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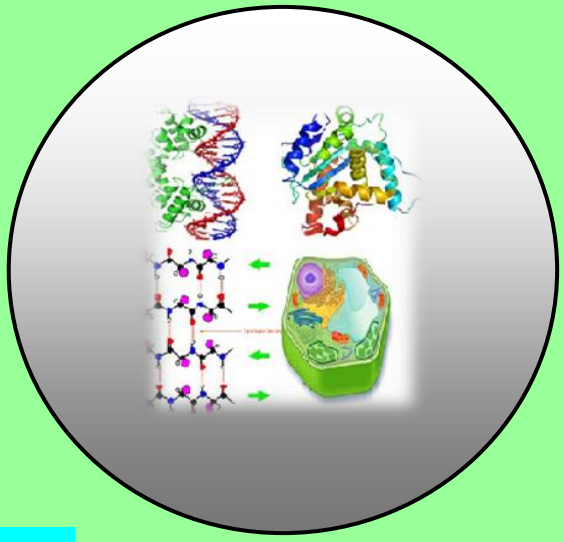
ISSN 0970-4973 Print
ISSN 2319-3077 Online/Electronic

Journal Impact Factor: 4.275
Global Impact factor of Journal: 0.876
Scientific Journals Impact Factor: 3.285
InfoBase Impact Factor: 3.66
Index Copernicus International Value
IC Value of Journal 47.86Poland, Europe

J. Biol. Chem. Research
Volume 33 (1) 2016 Pages No. 327-343



Journal of Biological and Chemical Research

An International Peer Reviewed / Refereed Journal of Life Sciences and Chemistry

Indexed, Abstracted and Cited in various International and National Scientific Databases

Published by Society for Advancement of Sciences®

J. Biol. Chem. Research. Vol. 33, No. 1: 327-343, 2016
(An International Peer Reviewed / Refereed Journal of Life Sciences and Chemistry)
Ms 33/1/71/2016

ISSN 0970-4973 (Print)
ISSN 2319-3077 (Online/Electronic)s

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http:// www.jbcr.in
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RESEARCH PAPER
Received: 17/01/2016 Revised: 07/03/2016 Accepted: 10/03/2016

Alterations in Weight and Protein Profile, Associated with the Administration of Glibenclamide or*Chrozophora tinctoria* Extraction in Alloxan-Induced Diabetic Rats

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ABSTRACT

The aim of thisstudy is to compare the therapeutic role of sulfonylurea drug "glibenclamide" with the extract of Chrozophora tinctoria leaves in alloxan-induced diabetic rats. Male Rattusnorvegicus albino ratsweighing between 80-90gm were intraperitoneal injectedby a single dose of 150 mg/kg body weight (b.wt.) alloxan. Diabetic animals exhibited hyperglycemia. There was significant decrease in body weight compared to control animals. Urea concentrations and uric acid were also elevated markedly compared to controls. In contrast, total protein and albumin concentrations showed significant decrease compared to controls. Treatment of alloxan-induced diabetic rats with glibenclamide or Chrozophora tinctoria extract ameliorates all of the previously mentioned parameters. However, glibenclamide therapy was more efficient.Theresults of the current study demonstrated the antidiabetic and hypoglycaemic effects of Chrozophoratinctoria extract and underscore its potentials in the management of diabetes mellitus.

Key words:Chrozophora tinctoria, Alloxan, Glibenclamide, Diabetes and Rats.

INTRODUCTION

Diabetes Mellitus (DM), a metabolic issue around the world, is described by hyperglycemia connected with weakness in insulin secretion and/or insulin activity and also adjustment in go-between digestion system of starch, protein and lipids Punitha and

Manoharan,(2006).DM is the third driving reason for death in numerous nations after cardiovascular diseases and cancerArinc, et al,(2007).High blood glucose levels are poisonous, bringing on genuine microvascular and macrovascular harms. In this way, diabetes prompts visual impairment, end-stage renal infection, atherosclerotic macrovascular ailment, and a variety of weakening neuropathies, patient's quality of life and life expectancy Kelly, et al, (2003).DM, also, may serve as a disease modifier of Apical periodontitis Marotta, et al,. (2012).

The liver is the essential site of endogenous glucose creation with a minor commitment from the kidney either from gluconeogenesis or by means of glycogenolysisMeyer, et al,. (2004). It is essentially an issue of sugar digestion system, yet with the movement of the ailment, protein digestion system is additionally influenced. Gluconeogenesis, a noteworthy biochemical procedure that creates glucose from protein, is quickened in diabetes mellitusKhan and Safdar, (2003). There are increases in both protein breakdown and protein synthesis during insulin deprivation. But the magnitude of the increase in protein breakdown is greater than that of the increase in protein synthesis, leading to a net protein loss during insulin deprivationCharlton, and Nair,(1998). Yet, particular metabolic renal adjustments are evident in experimental diabetes, prompting a negative nitrogen balance, upgraded proteolysis and brought down protein union Bhavapriya, et al,. (2001).

A large portion of type II DM patients need medication.Nowadays, a variety of agents with anti-diabetic action are available. Sulfonylureas(one of these agents), as glibenclamide, have been the main oral antihyperglycemic specialists utilized as a part of treating type II DM patients over the past half-century. It keeps on being the most famous antidiabetic medication endorsed today Misaki, et al,. (2007). Glibenclamide are long acting and have metabolites that are excreted renally Grail, and Wolffenbuttel,(1999).The sulfonylureas are insulin secretagogues that act to increase insulin secretion. Most of Studies done on sulfonylureas revealed that the antidiabetic sulfonylureas could accelerate (or at least do not protect against) progressive β-cell failure. It, also are similar to (or worse than) insulin in obtaining good metabolic control. Therefore, sulfonylureas should not be used as first-line therapy in DM patients Hanefeld, (2007).

Managing diabetes with no symptoms remains a challenge to the medicinal framework. This prompts expanding the interest for supplementary and alternative medication of differing modes of action to improve glycemic control with less symptoms. There is a developing enthusiasm for home grown cures due to their adequacy, negligible symptoms and generally low expenses. The plant kingdom with enormous chemical diversity may be looked as an important source of new chemical substances with effective control in different ailments Gautam, et al,. (2007). Thus, Traditional Medicine might be an alternative treatment of DMTian, et al, (2013).

Natural medications or their extracts are recommended generally, not with standing when their organic dynamic mixes are obscure Gupta, et al,. (2009).

Products such as hypoglycemic operators, that are natural, usually have more potential advantages in treatment of diabetes and its consequences are not as much as synthetic compounds Xie, et al,. (2005). For example, fenugreek, onion, lupine and garlic are some of customary plants that have a hypoglycemic impact Shabana, et al. (1990) and Sharma, et al,. (1990).

Ethnomedicinal plants, i.e. *Chrozophora tinctoria*, contain chemicals, Glycosides and flavonoids, with a variable medicinal effects Hashim, et al, (1990) and Abdallah, et al, (2015). However, *Chrozophora tinctoria* leaves extract are generally utilized as a part of the Palestinian popular Medicine as hypoglycemic specially in type II diabetes. The greater partof plants discovered contain substances like glycosides, alkaloids, terpenoids, flavonoids and so forth., that are as often as possible embroiled as having anti-diabetic impacts Loew, and Kaszkin, (2002). Differences in gender responses to therapy should be considered when individualizing treatment for people with type IIDM McGill, et al,. (2013).

The present study aims to compare the therapeutic role of sulfonylurea drug "glibenclamide" with the extract of *Chrozophora tinctoria* leaves in alloxan-induced diabetic rats.

MATERIAL AND METHODS

Male *Rattusnorvegicus* albino rats, weighing 80-90gm, were obtained from the breeding unit of Biology Department, Faculty of Applied Sciences, Al-Aqsa University, Gaza, Palestine. Animals were left for one week before experimentation to adapt to laboratory conditionsat a constant temperature of 25±2°C and 45-55% humidity, with 12 h light dark cycle.Animals had ad libitum access to food and water.Animals were fasted overnight and then treated by single intraperitoneal (ip) injection of a freshly prepared solution of alloxan monohydrated in a dose of 150 mg/kg b.wt. Barbosa, et al,. (2008). Alloxan monohydrated was purchased from Sigma-Aldrich, Egypt.

Glucose concentration in rats blood were measured after 48 hrs.' of injection. Only rats with fasting blood glucose levels greater than 200 mg/dL were considered diabetic and then employed in the study Kar, et al,. (2006).

Fresh leaves of *Chrozophora tinctoria* (*Ch.t*) plant were collected from Derelbalah area, Gaza strip, Palestine. The leaves were separated and dried under shade for about a week. Then leaves were crushed to moderately coarse powder and kept at a dark and dry place.

Two hundred grams of the fine powder were boiled in about 1 Liter of distilled water (100°C) up to 20 min. The obtained dark brown extract was cooled at room temp. The cold decoction was filtered with a cheese cloth to obtain clear solution. Then the solution was evaporated to dryness in a water bath at 55°C. The deep brown solid residue was stored in a refrigerator at 4°C until further use Adeneye, et., al (2007). When needed; the residue was suspended in distilled water and used in the study. However, Glibenclamide (Glucocare) tablets produced by pharmacare Co., Betonia, Palestine, were purchased from local pharmacies.The tablets were grinded using a mortar. The powder was dissolved in distilled water and orally administrated at a dose of 36 mg/kg b.wt. /day Ashour, et al., (2004).

A pilot experiment was conducted to select the suitable concentration of *Ch.t*extract. Thirty rats were divided randomly into 6 groups, 5 rats of each. The groups got different dose of freshly prepared plants leave extracts (150 mg, 200 mg, 250 mg, 300 mg, 350 mg and 400 mg/kg b.wt. /day). Rats were orally administrated with *Ch.t.* extract using special stomach tube with a smooth tip to protect the interior lining of the oral and buccal cavity from injury. However, Normal animals of similar initial weight were used as normal control and orally administrated with saline solution. Blood glucose levels were weakly determined electronically in rats of those groups using Glucometer, USA.

After two weeks there was a pronounced decrease in fasting sugar in the blood of rats administered doses ≥ 250 mg/kg b.wt./day. But, there was no significant differences between blood sugar decrements resulted from the doses of 250, 300, 350 and 400 mg/kg b.wt.. So the dose of 250 mg/kg b.wt./day was considered the suitable dose for this study.

Experimental Protocol

After acclimatization period, rats were divided into four groups comprising 48 animals in each group as follow:

Group I, Rats were untreated and served as normal control and were orally administrated with saline solution. Group II, diabetic rats remained without treatment (-ve control) "diabetic control". Group III, diabetic rats were orally administered with glibenclamide at a dose of 36 mg/kg b.wt. /day Ashour, et al., (2004).Group IV, diabetic rats were orally administered with 250mg /kg b.wt. /day *Ch.t.* extract.

Total body weights were recorded weekly for determination of growth rates all over the experimental duration. On the other hand, Animals from both experimental and control groups were decapitated at weekly intervals starting from the first week. Clear serum samples were separated by centrifugation at 3000 r.p.m. for 20 min. and then transferred into Eppendorf tubes and stored in a deep freeze (-20^{o}C) for biochemical analysis. However determination of glucose was carried out on fresh serum samples.

Determination of Biochemical parameter

Serum glucose, total protein, albumin, globulin, urea, creatinine and uric acid were analyzed by Kone lab 60 Auto analyzer in Al Shifa Hospital Clinical Chemistry Laboratory.

Serum Glucose was determined by glucose oxidase (GOD) Trinder, (1969) using Lab Kit kit, Spain. Serum total protein was determined by Biuret method using DiaSys kit spainJohnson, et al., (1999). On the other hand, Bio Systems kits, Spain were used in determinations of proteins, urea, Creatinine and uric acid.Serum Albumin was determined by Bromocresol green Doumas, et al., (1971). The concentration of globulins were calculated using the equation: Concentration of globulins (mg/dl) = Total protein –Albumin. Serum urea was determined by urease - glutamate dehydrogenase (GDH) Gutmann and Bergmeyer (1974). Serum Creatinine was determined by Alkaline Picrate method Fabiny and Ertingshausen (1971). Serum Uric Acid was determined by Uricase /POD method Fossati, et al., (1980).

Statistical Analysis

Statistical analysis of The obtained data was performed by one-way analysis of variance using SPSS version 11.0 for windows (Statistical Package from the Social Sciences Inc., Chicago, IL, USA). The data were subjected to Duncan's multiple range test to verify the differences between the means; $p < 0.05$ was considered as significant. Percentage change was also calculated. Data were expressed as Mean ± SE .

RESULTS

Table (1) showed that, ip injection of a single dose of alloxan (150 mg/kg b.wt.), caused a high significant decrease ($p<0.001$) in the body growth rate compared to control levels.

Treatment of diabetic animals with glibenclamide (36mg/kg b.wt./day) improved the body growth rate compared to the (-ve control) group. The body growth rate also recorded an improvement in *Ch.t* extract- treated rats compared to diabetic control. *Ch.t* extract therapy (250mg/kg b.wt./day) also amended the body growth rate, but to low extent compared to glibenclamide.

The data in table (2) indicated that, intraperitoneal injection of a signal dose of alloxan caused highly significant increase ($p<0.001$) in the serum glucosecompared to control levels. Treatment of diabetic animals with glibenclamide (36 mg/kg b.wt./day) improved glucose level compared to alloxan (-ve control) group. Serum glucose also recorded an improvement in *Ch.t* extract- treated rats compared to alloxan (-ve control). The change is significant at 1st week of the treatment ($p<0.001$) up to the end of the studiedperiods. *Ch.t*extract therapy (250mg/kg b.wt./day) also improves serum glucose, but to a lower extent compared to glibenclamide.

It's clear from the results depicted in Table (3) that alloxan caused a significant reduction of total protein in the studied weeks compared to normal controls ($P<0.001$). Treatment with glibenclamide and *Ch.t* extract caused a significant increase in total protein throughout the experimental period compared to alloxan (-ve control) group, but total protein still lower than those of the normal controls.

The findings mentioned in Table (4) showed a general highly significant decrease ($p<0.001$) in serum albuminof alloxan- diabetic rats compared to control levels. Oral administration of glibenclamide as well as *Ch.t* extract increased levels of serum albumin in alloxan- diabetic rats compared to –ve control. The presented data in Table (5) revealed that alloxan caused a significant reduction of serum globulin in all eight weeks compared to normal controls. Treatment with glibenclamide and *Ch.t* extract caused a gradual and significant increase in serum globulin throughout the experimental period compared to alloxan (-ve control) group, however, serum globulin did not reach those of the normal control.

Data in Table (6) showed that, ip injection of a single dose of alloxan generally provoked highly significant elevation ($p<0.001$) in serum urea concentration, compared to control values. Treatment of diabetic animals with gliben clamide and *Ch.t* extract lowered serum urea values compared to alloxan (-ve control) group.

Upon intraperitoneal injection of single dose of alloxan, creatinine control levels were increased gradually all over the experimental duration of 8 weeks. This increase becomes significant ($p<0.001$) at the 2nd week up to 8th weeks. Glibenclamide treatment lowered serum creatinine in diabetic rats compared to alloxan (-ve control) group values during the 8 weeks of the experiment, respectively . *Ch.t* extract administration also lowered creatinine levels of diabetic rats from the 3rd week compared to alloxan (-ve control) group values. (Table7). In alloxan- induced diabetic animals there were gradual decreases in uric acid concentration (table 8). Treatment of diabetic animals with glibenclamide returned uric acid concentration to near control levels. Similar, but less effective, action was observed in diabetic animals treated with *Ch.t* extract.

DISCUSSION

Diabetes is a chronic disease that requires persistent restorative consideration and patient self-management training to counteract intense complexities and to decrease the danger of long-term inconveniences American Diabetes Association. (2008).

The present study shows that diabetic group instigates a decline in body development rate during the trial period investigated when contrasted with control group. The decrease in b.wt. in diabetic rats could be attributed to the lack of hydration and catabolism of fats and proteins. Such results agreed with the studies of Subash, et al., (2007) and Gong, et al., (2009).

On the other hand, in group III, an increase in b.wt.was noticed that makes them close to the typical control group. The increase in b.wt. of glibenclamide treated rats may be because of expansion of insulin secretion or expanding nourishment utilization. Additionally, oral administration of *Ch.t*. extract for 8 weeks to diabetic rats expanded their sustenance utilization and enhanced their b.wt. This could be because of a superior control of the hyperglycemic state in the diabetic rats, yet this change is not as much as that seen with glibenclamide treatment. Not surprisingly, the present study uncovered that the greater part of rats created diabetes within 24 hours of injection of alloxan. The glucose level expanded altogether and systematically from the first week up to eighth week of the tested period. This increase is thought to be due to absence of insulin, expanded gluconeogenesis, and glycogenolysis. This is consistent with previous results obtained by Roy, et al.,(2005) and Shabeer, et al., (2009).

Treatment of diabetic rats with glibenclamide incited a vigorous number hypoglycemic activity. It was apparent from the findings that *Ch.t* extract decreased the fasting blood glucose (FBG) level in diabetic rats. The antihyperglycaemic impact of *Ch.t.* extract should be connected to more than one system. The conceivable instrument incorporates the incitement of β-cells and consequent arrival of insulin and actuation of the insulin receptor. The plant's antihyperglyacemic activity may be because of the potentiation of pancreatic secretion of insulin, which was confirmed by the expanded level of insulin in diabetic rats treated with *Ch.t.* extract alone.

The current data demonstrate a decrease in serum total protein in the diabetic rats which does not contradict with a previous study Yassin, et al., (2004). This decrease may be attributed to the repressed oxidative phosphorylation processes that prompt lessening of protein synthesis, increase in the catabolic procedures and decrease of protein absorption. The productivity of glibenclamide to restore all out protein fixations is possible because of its capacity to build insulin discharge Fern strom, and Fernstrom, (1993), Farouque, and Meredith, (2003).

In this study, serum total protein and albumin levels have expanded after treatment by Ch.t. extract. This demonstrates that the metabolism of proteins was evidently influenced by *Ch.t* extract. The change on the levels of protein and albumin in the diabetic treated groups demonstrate that *Ch.t* concentrate have noteworthy impact in glucose and protein level because insulin hinder gluconeogenesis from protein or could be caused by a change in renal capacity Tenpe, and Yeole, (2009). This affirmative concurs with the way that the heaviness of skeletal muscles significantly increased in diabetic rats treated with Ch.t. extract. Liver huge necrosis, deterioration of liver capacity, hepatic resistance to insulin and glycogen impedance of oxidative phosphorylation may be the reasons of the reduction in albumin level archived for diabetic rats Rao, (1995). Under the same experimental conditions serum globulin level uncovered a considerable decrease Wanke,. and Wong, (1991). The renal pathogenesis is associated with the length of diabetes in terms of time. The most devastating consequence with diabetes is nephropathy Lengyel, et al., (2003).

The current data demonstrated a profoundly noteworthy increase in serum urea concentration in diabetic group. This finding agrees with another study that showed that serum levels of each of the three tried renal markers urea, creatinine and uric corrosive altogether increased. Jarald, et al., (2009). Such findings showed that diabetes might lead to renal dysfunction Bangar, et al., (2009).

Depletion of serum protein, increase in the rate of circulating amino acids, and deamination might cause this increase in serum urea concentration in diabetic group. Deamination of amino acids, consequently, leads to the accumulation of large amount of ammonia that is ultimately transformed to urea. The decrease in uric acid concentration in the diabetic rats of the current study was only justified during the latter weeks of the present study. Furthermore, the results showed that diabetic rats initiated progressive increase in creatinine concentration that commenced from the first week onwards.

Table 1. Effects of glibenclamide and *ch.t* extract administration on body weight (g) of control and alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	85.66±1.68	78.33±0.91 -8.55 < 0.01 [a]	81.5±1.05 4.04 >0.05 [b]	78.8±0.87 0.60 >0.05 [b]
2nd	Mean ± S.E % of change P	117.0±1.4	94.8±1.16 -18.97 <0.001[a]	108.83±1.3 14.08 <0.001[b]	103.8±0.98 9.49 <0.001[b]
3rd	Mean ± S.E % of change P	144.66±1.3	106.83±0.79 -26.16 <0.001[a]	129.33±1.45 21.06 <0.001 [b]	117.17±1.22 9.67 <0.001[b]
4th	Mean ± S.E % of change P	177.83±1.77	113.17±0.94 -36.36 <0.001[a]	150.5±1.60 33.00 <0.001[b]	130.0±1.52 14.87 <0.001[b]
5th	Mean ± S.E % of change P	197.00±3.75	122.33±1.2 -37.9 <0.001[a]	167.5±1.47 36.92 <0.001[b]	145.0±1.29 18.50 <0.001[b]
6th	Mean ± S.E % of change P	220.17±3.06	131.5±0.99 -40.27 <0.001[a]	180.8±1.35 37.94 <0.001[b]	156.8±1.66 19.20 <0.001[b]
7th	Mean ± S.E % of change P	242.17±1.30	141.83±2.1 -41.4 <0.001[a]	193.0±1.93 36.07 <0.001[b]	163.0±1.48 14.92 <0.001[b]
8th	Mean ± S.E % of change P	256.13±1.2	147.83±1.2 -42.28 <0.001[a]	203.66±2.3 37.76 <0.001[b]	170.0±1.15 15.00 <0.001[b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

This increase was approximately reached to near to normal levels in response to the treatment with glibenclamide. Additionally, diabetic group receiving *Ch.t* extract reflected rapid normalization of blood kidney function test levels in comparison with zero time.

Decrease of urea level clearly indicated reduction in proteolysis and this might be the reason for the increase in the b.wt. of the animals Oliveira, et al., (2008).
To sum up, it is clearly seen from the present study that *Ch.t* extract has useful effects on blood glucose level as well as improvement of kidney function.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 2. Effects of glibenclamide and *ch. T* extract administration on blood glucose of control and alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	90 ± 1.39	330.33 ± 1.80 267.03 <0.001[a]	234.83 ±1.80 -28.91 <0.001[b]	245.17 ± 1.17 -25.78 <0.001[b]
2nd	Mean ± S.E % of change P	93.00 ± 1.46	340 ± 2.82 265.59 <0.001[a]	215.33 ±1.20 -36.76 <0.01[b]	225.00 ± 1.06 -33.82 <0.001[b]
3rd	Mean ± S.E % of change P	91.67 ±2.15	361.17 ± 4.10 293.99 <0.001[a]	195.33 ±1.87 -45.91 <0.001[b]	214.83 ± 1.33 -40.52 <0.001[b]
4th	Mean ± S.E % of change P	86.83 ±0.95	378.33 ± 4.57 335.71 <0.001[a]	202.67 ±1.63 -46.43 <0.001[b]	200.67± 1.20 -46.45 <0.001[b]
5th	Mean ± S.E % of change P	94.33 ±1.12	380.00 ± 3.49 302.84 <0.001[a]	169.83 ± 1.4 -55.30 <0.001[b]	184.50 ± 1.14 -51.45 <0.001[b]
6th	Mean ± S.E % of change P	96.00 ± 1.55	350.33 ± 2.85 264.93 <0.001[a]	166.17 ±5.69 -52.56 <0.001[b]	170.00 ± 1.94 -51.45 <0.001[b]
7th	Mean ± S.E % of change P	96.67 ±1.33	391.67 ± 2.46 305.16 <0.001[a]	140.33 ±1.33 -64.19 <0.001[b]	158.83 ± 1.22 -59.45 <0.001[b]
8th	Mean ± S.E % of change P	96.00 ± 1.51	410.83 ± 3.03 327.95 <0.001[a]	144.67 ±1.28 -64.78 <0.001[b]	161.66 ± 1.28 -60.65 <0.001[b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 3. Effects of glibenclamide and *Ch.t. extract* administration on serum T. protein of control and alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	6.87±0.06	5.2± 0.08 -24.31 <0.001[a]	5.7±0.07 9.62 <0.001[b]	5.51±0.07 6.0 <0.05 [b]
2nd	Mean ± S.E % of change P	6.7±0.10	5.3±0.10 -20.8 <0.001[a]	6.20±0.14 17.00 <0.001[b]	5.86±0.18 10.57 <0.001[b]
3rd	Mean ± S.E % of change P	6.86±0.18	5.40±0.12 -21.28 <0.001[a]	6.73±0.67 24.62 >0.05[b]	5.98±0.12 10.74 <0.01[b]
4th	Mean ± S.E % of change P	6.75±0.14	5.0±0.14 -25.92 <0.001[a]	6.52±0.17 30.40 <0.001[b]	6.26±0.14 25.2 <0.001[b]
5th	Mean ± S.E % of change P	6.75±0.14	5.34±0.18 -20.88 <0.001[a]	6.56±0.13 22.85 0.001[b]	6.43±0.16 22.84 <0.001[b]
6th	Mean ± S.E % of change P	6.75±0.11	5.22±0.10 -22.68 <0.001[a]	6.56±0.11 25.77 <0.001 [b]	6.52±0.10 24.90 <0.001 [b]
7th	Mean ± S.E % of change P	6.71±0.12	5.34±0.18 -20.41 <0.001[a]	6.69±0.14 25.28 <0.001 [b]	6.49±0.15 21.53 <0.001 [b]
8th	Mean ± S.E % of change P	6.71±0.62	5.34±0.18 -20.4 <0.001[a]	6.62±0.19 23.97 <0.001 [b]	6.31±0.10 18.16 <0.001[b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant. [a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 4. Effects of glibenclamide and *Ch.t. extract* administration on serum albumin of control and alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	3.96±0.97	2.88±0.54 -27.27 <0.001[a]	3.59±0.17 24.65 0.001[b]	2.98±0.042 3.47 0.01 [b]
2nd	Mean ± S.E % of change P	3.90±0.12	2.69±0.12 -31.02 <0.001[a]	3.58±0.14 33.00 0.001[b]	3.2±0.14 18.96 >0.05[b]
3rd	Mean ± S.E % of change P	3.90±0.12	2.69±0.12 -31.02 <0.001[a]	3.32±0.14 23.40 <0.001[b]	3.04±0.14 13.00 <0.01[b]
4th	Mean ± S.E % of change P	3.86±0.18	2.67±0.42 -30.82 <0.001[a]	3.53±0.02 32.21 <0.001[b]	3.24±0.17 21.34 <0.001[b]
5th	Mean ± S.E % of change P	3.79±0.11	2.50±0.28 -34.03 <0.001[a]	3.43±0.05 37.21 <0.01[b]	3.18±0.18 27.20 <0.001[b]
6th	Mean ± S.E % of change P	3.91±0.10	3.12±0.22 -20.20 0.01[a]	3.49±0.17 11.86 >0.05[b]	3.26±0.15 4.40 >0.05[b]
7th	Mean ± S.E % of change P	3.93±0.11	2.41±0.35 -38.68 <0.001[a]	3.51±0.17 45.64 <0.001[b]	3.36±0.15 39.42 <0.001[b]
8th	Mean ± S.E % of change P	4.29±0.46	2.37±0.39 -44.75 <0.01[a]	3.67±0.18 54.85 <0.001[b]	3.44±0.17 45.15 <0.001 [b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 5. Effects of glibenclamide and *Ch.t. extract* administration on serum globulin of alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean % of change P	3.0 ± 0.09	2.39 ± 0.08 - 20.3 <0.001[a]	2.19 ± 0.18 -8.36 >0.05 [b]	2.52 ± 0.08 5.44 >0.05 [b]
2nd	Mean % of change P	2.84 ± 0.10	2.37± 0.17 - 16.5 <0.05[a]	2.71± 0.04 14.34 >0.05 [b]	2.66± 0.16 12.24 >0.05 [b]
3rd	Mean % of change P	2.89 ± 0.13	2.70± 0.12 - 6.57 >0.05 [a]	3.42 ± 0.64 26.67 >0.05 [b]	2.94 ± 0.06 14.04 >0.05 [b]
4th	Mean % of change P	2.92 ± 0.11	2.32 ± 0.12 - 20.55 <0.01[a]	2.99 ± 0.06 28.88 <0.001 [b]	2.95 ± 0.07 27.16 <0.001[b]
5th	Mean % of change P	2.95± 0.14	2.84 ± 0.11 - 3.73 >0.05 [a]	3.18 ± 0.07 11.97 <0.05[b]	3.34 ± 0.05 17.61 <0.001[b]
6th	Mean % of change P	2.83 ± 0.16	2.04 ± 0.30 - 27.91 <0.05[a]	3.06 ± 0.08 50.00 <0.01[b]	3.26 ± 0.05 59.80 <0.01[b]
7th	Mean % of change P	2.78 ± 0.13	2.93 ± 0.07 5.39 >0.05 [a]	3.18 ±0.09 8.53 <0.05[b]	3.19 ± 0.11 8.87 >0.05 [b]
8th	Mean ± S.E % of change P	2.87 ± 0.077	2.74 ± 0.17 - 4.53 >0.05 [a]	3.03 ± 0.11 10.58 >0.05 [b]	2.95 ± 0.13 7.66 >0.05 [b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 6. Effects of glibenclamide and *Ch.t. extract* administration on blood urea of alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	30.17±0.75	49.83± 1.01 65.16 <0.001[a]	48.50± 0.96 -2.67 >0.05 [b]	49.50± 1.06 -0.66 >0.05 [b]
2nd	Mean ± S.E % of change P	29.67 ±0.67	57.17 ± 1.12 94.65 <0.001[a]	52.17 ± 0.75 -8.70 <0.01[b]	54.50±0.84 -4.67 >0.05 [b]
3rd	Mean ± S.E % of change P	30.83 ±0.75	56.17 ± 0.95 82.19 <0.001[a]	51.33 ±1.05 -8.62 <0.01[b]	55.50 ± 0. 85 -8.12 >0.05 [b]
4th	Mean ± S.E % of change P	28.67 ± 0.67	79.83± 1.11 178.44 <0.001[a]	55.50±1.06 -30.48 <0.001[b]	55.83 ± 1.13 -30.00 <0.001[b]
5th	Mean ± S.E % of change P	32.00± 0.77	82.50±2.00 157.81 <0.001[a]	48.83±0.87 -40.80 <0.001[b]	54.50 ±0 .71 -33.00 <0.001[b]
6th	Mean ± S.E % of change P	33.17 ± 0.75	93.33 ± 2.11 181.37 <0.001[a]	48.83±0.94 -47.68 <0.001	56.33 ± 1.18 -39.64 <0.001
7th	Mean ± S.E % of change P	29.33±0.95	96.67 ± 2.00 229.59 <0.001[a]	46.00±1.08 -52.42 <0.001[b]	54.67± 1.02 -43.45 <0.001[b]
8th	Mean ± S.E % of change P	30.33 ±0.88	101.67±2.16 235.21 <0.001[a]	47.17±0.95 -53.60 <0.001[b]	53.67 ± 0.95 -47.20 <0.001[b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 7. Effects of glibenclamide and *Ch.t. extract* administration on serum creatinine of alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	0.59±0.010	0.70±0.024 18.64 <0.01[a]	0.62±0.017 -11.4 <0.05 [b]	0.67±0.015 -0.42 >0.05 [b]
2nd	Mean ± S.E % of change P	0.60± 0.037	0.72±0.027 20.0 <0.001[a]	0.64±0.016 -11.00 <0.01 [b]	0.68±0.012 -5.56 >0.05 [b]
3rd	Mean ± S.E % of change P	0.58±0.013	0.79±.012 36.2 <0.001[a]	0.65±0.031 -17.70 <0.01[b]	0.68±0.01 -13.92 <0.001 [b]
4th	Mean ± S.E % of change P	0.59±0.02	0.87±0.021 47.45 <0.001[a]	0.64±0.012 -26.40 <0.001[b]	0.67±0.011 -22.98 <0.001[b]
5th	Mean ± S.E % of change P	0.58±0.015	0.95±.011 63.79 <0.001[a]	0.64±.014 -32.63 <0.001 [b]	0.65±0.013 -31.58 <0.001 [b]
6th	Mean ± S.E % of change P	0.58±0.019	0.99±0.011 70.68 <0.001[a]	0.63±.013 -36.43 <0.001 [b]	0.65±0.011 -34.34 <0.001 [b]
7th	Mean ± S.E % of change P	0.59±0.022	1.01±0.017 71.18 <0.001[a]	0.62±0.013 -38.60 <0.001 [b]	0.66±0.015 -34.65 <0.001 [b]
8th	Mean ± S.E % of change P	0.60±0.017	1.01±0.010 68.33 <0.001[a]	0.63±0.010 -37.62 <0.001 [b]	0.64±0.019 -36.63 <0.001 [b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

Table 8. Effects of glibenclamide and *Ch.t. extract* administration on serum Uric Acid of alloxan induced diabetic male rats for 8 weeks.

Duration of Experiments (week)		Group I	Experimental Groups		
			Group II	Group III	Group IV
1st	Mean ± S.E % of change P	1.70±0.006	1.50±0.006 -11.76 <0.001[a]	1.50±0.009 0.00 >0.05 [b]	1.50±0.007 0.00 >0.05 [b]
2nd	Mean ± S.E % of change P	1.69±0.007	1.49±0.006 -11.83 <0.001[a]	1.50±0.011 0.67 >0.05 [b]	1.49±0.007 0.00 >0.05 [b]
3rd	Mean ± S.E % of change P	1.70±0.012	1.45±0.019 -14.70 <0.001[a]	1.49±0.014 2.76 >0.05 [b]	1.50±0.004 3.45 <0.05[b]
4th	Mean ± S.E % of change P	1.70±0.014	1.45±0.011 -14.70 <0.001[a]	1.50±0.010 3.45 <0.01[b]	1.50±0.003 3.45 0.001[b]
5th	Mean ± S.E % of change P	1.70±0.018	1.4±0.014 -17.64 <0.001[a]	1.59±0.010 13.57 <0.001[b]	1.60±0.006 14.29 <0.001[b]
6th	Mean ± S.E % of change P	1.7±0.089	1.33±0.008 -21.76 <0.001[a]	1.59±0.012 19.55 <0.001	1.60±0.004 20.30 <0.001
7th	Mean ± S.E % of change P	1.69±0.006	1.35±0.006 -20.11 <0.001[a]	1.59±0.013 17.78 <0.001[b]	1.60±0.007 18.52 <0.001[b]
8th	Mean ± S.E % of change P	1.69±0.007	1.35±0.007 -20.11 <0.001[a]	1.57±0.019 16.30 <0.001[b]	1.60±0.008 18.52 <0.001[b]

SE: Standard error; P value <0.05 significant; NS (nonsignificant, P >0.05); P<0.01 highly significant; P<0.001 more highly significant .[a]: compared with normal control; [b] compared with alloxan (negative control) group.

***Group I**, Rats were untreated and served as normal control. **Group II**, diabetic rats without treatment (-ve control) "diabetic control". **Group III,** diabetic rats orally administered with glibenclamide. **Group IV**, diabetic rats orally administered with Ch.t. extract.*

ACKNOWLEDGEMENTS

Authors would like to thank Alaqsa University for providing facilities and laboratories that facilitated conducting this research. They also would like to thank Al Shifa Hospital Clinical Chemistry Laboratoryfor theirkind help.

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