome), central nervous system (mental retardation), eyes (development of cataract), ovaries (primary or secondary amenorrhea) and other body systems. It has been estimated that hereditary intolerance to galactose occurs in approximately one in 18,000 infants. It occurs in approximately 1 out of every 60,000 births among Caucasians, while the rate is different for other groups [3].

Galactosemia is inherited in an autosomal recessive manner. It affects both boys and girls equally. Infants with galactosemia appear normal at birth; however, symptoms usually appear a few days to two weeks after initiating milk

feedings. The early clinical features of severe galactosemia include liver dysfunction; manifested by jaundice and hypoglycemia, neurological findings of irritability and seizures, and gastrointestinal findings of poor feeding, vomiting and diarrhea [4]. Galactosemia can kill quickly. it should be considered in any infant with nonglucose reducing substances in the urine. It is important to emphasize that testing of urine with glucose oxidase (Clinistix, Tes-tape) will not detect galactose. This is a strong argument for continued use of the older methods for the screening of urine for reducing substance (Benedict or Fehling test, Clinitest) [5]. There are 3 forms of the disease: galactose-1 phosphate uridyl transferase deficiency (classic galactosemia, the most common and most severe form), deficiency of galactose kinase, and deficiency of galactose-6-phosphate epimerase) [6].

High blood sugar is toxic to many parts of the body, and the eyes are no exception. Indeed, people with diabetes and galactosemia are 25 times more likely to go blind than those who do not have the disease. Elevated plasma glucose or galactose in the blood results in accumulation of sorbitol or galactitol in the lens. They are difficult to diffuse out of lens; causing increased osmotic pressure. High glucose also causes nonenzymatic glycation of lens proteins. All these cause an opaque cloudiness of the lens known as cataract. Galactitol is not further metabolized and diffuses out of the lens very slowly. Thus, hypergalactosemia is even more likely to cause cataract than hyperglycemia [7].

However, galactose, has been used to study the development of hyperglycemia and its complications due to its ability to increase glycation–[8]. The abnormalities detected in experimental galactosemic animals including retinopathy, cataracts, neuropathy, nephropathy and generalized basement membrane thickening are more compatible with findings in experimental diabetes mellitus than in human galactosemia. Because patients with galactokinase deficiency fail to manifest the central nervous system (CNS) and ovarian complications which characterize classic galactosemia [2], current diabetes research makes use of hypergalactosemic rat models to reproduce pathologic seen in type I diabetes mellitus [9].

Taurine (2-aminoethane sulphonic acid) is the most abundant free amino- acid present in animal tissues and is involved in the development and function of many organs such as brain, liver, kidney, eye and heart [10]. Taurine is found to stimulate glycolysis, glucogenesis and also reported to have insulin-like action [11]. So many beneficial effects of taurine supplementation in diabetes mellitus have been recently suggested as a result of its hypoglycaemic (taurine decrease the concentration of glucose & fructosamine and increase the contents of insulin & glycogen in the liver and has a positive effect on β-cell function), antioxidative & nephroprotective effects [12].

2. MATERIAL AND METHODS

2.1. Animals:

70 male albino rats weighing 175-200 g were used in this study obtained from Laboratory Animals Research Center, Faculty of Veterinary Medicine, Benha University, Egypt. Rats were housed in separated metal cages 10-25 per cage and kept under the same constant environmental and nutritional condition throughout the period of investigation; water was supplied *ad Libitum*, in the special laboratory animal room at Faculty of Vet. Med. Moshtohor, Benha University.

2.2. Chemicals:

Galactose and taurine were purchased from El-Goumhouria Co. for trading chemicals, medicines and medical appliances, Egypt. other chemical and kits were purchased from Sigma (USA).

2.3.Nutraceuticals preparation:

a. Preparation of 4% Taurine:
0.8 gram of taurine was dissolved in 3 ml distilled water and given as oral daily dose for each rat in (B & D groups) for 21 days.

b. Preparation of 30% Galactose:
6 g/kg of galactose was dissolved in 3 ml distilled water and given as oral daily dose for each rat in (C & D groups) for 80 days according to [8] and [13].

2.4. Experimental design:

Seventy male rats were divided into 4 groups placed in individual cages and classified as follows:

1. Group (A): served as a negative control (n=10), left untreated and fed on chow diet and saline.
2. Group (B): considered as positive control group (n=10), rats fed on chow diet & 4% taurine added to the drinking water.
3. Group (C): untreated hyper-galactosemic group (n=25), rats fed on chow diet and 30% galactose for two months & receiving no therapy.
4. Group (D): hypergalactosemic rats treated with taurine (n=25), rats fed on chow diet and 30% galactose and 4% taurine added to the drinking water.

The administration of taurine started after 80 days from the beginning of the experiment. Control animals received physiological saline alone [14].

2.5. Blood sampling:

blood samples were collected after overnight fasting by ocular vein puncture from all rats (control and experimental groups) weekly along the duration of experiment, at 2,4,6,8,10 and 12 weeks from the beginning of the experiment. Directly, after rats were anaesthised using diethyl ether, blood samples were collected from all rats and divided into two parts for determination of the following Biochemical parameters:

- Serum samples**:** to determine SOD, GPx, catalase activities and GSH level galactose, glucose, total cholesterol, HDL, LDL, TG, total protein and insulin [18].
- Edta samples**:** to determine HbA1c [11].

*2.6. Tissue sampling***:**

The second part was used for preparation of tissue (liver) homogenate with 0.9% saline using electrical homogenizer, centrifuged at 3000 r.p.m for 15 minutes, the resulting supernatant were collected and used for estimation of glycogen concentration. Livers from rats were preserved at -20ºC until performing the investigations. [15]

2.7. Statistical analysis:

Statistical analysis was done using SPSS software version 15. The inter-group variation was measured by one way analysis of variance (ANOVA) followed by Post Hoc LSD test. Results were expressed as mean ± SE (standard error). The mean difference is significant at the 0.05 level [16].

3. RESULTS

3.1. Galactose analysis:

The recorded data demonstrated in (Table 1) showed significant increase in serum galactose concentrations in experimental hypergalactosemic rats all over the periods of the experiments.

Treatment with taurine in normal rats significantly decreased serum blood galactose concentration after 21 days of treatment (at the ninth weak). Meanwhile, Treatment with Taurine significantly reduced elevated serum galactose level in experimental hypergalactosemic rats allover the periods of the experiments.

3.2. Glucose analysis:

The recorded data demonstrated in (Table 2 and 3) showed significant increase in

Table (1): Effect of treatment with Taurine on serum galactose in Treated and Untreated Hypergalactosemic Rats (mg/dl).

grups / duration	Group (A)	Group (B)	Group (C)	Group (D)
Two weeks	0.0 ± 0.0^{c}	0.0 ± 0.0^{c}	10 ± 2.0^{a}	5 ± 3.0^{b}
Four weeks	0.0 ± 0.0^{c}	0.0 ± 0.0^{c}	75 ± 21^{a}	50 ± 15^{b}
Six weeks	0.0 ± 0.0^{c}	0.0 ± 0.0^{c}	130 ± 26^{a}	96 ± 22^{b}
Eight weeks	0.0 ± 0.0^{c}	0.0 ± 0.0^{c}	180 ± 23^{a}	129 ± 34^{b}
Ten weeks	0.0 ± 0.0^{c}	0.0 ± 0.0^{c}	195 ± 27^{a}	120 ± 32^{b}
Twelve weeks	0.0 ± 0.0^{c}	0.0 ± 0.0^{c}	275 ± 28^{a}	132 ± 26^{b}

Data are represented as mean ± SE. Mean values with different superscript letters in the same row are significantly different at $P \leq 0.05$.

Table (2): Effect of treatment with Taurine on serum Glucose in Treated and UntreatedHypergalactosemic Rats (mg/dl).

grups / duration	Group (A)	Group (B)	Group (C)	Group (D)
Two weeks	100 ± 20^{b}	97 ± 18^{b}	150 ± 19^{a}	155 ± 21^{a}
Four weeks	93 ± 23^{b}	102 ± 20^{b}	178 ± 21^{a}	180 ± 15^{a}
Six weeks	97 ± 16^{b}	95 ± 15^{b}	190 ± 26^{a}	193 ± 22^{a}
Eight weeks	110 ± 24^{c}	103 ± 10^{c}	215 ± 23^{a}	129 ± 24^{b}
Ten weeks	102 ± 18^{c}	95 ± 19^{c}	235 ± 27^{a}	120 ± 23^{b}
Twelve weeks	104 ± 29^{c}	98 ± 16^{c}	275 ± 28^{a}	122 ± 26^{b}

Data are represented as mean ± SE. Mean values with different superscript letters in the same row are significantly different at $P \leq 0.05$.

Table (3): Effect of Taurine on several physiological and Metabolic Parameters in Rats with Experimentally hypergalactosemic rats.

Groups	Group (A)	Group (B)	Group (C)	Group (D)
Survived animals,(%)	100	100	70	80
Bodyweight,(g)	200 ± 15	200 ± 15	175 ± 10	189 ± 18
HbA1C , (%)	2.0 ± 0.1^{a}	1.2 ± 0.2^{b}	3.8 ± 0.1^{a}	1.9 ± 0.35^{b}
Fructosamine, (ml/dl)	40.58 ± 0.32^{b}	24.12 ± 0.12^{c}	128.8 ± 1.03^{a}	17.76 ± 0.34^{c}

Data are represented as mean ± SE. Mean values with different superscript letters in the same row are significantly different at $P \leq 0.05$.

Table (4) :Effect of treatment with Taurine on serum cholesterol, TG, HDL, LDL, Insulin, Glycogen in Liver and Total protein concentrations in treated and untreated hypergalactosemic Rats.

Groups / parameters	Group (A)	Group (B)	Group (C)	Group (D)
Cholesterol (mg/dl)	77 ± 3.33^{a}	76 ± 6.53^{a}	89.3 ± 4.23^{a}	81.6 ± 3.13^{a}
Triglyceride (mg/dl)	124.4 ± 11.86^{a}	110.74 ± 9.6^{a}	133 ± 6.91^{a}	87.6 ± 9.36^{b}
HDL (mg/dl)	36.8 ± 5.49^{a}	34.33 ± 4.46^{a}	25.2 ± 4.15^{a}	35.5 ± 2.9^{a}
LDL (mg/dl)	15.6 ± 2.59^{b}	12.4 ± 2.82^{b}	25.5 ± 1.31^{a}	19.3 ± 1.2^{b}
Insulin (ng/l)	14.07 ± 0.65^{c}	22.24 ± 0.61^{a}	12.37 ± 0.35^{d}	19.63 ± 0.58^{b}
Total Protein(g/dl)	$6.84 \pm 0.44^{b,c}$	9.26 ± 0.27^{a}	5.84 ± 0.39^{c}	7.84 ± 0.33^{b}
Glycogen in Liver (µg/mg)	16.73 ± 0.98^{b}	19.66 ± 0.74^{a}	12.10 ± 0.28^{c}	16.47 ± 0.35^{b}

Data are represented as mean ± SE. Mean values with different superscript letters in the same row are significantly different at $P \leq 0.05$.

Table (5): Effect of Taurine on Catalase, Superoxide Dismutase, Glutathione Peroxidase activities and Reduced Glutathione content in treated and untreated hypergalactosemic rat.

Groups / parameters	Group (A)	Group (B)	Group (C)	Group (D)
Catalase (u/l)	0.78 ± 0.03^{b}	1.02 ± 1.81^{a}	0.40 ± 0.74^{c}	0.87 ± 3.52^{b}
(SOD) (u/l)	4.18 ± 0.69^{b}	5.09 ± 0.54^{a}	2.83 ± 0.47^{c}	4.03 ± 0.78^{b}
(GSH) (ml/dl)	145.18 ± 7.16^{b}	157.39 ± 4.21^{a}	124.13 ± 5.14^{c}	141.88 ± 0.21^{b}
(GSH-Px) (CRG/min / ml)	0.93 ± 0.39^{b}	1.63 ± 0.94^{a}	0.33 ± 0.51^{c}	0.96 ± 1.29^{a}

Data are represented as mean ± SE. Mean values with different superscript letters in the same row are significantly different at $P \leq 0.05$.

serum glucose concentrations, glycated hemoglobin HbA1c and fructosamine in experimental hypergalactosemic rats.

Treatment with taurine in normal rats significantly decreased serum blood glucose concentration after 21 days of treatment. Meanwhile, treatment with taurine significantly reduced elevated serum glucose level in experimental hypergalactosemic rats all over the periods of the experiments.

3.3. Lipid profile:

In our study, we found that hypergalactosemia in the short term (3 months) were obtained results in (Tables 3) which revealed that non-significant increase in serum total cholesterol, and triacylglycerols TG concentrations. While there was a significant increase in LDL concentration. In addition, our results revealed a non-significant decrease in HDL concentration in experimentally hypergalactosemic rats.

Treatment with taurine in experimental hypergalactosemic rats non-significantly reduced serum LDL, total cholesterol and significantly reduced serum TG concentration. In addition, treatment with

taurine non-significantly increased HDL concentration in rats after 21 days of drug administration.

3.4. Antioxidants parameters:

The presented data in table (4) revealed a significant decrease in GSH level, GSH-px, catalase and SOD activities in hypergalactosemic group when compared to control group. Treatment with taurine in normal rats and in experimental hypergalactosemic rats significantly increases GSH content, GSH-px, CAT. And SOD activities after 21 days of drug administration.

4. DISCUSSION

Concerning galactose level in hypergalactosemic rats the recorded data demonstrated in (Table 1) showed a significant increase in serum galactose concentrations in experimental hypergalactosemic rats all over the periods of the experiments. These results were nearly similar to those reported by Ronald and Timothy [13] who observed that, in animals offered the galactose-rich diet, blood galactose concentrations were found to vary from hour to hour throughout the day, from values near 0 after an overnight fast, up to values of about 150-250 mg/dl later in the day after the galactose-rich diet had been eaten.

To clarify the relationship between glucose utilization and hyperglycemia because of galactose feeding, this study showed a significant increase in serum glucose concentrations, glycated hemoglobin HbAic and fructosamine in experimental hypergalactosemic rats allover the periods of the experiments. These results were in agreement with Ramana *et al.,* [8] who reported that, Blood glucose levels in hypergalactosemic rats were significantly higher than in control rats (p<0.01). Moreover, Mary Otsyula *et al*., [17] who reported that, Galactose-fed animals have elevated levels of blood hexose and as expected, 3 months of galactose feeding resulted in significant elevations in glycated hemoglobin, and cessation of experimental galactosemia had a marginal, but statistically significant, effect on glycated hemoglobin values. Furthermore, Peter F. Kador *et al.,* [18] who reported that, the levels of Hb A1C increased in dogs fed a galactose diet and decreased in the control dogs. Experimental galactosemia, which activates the polyol pathway, has been used extensively to explore the pathogenesis of diabetic complications. The galactose-fed dog is an animal model that both histologically and clinically demonstrates retinal vascular changes associated with diabetic retinopathy.

Treatment with taurine in normal rats significantly decreased serum blood glucose concentration after 21 days of treatment. Meanwhile, treatment with taurine significantly reduced elevated serum glucose level in experimental hypergalactosemic rats all over the periods of the experiments. These results are nearly similar to those reported by Katarzyna *et al.,* [12] who showed that, three weeks of taurine administration to diabetic rabbits resulted in 30% decrease in serum glucose level and the normalization of diabetes-elevated rate of renal gluconeogenesis.

On contrary to diabetes, which believed to cause vascular dysfunction via a common biochemical mechanism, Galactose-fed animals have elevated levels of blood hexose, but do not develop other sequelae of insulin deficiency, such as alterations in lipids and protein metabolism [17].

In our study, we had found that hypergalactosemia (Tables 3) in the short term (3 months) revealed that, a non-significant increase in serum total cholesterol, LDL and triacylglycerols TG concentrations. In addition, our results revealed a non-significant decrease in HDL concentration in experimentally hypergalactosemic rats.

Treatment with taurine in experimental hypergalactosemic rats non-significantly reduced serum LDL, total cholesterol and significantly reduced serum TG concentration. In addition, treatment with taurine non-significantly increased HDL concentration in rats after 21 days of drug administration. These results are nearly similar to those reported by John and Julius [19] reported that, administration of taurine to diabetic rats decrease the concentration of serum total cholesterol, TG and LDL. Also, Sun *et al.,* [20] who reported that, Serum total cholesterol (TC) levels of the taurine group were significantly reduced compared to those of the other groups. Therefore, these results suggest a possible effect of PTP1B inhibitors and taurine on blood total cholesterol in the obese adolescent. Also, Tawfek and Taha [21] reported that, Diabetic rats supplemented with antioxidants (taurine) showed decrease in serum total lipid. Also, Choi [22] reported that, The purpose of this study was to investigate the effect of dietary taurine supplementation on plasma and liver lipid content in ovariectomized (OVX) rats. The concentrations of plasma total cholesterol, LDL and triglycerides were lower in the taurine treated groups. In addition, Mi-Ja [23] reported that, the plasma concentration of HDL cholesterol (HDL-C) significantly increased while total cholesterol, TG and LDL showed significantly decreased in the rats fed TSD (taurine diet) compared to those fed control diet.

Also, treatment with taurine in experimental hypergalactosemic rats significantly decreased glycogen concentration in liver and significantly increased insulin concentration. These results were nearly similar to Nagakatsu [24] who reported that, the treatment of otsuka long-evans takushima fatty (OLETF) rats with taurine increased muscle glycogen content in the OLETF rats. Also, Nandhini *et al.,* [25] reported that, Taurine administration improved insulin sensitivity and controlled hyperglycemia and hyperinsulinemia in fructose-fed rats. Taurine treatment also restored the glucose metabolizing enzyme activities in fructose-fed rats.

Regarding to antioxidants the presented data in tables (4) revealed a significant decrease in GSH level, GSH-px, catalase and SOD activity in hypergalactosemic group when compared to control group.

These results were in agreement with Mary *et al.,* [9] who reported that, the effect of 60 days of streptozotocin-induced diabetes and 50% galactose diet is a decrease in the enzymatic activities of catalase (cat) in liver, kidney, and heart in the hypergalactosemic rats. Also, (Robert *et al.,* [26] reported that, the Effects of 30 days of streptozotocin-induced diabetes, insulin-treated diabetes, and 50% galactose diet on enzyme activities in liver, kidney, and heart is Hepatic catalase activity levels in both diabetic and galactosemic rats were significantly decreased when compared to normal. Hepatic and renal levels of GSSG were significantly diminished compared to normal in both diabetic and galactosemic rats.

Treatment with Taurine in normal rats and in experimental hypergalactosemic rats significantly increases GSH content, GSH-px, CAT. In addition, SOD activities after 21 days of drug administration. These results were nearly similar to those reported by Yu and Kim [27] they reported that, Taurine increased the activities of superoxide dismutase, glutathione peroxidase and Catalase compared to those of the control group. Likewise, Henry *et al.,* [11] reported that, taurine is a unique antioxidant and has beneficial effects on glutathione redox state metabolism. In addition, taurine-induced increase in the activities of catalase and the enzymes of glutathione metabolism was of importance for antioxidative action of this amino acid.

It can be concluded that, taurine plays an important role in experimental hypergalactosemia, because it shows

preventive action against its secondary complications due to hypoglycaemic, antioxidative and Hypolipidemic action also, its important effect on insulin.

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التأثير الكيميائي الحيوي للتورين على الفئران المحدث فيها زيادة جالاكتوز الدم تجريبيا

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[1]قسم الكيمياء الحيوية والإكلينيكية-كلية الطب البيطري بمشتهر-جامعة بنها، [2]قسم الكيمياء الحيوية-كلية العلوم -جامعة الزقازيق

الملخص العربي

في الآونة الأخيرة كان هناك طلب كبير على المنتجات الطبيعية التي لديها تأثيرات وقائية محتملة ضد مرض السكري ومضاعفاته الثانوية. وفي الابحاث الحالية للسكري تم استخدام الفئران المحدث فيها زيادة جالاكتوز الدم تجريبيا كنموذج للمضاعفات المرضية للنوع الاول من مرض السكري. بأخذ ذلك في الاعتبار، أجريت هذه الدراسة لمعرفة تأثير التورين، في علامات الاكسدة ومستويات السكر والدهون في الدم في الفئران المحدث فيها زيادة جالاكتوز الدم تجريبيا. أجريت هذه الدراسة على عدد 70 فطيم من ذكورالفئران البيضاء والتي تزن من 175–200 جم، تم تقسيم الفئران إلى 4 مجموعات كما يلي: المجموعة A: مجموعة ضابطة سليمة تم تجريعها بالمحلول الملحي. المجموعة B: مجموعة ضابطة سليمه تم تجريعها ب 4% تورين (0.04g/kg/day) لمده 21 يوم. المجموعة C: مجموعة محدث فيها زياده جالاكتوز الدم عن طريق تجريعها ب 30% جالاكتوز (0.5g/kg/day) لمدة80 يوم . المجموعة D: مجموعة محدث فيها زياده جالاكتوز الدم وتم تجريعها ب 4% تورين لمده 21 يوم. تم قياس مستويات السكر (الجالاكتوز والجلوكوز) ووزن الجسم أسبوعيا لمتابعة زيادة سكر الدم الناجم عن التغذية بالجالاكتوز. كما تم تجميع عينات الدم لقياس بعض الدلالات الحيوية بها مثل قياس مستوي الجلوتاثيون المختزل (GSH) وفاعلية كل من إنزيم الجلوتاثيون بيروكسيديز (GSH–px)، السوبر اوكسيد ديسميونيز (SOD) والكاتلاز ((CAT. وأيضا تم قياس كلا من الانسولين، ونسبةHbA1c ، فركتوزامين، الجليكوجين في الكبد، الدهون، والبروتين الكلي. وأوضحت النتائج زيادة معنويه في مستوي الجالاكتوز والجلوكوز وزيادة غير معنوية في مستوي الدهون ونقص معنوي في مستوي الانسولين والجليكوجين في الكبد وأيضا نقص معنوي في فاعليه كلا من إنزيم الكاتلاز والسوبر اوكسيد ديسميوتيز والجلوتاثيون بيرواكسيديز وفي مستوي الجلوتاثيون المختزل في الفئران المحدث فيها زيادة جالاكتوز الدم بالمقارنة بالمجموعة الضابطة وحدث تغير في هذه النتائج إلى الأفضل بعد التجريع بالتورين. لذا نوصى بتناول التورين كعامل وقائي أو كعلاج مساعد في علاج مرض السكري ومضاعفاته.

(مجلة بنها للعلوم الطبية البيطرية: عدد 24 (2)، يونيو 2013: 1–10)