Effect Of Dietary Supplementation With Tigernut Tubers On Streptozotocin-Induced Diabetic Rats

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Abstract

This work has been carried out to detect the effect of tigernut tubers as an antidiabetic plant on some biochemical parameters in streptozotocin (STZ)-induced diabetic rats. Diabetes mellitus was induced by a single intraperitoneal injection of 45 mg STZ /kg body weight. The present results indicated an increase in both serum glucose level and liver glucose-6-phosphatase (G6P) activity in STZ-diabetic rats. In addition, a significant decrease in serum insulin level and liver glycogen content was recorded in the same rats. Moreover, serum total lipids, total cholesterol, triglycerides and LDL-cholesterol levels revealed a significant increase, while a decrease in the level of HDL-cholesterol was observed in STZ-diabetic rats. The activity of serum aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) showed marked increase in STZ-diabetic rats. On the other hand, tigernut-supplemented diet (25% w/w) for two or four weeks recorded a significant improvement in all the above biochemical parameters affected by STZ injection especially after four weeks of treatment. Therefore, it was concluded that tigernut tubers had appreciable hypoglycemic and hypolipidemic effects on STZ-diabetic rats.

Key Words: Tigernut (Cyperus esculentus) – STZ-diabetic rats – lipid profiles

Introduction

Diabetes mellitus is one of the most common non-communicable world wide diseases. It is the major metabolic disorder characterized by insufficient insulin secretion and/or insensitive target tissues to metabolic actions of insulin (Motala et al., 2000., Pignone, 2007). Despite the progressive efforts to produce synthetic drugs for treating diabetic patients, there is a widespread propensity of patients to herbal medicine which can face the high cost and the poor availability for many rural populations, particularly in developing country (Marles and Fransworth, 1995., Somani et al., 2007). Recently, traditional plant medicines or herbal formulations to treat diabetes could form the basis for new treatments for the disease (Abdel-Monem, 2005; Brownlee, 2006., Rao, 2007). Also it might offer a natural key to unlock diabetic complications (Babu et al., 2006). In addition, the Egyptian herbal medicine was widely respected throughout the ancient Mediterranean world. Among these herbal remedies consumption of tigernut (yellow nutsedge) (Cyperus esculentus) is relatively popular in some societies as an antidiabetic agent (Gupta et al., 1971., Ghazanfar, 1994).

Tigernut tubers (Cyperus esculentus) are edible, with a slightly sweet and nutty flavor. The tubers are used as a foodstuff, particularly in Africa, where it’s an important food crop with certain tribes. Tigernut have excellent nutritional qualities with a fat composition similar to olives (Coskunerm et al., 2002). It is also gluten and cholesterol free (Wills et al., 1980).

Moreover, it is the richest food source of flavonoids (El-Habashy, 1988) and also rich in water, fibers, alkaloids, digestible carbohydrates, saponins and fatty oils (glycerides), in addition to some elements, like phosphors, potassium, calcium, iron, zinc, magnesium and manganese (Addy and Eteshola, 1984; Jeong et al., 2000). The fatty oil content in tigernut was 25-27%, these fatty acids classified into dominant saturated fatty acids such as miristic acid as well as dominant unsaturated fatty acids
such as oleic acid, linoleic acid, palmitic acid and stearic acid (Eteshola and Oraedu, 1996; Salem et al., 2005).

Chung et al. (1992) denoted that tigernut had marked therapeutic effects for intestinal metaplasia and hyperplasia and the animal experiments revealed no toxic effect, so safety guarantee was provided for its clinical application. In addition, it is considered as alterative, analgesic, antibacterial, antimalarial, antimicrobial, antipyretic, astringent, carminative, demulcent, diaphoretic, diuretic, hypoglycemic, hepatoprotective, hypotensive, stomachic, tonic and vermifuge (Gupta et al., 1971; Rahman and Zaman, 1989; Ghazanfar, 1994; Mehta et al., 1999). Also it exhibit anti-inflammatory properties upon inflammation, and immunostimulatory effects (Salem et al., 2005).

It was observed that there is a lack of experimental work on the beneficial effects of Egyptian tigernut. So the purpose of this study was to evaluate the hypoglycemic effect of tigernut tubers on STZ-induced experimental diabetes mellitus and its role in modulating the abnormal biochemical parameters associated with this disease.

**Material and methods**

**Materials:**
Streptozotocin (STZ) was purchased from Sigma company, USA. Tigernut tubers were obtained from the local market at Tanta city, Egypt. For the preparation of tigernut powder, the tubers were cleaned, washed and dried in a stream of hot air for an hour. The dried tubers were milled using a laboratory electric mill pass through a 40-mesh sieve.

**Animals, experimental design and blood sampling:**
Male albino rats (Rattus rattus) weighing 160-180 g were used in this study. Animals were kept under good ventilation and received a balanced diet and water *ad libitum* through out the experiments. The standard diet consisted of casein 15.0 %, starch, 67.0 %, corn oil 8.0 %, salt mixture 4.0 %, vitamin mixture 1.0 % and wood fiber 5.0 % as reported by Ulloa et al. (1988). Experimental animals were divided into four main groups of six rats each, as follows:-

1- Control rats group: Rats of this group were injected intraperitonealy with a single dose of citrate buffer (PH 4.5) and considered as a control rats group.

2- Tigernut-fed rats group: These rats received a balanced diet supplemented with 25 % whole powder of tigernut tubers (experimental diet) (Salem et al., 2005). The average chemical composition (g/100g) of tigernut tubers includes starch 34, oil 25, protein 8, sucrose 16, fiber 10 and others 7. The diets were designed to contain equal amounts of energy (about 3606 Kcal/Kg diet) (Salem et al., 2005).

3- Diabetic rats group: The rats of this group were made diabetic by a single intraperitoneal injection of 45 mg STZ /kg of body weight dissolved in citrate buffer (PH 4.5) to overnight fasted animals (Ahmed, 2001). The rats with glucose level 180 mg/L or more were considered as STZ-diabetic rats.

4- Diabetic and tigernut-fed rats group: The diabetic rats were fed on tigernut supplemented diet (25% w/w).

After both treatment periods; two and four weeks, rats of each group were sacrificed. Blood samples were collected and the clear non-haemolised sera were quickly removed and stored at -20 °C for subsequent biochemical measurements.

**Methods and techniques**

1- Serum levels of glucose, total lipids, total cholesterol, HDL-cholesterol and triglycerides were determined according to the methods of Trinder (1969), Frings et al. (1972), Allian et al. (1974), Burstein et al. (1970) and Fossati and Prencipe (1982), respectively, using kits from Diamond Diagnostic Chemical Company (Egypt).

2- Serum aminotransferases (AST and ALT) enzymes activity was measured by the method described by Reitman and Frankel (1957), and alkaline phosphatase activity was determined depending on the method of Belfield and Goldberg, (1971) using a
commercially available reagent kits obtained from Randox Lab Ltd, U.K.

3- Serum LDL-cholesterol (LDL-C) level was calculated according to Friedewald (1972) formula:

\[ \text{LDL-C} = \text{Total cholesterol concentration} - \text{Triglyceride concentration} - \text{HDL-C concentration} \]

4- Serum insulin level was measured using immuno-enzymatic assay Kits for the quantitative measurement of insulin in serum (Flier et al., 1979).

5- Liver glycogen content was estimated according to the technique of Van Handle (1965).

6- Glucose-6-phosphatase (G6P) activity was estimated depending on the method of Swanson (1955).

Statistical analysis:
The results obtained in the present study were evaluated by One Way ANOVA (analysis of variance) test and post-comparison was carried out with Tukey test. The results were expressed as mean ± standard error and values of P< 0.05 were considered statistically significant (Snedecor and Cochran, 1982).

Results
As shown in Table 1, a single injection of streptozotocin (STZ) caused a significant increase in the level of serum glucose accompanied by a marked decrease in serum insulin and liver glycogen levels however the activity of liver G6P was significantly increased in STZ-diabetic rats. However, STZ-diabetic rats which were supplied with tigernut tubers for two experimental periods of treatment showed an improvement in these parameters.

Table 2 illustrates significant increases in serum total lipids, total cholesterol, LDL-cholesterol and triglycerides in STZ-diabetic rats, while a significant decrease in HDL-cholesterol was recorded. Meanwhile, the feeding on tigernut tubers-supplemented diet ameliorated the level of the mentioned lipid fractions especially after four weeks of treatment.

Table 3 shows the activity of liver enzymes such as AST, ALT and ALP which were increased in STZ-diabetic rats. However a significant decrease was noticed in these enzymes activity in STZ-diabetic groups that received tigernut tubers for two or four weeks. The observed ameliorative effect of tigernut supplemented diet was more pronounced after four weeks of the treatment.

Concerning ANOVA analysis of the investigated parameters it was revealed that the general effect between groups was significant (P<0.05) throughout the experiment.
### Table 1. Serum glucose and insulin levels, liver glycogen content and G6P enzyme activity in control and treated groups.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Animal Groups</th>
<th>ANOVA</th>
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<td></td>
<td>Control</td>
<td>Tigernut tuber</td>
<td>STZ-Diabetic</td>
<td>STZ-Diabetic + Tigernut tuber</td>
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<td>2 wk</td>
<td>4 wk</td>
<td>2 wk</td>
<td>4 wk</td>
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</tr>
<tr>
<td>Serum Glucose</td>
<td>104.7</td>
<td>99.7</td>
<td>97.0</td>
<td>209.5</td>
<td>202.5</td>
<td>110.3</td>
<td>97.0</td>
</tr>
<tr>
<td>mg/dl</td>
<td>±0.88</td>
<td>±0.88</td>
<td>±0.81a</td>
<td>±1.31a</td>
<td>±1.18a</td>
<td>±0.98ab</td>
<td>±1.71ac</td>
</tr>
<tr>
<td>Insulin</td>
<td>4.2</td>
<td>4.4</td>
<td>4.5</td>
<td>2.4</td>
<td>2.5</td>
<td>3.4</td>
<td>3.7</td>
</tr>
<tr>
<td>µU/L</td>
<td>±0.08</td>
<td>±0.06</td>
<td>±0.03a</td>
<td>±0.04a</td>
<td>±0.04ab</td>
<td>±0.04ab</td>
<td>±0.08ac</td>
</tr>
<tr>
<td>Liver Glycogen</td>
<td>35.8</td>
<td>35.8</td>
<td>35.5</td>
<td>21.0</td>
<td>19.1</td>
<td>28.5</td>
<td>31.8</td>
</tr>
<tr>
<td>mg/100 g wt</td>
<td>±0.41</td>
<td>±0.22</td>
<td>±0.18</td>
<td>±0.13a</td>
<td>±0.21a</td>
<td>±0.23ab</td>
<td>±0.24ac</td>
</tr>
<tr>
<td>G6P</td>
<td>0.68</td>
<td>0.68</td>
<td>0.68</td>
<td>0.99</td>
<td>1.1</td>
<td>0.96</td>
<td>0.81</td>
</tr>
<tr>
<td>µMol Pi/min/g wt</td>
<td>±0.004</td>
<td>±0.003</td>
<td>±0.004a</td>
<td>±0.004a</td>
<td>±0.03a</td>
<td>±0.02a</td>
<td>±0.004ac</td>
</tr>
</tbody>
</table>

Data are expressed as means ± S.E. of six rats.
ANOVA: P = probability
Tukey test: a = significant difference as compared to control group.
b = significant difference as compared to STZ-diabetic group after 2 wk.
c = significant difference as compared to STZ-diabetic group after 4 wk.

### Table 2. Serum total lipids, total cholesterol, triglycerides, HDL-C and LDL-C levels in control and treated groups.

<table>
<thead>
<tr>
<th>Parameters</th>
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<td></td>
<td></td>
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<td>STZ-Diabetic + Tigernut tuber</td>
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<td></td>
<td></td>
<td></td>
<td>2 wk</td>
<td>4 wk</td>
<td>2 wk</td>
<td>4 wk</td>
<td>2 wk</td>
</tr>
<tr>
<td>Total lipids</td>
<td>802</td>
<td>791</td>
<td>784</td>
<td>887</td>
<td>892</td>
<td>829</td>
<td>821</td>
</tr>
<tr>
<td>mg/100 ml</td>
<td>±1.7</td>
<td>±1.8a</td>
<td>±1.4a</td>
<td>±1.1a</td>
<td>±1.1a</td>
<td>±0.88ab</td>
<td>±0.73ac</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>99.3</td>
<td>94.8</td>
<td>90.7</td>
<td>140.3</td>
<td>148.7</td>
<td>121.0</td>
<td>114.0</td>
</tr>
<tr>
<td>mg/100 ml</td>
<td>±0.80</td>
<td>±0.60a</td>
<td>±0.49a</td>
<td>±0.76a</td>
<td>±0.76a</td>
<td>±0.76ab</td>
<td>±0.88ac</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>86.9</td>
<td>84.0</td>
<td>81.2</td>
<td>115.2</td>
<td>118.3</td>
<td>103.8</td>
<td>98.3</td>
</tr>
<tr>
<td>mg/100 ml</td>
<td>±0.59</td>
<td>±0.58</td>
<td>±0.31a</td>
<td>±1.1a</td>
<td>±1.2a</td>
<td>±1.3ab</td>
<td>±0.67ac</td>
</tr>
<tr>
<td>HDL-C</td>
<td>42.9</td>
<td>44.3</td>
<td>46.5</td>
<td>34.8</td>
<td>31.7</td>
<td>36.8</td>
<td>40.7</td>
</tr>
<tr>
<td>mg/100 ml</td>
<td>±0.60</td>
<td>±0.49</td>
<td>±0.33a</td>
<td>±0.57a</td>
<td>±0.44a</td>
<td>±0.60a</td>
<td>±1.01ac</td>
</tr>
<tr>
<td>LDL-C</td>
<td>39.02</td>
<td>33.7</td>
<td>27.96</td>
<td>82.5</td>
<td>93.3</td>
<td>63.4</td>
<td>53.6</td>
</tr>
<tr>
<td>mg/100 ml</td>
<td>±0.90</td>
<td>±0.85a</td>
<td>±0.37a</td>
<td>±0.59a</td>
<td>±0.56a</td>
<td>±0.47ab</td>
<td>±0.49ac</td>
</tr>
</tbody>
</table>

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<th>ANOVA</th>
<th>P</th>
</tr>
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<tr>
<td></td>
<td></td>
<td></td>
<td>2 wk</td>
<td>4 wk</td>
<td>2 wk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST U/IL</td>
<td></td>
<td>35.6</td>
<td>34.5</td>
<td>31.0</td>
<td>44.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±0.76</td>
<td>±0.43</td>
<td>±0.37*</td>
<td>±0.83*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALT U/IL</td>
<td></td>
<td>26.7</td>
<td>25.7</td>
<td>24.5</td>
<td>46.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>±0.39</td>
<td>±0.56</td>
<td>±0.43</td>
<td>±0.49*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALP KArm μ/100 ml</td>
<td></td>
<td>118.4</td>
<td>114.0</td>
<td>112.8</td>
<td>148.8</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td>±0.86</td>
<td>±0.58*</td>
<td>±0.91*</td>
<td>±0.79*</td>
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</tr>
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Discussion

In the present study, STZ-induced diabetic rats showed developed progressive increase in serum glucose level, which seems to be explained by deficient insulin secretion as a result of β-cell destruction by STZ (Santini et al., 1997; Szkudelski, 2001; Mazhar et al., 2005). However, it is now knowned that when β-cell destruction is the primary cause of diabetes, insulin resistance in addition to deficient insulin secretion is thought also to be necessary for occurrence of hyperglycemia (Scoppola et al., 2001; Abdel-Monem, 2005). The biochemical basis of diabetes-induced insulin resistance could involve changes in glucose transporter number (Lillioja et al., 1988) or in insulin-induced activation of glycogen synthase, which reflect primarily depressed glycogen synthesis (Ortmeyer et al., 1994). This could provide explanation for the decreased ability of the liver to accumulate glycogen in the present and other diabetic studies (Margolis et al., 1985). However, in the present study it is more likely that the depleted hepatic glycogen stores could originate from the increased glycogenolysis as indicated by the presently increased G6P enzyme activity. In previous studies, the increased glycogenolysis was suggested to be responsible for the increase in hepatic glucose overproduction in diabetes (Venkatraman et al., 1991) and thus the increased blood glucose level (Pignone, 2007). However, the increase in gluconeogenesis in diabetes was also shown to play a determinant role in these phenomena (Beck-Nielsen et al., 1994). An explanation for the increased gluconeogenesis is that the state of insulin deficiency or insulin resistance in diabetes is almost associated with the relative insufficient tissue insulin suppression of lipolysis, which in turn may increase the rate of lipid oxidation. In association with these reportedly diabetes-induced changes in carbohydrate metabolism, various studies have demonstrated a general elevation in almost plasma lipid fractions that often coexist with hyperglycemia (Santini et al., 1997; Mazhar et al., 2005). Insulin can affect the adipocytes by inhibiting lipolysis and promoting storage of triglycerides in adipocytes (Zaahkouk, 2001). Thus, insulin lack in diabetes enhances hydrolysis of triglycerides into diglycerides, unesterified fatty acids and free glycerol (Ebara, 1994). These fatty acids may diffuse out of the cells or may be reesterified into triglycerides for storage or
Effect Of Dietary Supplementation With Tigernut........

secretion of VLDL. Because insulin is known to suppress VLDL secretion, the lack of this suppression by deficient insulin secretion in diabetes may lead to hypertriglyceridemia, which possibly can mediate the mechanism by which diabetes causes hyperlipidemia (Saudek and Eder, 1979). As the concentration of triglycerides increases in the circulation, this increases the hydrolysis of triglycerides from VLDL, yielding LDL. These particles carry most of the cholesterol in the blood and are cleared mainly by the hepatic LDL receptor, which are increased by insulin (Saudek and Eder, 1979). Consequently, the clearance of LDL can be delayed resulting in hypercholesterolemia (Tsutsunmi et al., 1995). In other words, the reduction in the rate of cholesterol removal from circulation appears responsible for hypercholesterolemia recognized in diabetic states. In this field of research, other data documented a marked increase of serum AST and ALT and ALP activities in diabetic animals (Eskander et al., 1995). These findings have further confirmed by Rawi et al. (1998).

This elevation in enzymatic activities was attributed to their greater need for gluconeogenic substrates. Eskander et al. (1995) found that liver was necrotized in diabetic rats. Moreover, striking elevations of serum AST and ALT activities were observed in patients with acute hepatic necrosis. In addition, the elevation of the serum ALP activity in diabetic rat; may be attributed to the elevation of a small intestinal alkaline phosphatase activity in these animals (Unakami et al., 1990; Mansour et al., 2002).

On the other hand, several medicinal plants, their extracts or different formulations have been given in the treatment of diabetes mellitus. The major merits of herbal medicines are their inherent efficacy, low incidence of side effects and low cost. Recently, Nutrition recommendation suggested that diet is the mainstay therapy in people with type 2-diabetes, yet the ideal dietary guidelines for people with diabetes remain controversial (Somani et al., 2007). Recommendations aim to promote nutritional factors that have been shown to improve outcome, such as good glycemic control and maintaining ideal body weight, while reducing the risk of coronary heart disease through improved lipid profiles (Nutrition recommendation, 1999). Herbal botanical products have benefits for controlling diabetes beyond the inhibition of glucose absorption, including stimulating insulin secretion and/or action, improving insulin binding, improving capillary function, and preventing polyunsaturated fatty acids (PUFAs) peroxidation. (Broadhurst, 1997; Abdel Moneim, et al., 2001; Mansour et al., 2002; Babu et al., 2006).

The present results, on the other hand, demonstrated that the dietary supplementation with tigernut had an improving effect on the various metabolic disturbances associated with diabetes in rats. This is indicated by an improvement of glucose and insulin levels, concomitantly with increased hepatic glycogen content, indicating stimulated glucose utilization. Furthermore, the concentration of lipid fractions as well as liver function enzymes was found to be lowered near to normal values. These findings are agreement with the recent study of Raut and Gaikwad (2006) who suggested the hypoglycemic effect of tigernut on alloxan-diabetic rats. The observed metabolic improvements in the present study could contribute to the presence of the important nutritive agents of the tigernut tubers; polyunsaturated fatty acids (PUFAs). Epidemiological evidence and intervention studies clearly indicate the beneficial effects of polyunsaturated fatty acids (PUFAs) in diabetic state as well as in the prevention and treatment of long-term metabolic abnormalities associated with diabetes by the reduction of hyperlipidemia and hyperglycaemia (Merzouk and Khan, 2003; Ristic and Ristic, 2003; Rivellese and Lilli, 2003). The hypoglycemic effect of tigernut may be related to the ability of PUFAs to increase the number of insulin receptors, therefore increasing insulin activity. Similar results were seen by Das (1995) who suggested that feeding on high PUFAs diets can significantly affect insulin efficiency and glucose response in STZ-induced diabetic rats. In addition, this antihyperglycemic activity may be attributed to the antioxidant activity of tigernut as it has the strong free radical scavenging action (Raut and Gaikwad, 2006). Furthermore, the antihyperglycemic
effect of tigernut may be due to its fibers and/or manganese contents (Abdel-Kader, 1986). Based on the results of Abdel-Barry et al. (1997) and Stillie et al. (2005), plants fibers-enriched diets cause delay in the absorption of carbohydrates from the diet thereby reducing blood glucose level. Also, it has been generally assumed that manganese has been shown to exert a hypoglycemic action in independent diabetic mellitus (IDDM) patients and it is now recognized as necessary co-factor for ATP phosphorylation of β-subunit of the insulin receptor (Reddy and King, 1987). The treatment with manganese salt resulted in a net increase in acute insulin response and the rate of glucose disappearance after glucose loading (White and Campell, 1993). Moreover, the present data revealed a hypocholesterolemic and hypolipidemic action of tigernut tubers. These data were agreement with previous studies (Salem et al., 2004). Also, our previous results suggested that dietary tigernut possesses considerable a hypocholesterolemic potential in both experimental aged rats (Hassan, 2005) and hypercholesterolemic rats (Hassan, 2006). The observed ameliorative effect of tigernut on lipid metabolism may be due to its poly unsaturated fatty acid contents. Diets enriched with poly unsaturated fatty acids have been recommended by several medical and nutritional aims because of the lipid lowering effects of PUFAs in the serum (An et al., 1995; Schmid and Woollett, 2003).

Also, tigernut administration could ameliorate the increase of serum enzymes activity through improvement of liver functions indicating its protective effect through conservation of glutathione; the main protective intracellular sulphhydryl peptide of the hepatocytes (Bamgbose et al., 1997; Mehta et al., 1999). Glutathione (GSH) plays an important role in protecting cells against oxidative stress by virtue of being a substrate for glutathione peroxidase and glutathione-S-transferase (Kurata et al., 1993). In addition, GSH disposes oxygen radical non-enzymatically (Wefers and Sies, 1983; Mosoni et al., 2004). Recently, several epidemiological studies and laboratory experimentation have yielded sound data and evidence in support of the fact that medicinal plants enhance the antioxidant defense against reactive oxygen species produced under hyperglycemic condition and this protects β-cells against loss, exhibit antidiabetic property and can attenuate the hazard effects of diabetes (Anuradha and Ravikumar, 2001; Badami and Channabasavaraj, 2007; Kumaran and Joel Karunakaran, 2007).

**In conclusion**, this study supports the concept that tigernut tubers exerted anti-hyperglycemic effects and consequently may alleviate liver dysfunction caused by STZ-induced diabetes, suggesting that the addition of this plant to the treatment protocols used for diabetic patients may improve diabetic therapy. However, further studies are also required to define the active ingredients of this promising plant. Taken together, the results of the present work highlight the nutritional benefits of tigernut in case of diabetes.

**References**


Effect Of Dietary Supplementation With Tigernut........


تأثير وجبة غذائية مدعمة بدرنات حب العزيز على الجرذان المصاب بمرض السكر المستحدث بالستريتزوتوسین

هنا على حسن
قسم علم الحيوان - كلية العلوم - جامعة المنصورة

جاء حب العزيز ضمن الوصفات العلاجية الفرعونية القديمة، ثم وصفه الأطباء العرب القدامى لعلاج العديد من الأمراض، وقد جاءت الدراسات الطبية الحديثة لتؤكد أن هذا النبات ذو فوائد علاجية عظيمة. ولذا تهدف هذه الدراسة إلى تقييم تأثير تناول وجبة مزودة بمسحوق درنات حب العزيز على مستوى الجلوكوز والإنسبولين بالصين وبعض المتغيرات المرتبطة بأيض الكربوهيدرات والدهون في الجرذان المصاب بالسكر المستحدث بواسطة الستريتزوتوسین.

وقد أظهرت النتائج أن الحقن بجرعة واحدة من عقار الستريتزوتوسین (45 ملجم/كم من وزن الجسم) أدى إلى زيادة في مستوى الجلوكوز في الصين وكذلك زيادة نشاط إنزيم الجلوكوز-6-فوسفاتاز في الكبد وخفض مستوى الجليكوجين في الكبد بالإضافة إلى نقص مستوى الإنسولين في مصل الدم. وقد سجلت الدراسة أيضاً زيادة في نسبة الدهون الكلية والكوليسترول والدهون ذات الكثافة المنخفضة والجليسريدات الثلاثية مصحوبة بنقص في مستوى الدهون ذات الكثافة المرتفعة، بالإضافة إلى زيادة ملحوظة في إنزيمات الكبد مثل إنزيمات النقل الأمين (ALT & AST) وكذلك إنزيم الفوسفات القاعدى (ALP) فى الجرذان المصابية بمرض السكر المستحدث بالستريتزوتوسین.

وعلى الجانب الآخر فقد أوضحت الدراسة أن إضافة حب العزيز بنسبة 25% في الغذاء لمدة أسبوعين أوربيجين أن أسباب قد أحدث تحسناً ملحوظاً في تركيز الجلوكوز بالصين والقياسات الخاصة بأيض الكربوهيدرات والدهون بالإضافة إلى اضطرابات الكبد المفيدة حيث اقتربت من معدلها الطبيعي وقد كان التحسن أكثر وضوحا بعد أربع أسابيع من المعاملة بحب العزيز.

من الممكن التسخيف أن استخدام حب العزيز له تأثير مضاد للسكر وخفض للدهون في الجرذان المصابية بالسكر. وتوصى البحوث بإجراء المزيد من الدراسات عليه للتعرف على الأليات المختلفة له و مدى إمكانية استخدامه في المجال الطبي.