

**Abstract:** 

Journal of Home Economics Volume 30, Number (1), 2020 Journal of Home Economics

http://homeEcon.menofia.edu.eg

ISSN 1110-2578

### Possible Effects of Sweet Potato Leaves, Roots and their Mixture Feeding on Alloxan-Induced Diabetic Ratsm

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The present study was designed to study the effect of sweet potato (Ipomoea batatas, L.) leaves, roots and their mixture on diabetic rats. Forty eight adult male albino rats were used in this study, weighting (150±10g) were divided into eight groups, six rats each. Sweet potato leaves, roots and their mixture as powder were added at percent 2.5 % and 5% from the main diet for 28 days. Diabetic rats were induced alloxan subcutaneously injection (150 mg/kg body weight). Serum lipid profiles (TG, TC, LDL-c, VLDL-c, HDL-c), serum glucose, liver enzymes activities (ALT, AST and ALP) and kidney functions (creatinine, uric acid and urea levels) were also determined. From the obtained results it could be concluded that feeding on sweet potato leaves, roots and their mixture as powder caused significant ( $P \le 0.05$ ) increase in HDL-c, as compared with control (-ve) group, and enhanced the kidney and liver functions with the decrease of ALT, AST, ALP, serum glucose, creatinine, uric acid and urea which reflects the powerful nutraceutical therapeutic effects. Feeding on sweet potato leaves, roots and their mixture, as a powder attenuated diabetic effects in rats, the best result was to concentrate 5% mixture of sweet potato leaves and roots powder.

Key words: Sweet potato, Diabetic, Rats and Biochemical analysis.

#### Introduction:

Diabetes mellitus (DM) is a group of metabolic diseases characterized by hyperglycemia, hypertriglyceridemia and hypercholesterolemia, resulting from defects in insulin secretion, its action or both (Georg and Ludvik, 2000).

Diabetes is a chronic disease that occurs when the pancreas does not produce enough insulin (a hormone that regulates blood sugar) or alternatively, when the body cannot effectively use the insulin it produces. The overall risk of dying among people with diabetes is at least double the risk of their peers without diabetes (WHO, 2014).

Diabetes mellitus is a clinical syndrome due to relative or absolute deficiency of insulin or resistance to the action of insulin at the cellular level; as a result, hyperglycemia and glucosurea occurs (Al-Ali, 2016).

Diabetes is the world's largest endocrine disease associated with increased morbidity and mortality rate. Diabetes mellitus is also associated with long term complications including retinopathy, nephropathy, neuropathy and angiopathy and several others (Sharma *et al.*, 2010).

All forms of diabetes increase the risk of long-term complications. These typically develop after many years (10–20), but may be the first symptom in those who have otherwise not received a diagnosis before that time. The major long-term complication relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease and about 75% of deaths in diabetics are due to coronary artery disease. Other "macrovascular" diseases are stroke, and peripheral vascular disease (**O'Gara** *et al.*, **2013**).

Taxonomically, *I. batatas* belongs to the morning glory family, *Convolvulaceae*, and the only member of the genus *Ipomoea* whose roots are edible. It is speculated to be a native of South America but presently grown throughout the tropical and subtropical regions of the world (Scott, 2003).

*Ipomoea batatas,* L. (Family: *Convolvulaceae*) is commonly known as sweet potato, it is the world's sixth largest food crop, which is widely grown in tropical, subtropical and warm temperate regions. It is an important food crop in many countries, also cultivated for its use as animal feed and as a medicinal plant (**Truong and Avula, 2010**).

Sweet potatoes are rich in complex carbohydrates, dietary fiber and beta carotene (a precursor of vitamin A), vitamin B6 and vitamin C.

In addition to this, various parts of the crop have been reported to also contain mineral nutrients such as zinc, potassium, sodium, manganese, calcium, magnesium and iron (**Kumar** *et al.*, **2006**).

Sweet Potatoes (SP) can be differentiated into several types based on the color of the tuber, like white, yellow, orange, white striped purple and purple. In each type of sweet potato has a nutrient content and functionally different. Both sweet- potato roots and leaves are considered to be rich sources of phenolic compounds, high contribute toward the antioxidant activity of sweet potato tissues (Utami and Rahayn, 2012).

Sweet potatoes are also good for diabetics because they contain a good deal of fiber, particularly when the peels are left on. The amount of fiber in a food slows down the rate of digestion of the starches. This action in turn lowers the glycemic index of the sweet potato and helps keep blood sugar levels within a manageable range (Velloso *et al.*, 2004).

Sweet potatoes are a good food choice for diabetics as they are high in fiber and have a low glycemic index. Foods with a low glycemic index have less of an immediate impact on blood glucose levels, and therefore can help diabetics control their blood sugar (**Dutta, 2015**).

Akhtar et al., (2018) concluded that anti-diabetic potential of white skinned sweet potato (WSSP) extract is due to the presence of bioactive compounds like glycoprotein, anthocyanins, alkaloids, and flavonoids, which act as insulin-like molecules or insulin secretagogues constituents in sweet potatoes peel-off and these anti-diabetic proteins were extracted out in more concentration in methanol due to its organic nature.

**Strugala** *et al.*, (2019) showed that the administration of purple sweet potato (PSP) lowered blood glucose, improved glucose tolerance, and decreased the amount of glycated hemoglobin. Furthermore, PSP demonstrated an antioxidative effect, suppressed malondialdehyde levels, and restored antioxidant enzyme activities in diabetic rats. After administration of PSP, we also noticed inhibition of OMP, AGE, and AOPP formation in the rats blood plasma.

This work was conducted to study the effect of different concentrations of sweet potato leaves (SPL), sweet potato roots (SPR) and their mixture as powder on biological and biochemical complications of diabetic rats.

### Material and Methods Materials:

#### Source of sweet potato leaves and roots:

Sweet potato (*Ipomoea batatas*, L) leaves and roots were obtained from local Farm, Sheben El-Kom City, Menoufia Governorate, Egypt. **Alloxan:** 

Alloxan, which is chemically known as 5, 5-dihydroxyl pyrimidine-2, 4, 6-trione is an organic compound, a urea derivative, a carcinogen and cytotoxic glucose analog which was obtained from Al-Gomhoria Company for Drugs, Chemical and Medical Instruments, Cairo, Egypt.

#### **Experimental animals:**

A total of 48 adult normal male albino rats "Sprague Dawley" strain weighing  $150\pm10$  g were obtained from Vaccine and Immunity Organization, Ministry of Health, Helwan Farm, Cairo, Egypt.

#### The chemicals and kits:

Pure white crystalline cholesterol powder and saline solutions were purchased from SIGMA Chemical Co., Egypt. Casein, cellulose, choline chloride powder, and DL methionine powder, were obtained from Morgan Co. Cairo, Egypt. Chemical kits used in this study (TC, TG, HDL-c, ALT, AST, ALP, bilirubin, urea, creatinine, albumin) were obtained from Al-Gomhoria Company for Drugs, Chemical and Medical Instruments, Cairo, Egypt.

#### Methods

#### **Preparation of sweet potato leaves and roots:**

To prepare the dried sweet potato leaves and roots were washed thoroughly under running tap water, summarize dried at 45 °C for 24 hr., and ground to a fine powder using an air mill, high speed mixture (Molunix, Al-Araby company, Benha, Egypt) and then serving as powder seize.

#### **Experimental design:**

Forty Eight (48) adult male white albino rats, "Sprague Dawley" Strain, 10 weeks age, weighing  $(150\pm10g)$  were used in this experiment. All rats were fed on basal diet (casein diet) prepared according to **AIN**, (1993) for 7 consecutive days for adaptation. After this adaptation period, rats were divided into 8 groups, six rats per each as follows: group (I): rats fed on basal diet only as negative control. Group (2): injected with alloxan at dose of 150 mg/kg and were fed on standard diet

only without any treatment and used as a positive control group. Group (3): diabetic rats fed on sweet potato leaves as powder by 2.5% of diet. Group (4): diabetic rats fed on sweet potato leaves as powder by 5% of diet Group (5): diabetic rats fed on the sweet potato roots 2.5% of diet. Group (6): diabetic rats fed on the sweet potato roots 5% of diet. Group (7): diabetic rats fed on the mixture (1:1) of sweet potato leaves and roots 2.5% of diet. Group (8): diabetic rats fed on the mixture (1:1) of sweet potato leaves and roots 5% of diet. The experiment continued for 28 days, at the end of the experimental period each rat weight separately, then slaughtered and blood samples were collected.

#### **Blood sampling:**

After fasting for 12 hours, blood samples were obtained from hepatic portal vein at the end of each experiment. The blood samples were collected into a dry clean centrifuge glass tubes and left to clot in water bath (37°C) for 30 minutes, then centrifuged for 10 minutes at 3000 rpm to separate the serum, which were carefully aspirated and transferred into clean vile tube and stored frozen in deep freezer (-20°C) till analysis according to method described by Schermer (1967).

#### **Biochemical analysis**

#### **Determination of blood glucose**

Serum glucose was measured using the modified kinetic method according to Kaplan, (1984) by using kit supplied by spin react. Spain. **Liver functions** 

#### Determination of alanine amino transferase (ALT)

ALT activities were measured in serum using the modified kinetic method of Hafkenscheid (1979) by using kit supplied by Human, Germany.

#### **Determination of aspartate amino transferase (AST)**

AST activities were measured in serum using the modified kinetic method of Henry, (1974) by using kit supplied by human, Germany.

### **Determination of serum alkaline phosphatase (ALP):**

ALP was carried out according to the method of Moss (1982).

### **Kidney functions**

#### **Determination of urea nitrogen**

Urea was determination in serum using the modified kinetic method or liquicolor of Patton and crouch, (1977) by using kit supplied by Human, Germany.

#### **Determination of creatinine**

Serum creatinine was measured using the modified kinetic method according to Schirmeister, (1964) by using kit supplied by Human, Germany.

#### **Determination of uric acid**

Serum uric acid was measured using the modified kinetic method according to While *et al.*, (1970) by using kit supplied by Human, German.

#### Lipids profile

#### **Determination of total cholesterol (T.C)**

Serum cholesterol was measured using the modified kinetic according to **Richmond**, (1973) by using kit supplied by Hu Germany. **Determination of triglycerides** (T.G)

Serum triglycerides (T.G) were measured using the modified kinetic method according to the method described by **Fossati and Prencipe (1982)** by using kit supplied by Spinreact, spain.

#### **Determination of high density lipoprotein cholesterol (HDL-c)**

Serum high density lipoprotein cholesterol (HDL-c) was measured using the modified kinetic method according to Allain, (1974) by using kit supplied by Human, Germany.

#### Determination of very low density lipoprotein cholesterol (VLDL-c)

Serum very low density lipoprotein cholesterol (VLDL-c) was calculated as mg/dl according to Lee and Nieman, (1996) equation: VLDL-c Concentration mg/dl = TG/5

### Determination of low density lipoprotein cholesterol (LDL-c)

Serum low density lipoprotein cholesterol (LDI-c) was calculated as mg/dl according to **Castelli** *et al.*, (1977) equation:

#### LDL Concentration mg/dl = Total Cholesterol – HDL-c – VLDL-c Statistical analysis

The data were analyzed using a completely randomized factorial design **SAS**, (1988) when a significant main effect was detected; the means were separated with the Student-Newman-Keuls Test. Differences between treatments of ( $P \le 0.05$ ) were considered significant using Costat Program. Biological results were analyzed by One Way ANOVA.

#### **Results and Discussion**

# Effect of sweet potato root, leaves and their mixture on glucose levels of diabetic rats:

Data given in Table (1) show the effect of sweet potato root, leaves and their mixture as powder on glucose of diabetic rats. It is evident that there are significant differences between negative control group and positive control group, the mean values were 95.75 and 236.0 mg/dl, respectively.

The lowest glucose level of treated groups (diabetic) recorded for diabetic group rats fed on 5% mixture powder. While, the highest value recorded for diabetic group rats fed on 2.5% SPL powder with significant difference ( $P \le 0.05$ ), the mean values were 125.50 and 152.75 mg/dl, respectively. These results are in agreement with that of **Akhtar** *et al.*, (2018), they concluded that anti-diabetic potential of white skinned sweet potato (WSSP) extract is due to the presence of bioactive compounds like glycoprotein, anthocyanins, alkaloids and flavonoids, which act as insulin-like molecules or insulin secretagogues constituents in sweet potatoes peel-off and these anti-diabetic proteins were extracted out in more concentration in methanol due to its organic nature.

Also, **Oki** *et al.*, (2011) indicated that sweet potato, containing 5% of this distinct glycoprotein, could effectively reduce fasting blood glucose levels, increase glucose and insulin sensitivity, and finally, improve insulin resistance in diabetic rats.

| Parameters        | Glucose level             |
|-------------------|---------------------------|
| Groups            | (mg/dl)                   |
| Control group (-) | 95.75 <sup>t</sup> ±0.21  |
| Control group (+) | $236.0^{a}\pm1.10$        |
| SPL (2.5%)        | 152.75 <sup>b</sup> ±.14  |
| SPL (5%)          | $147.50^{\circ}\pm 2.02$  |
| SPR (2.5%)        | $148.25^{\circ} \pm 1.05$ |
| SPR (5%)          | $137.25^{d} \pm 1.13$     |
| Mixture (2.5%)    | $136.85^{d} \pm 1.26$     |
| Mixture (5%)      | $125.50^{e} \pm 1.30$     |
| LSD (P≤0.05)      | 4.150                     |

 

 Table (1): Effect of different levels of sweet potato root, leaves and their powder mixture on glucose of diabetic rats

**SPR=** Sweet potato root SPL= Sweet potato leaves.

Each value represents the mean  $\pm$  SD of three replicates.

Mean under the same column bearing different superscript letters are different significantly ( $p \le 0.05$ ).

# Effect of sweet potato root, leaves and their mixture on liver functions of diabetic rats:

Data presented in Table (2) show the effect of sweet potato root, leaves and their powders mixture on liver functions (ALT, AST and ALP) of diabetic rats. It is clear to notice that ALT liver enzyme showed a significant difference between negative and positive control group. The mean values were 32.00 and 90.50 U/L, respectively.

The lowest ALT enzyme of treated group recorded for diabetic group rats fed on 5% powders mixture. While, the highest value was recorded for diabetic group rats fed on 2.5% SPL powder with significant difference ( $P \le 0.05$ ), the mean values were 39.50 and 75.00 U/L, respectively.

In case of AST data showed that there was a significant difference between negative control group and positive control groups. The mean values were 20.50 and 85.00 IU/L, respectively.

The lowest AST enzyme of treated group recorded for diabetic group rats fed on 5% powders mixture. While, the highest value recorded for diabetic group rats fed on 2.5% SPL powder with significant difference (P $\leq$  0.05), the mean values were 25.50 and 63.00 U/L, respectively.

It is evident that ALP liver enzyme showed a significant difference between negative control group and positive control groups. The mean values were 34.90 and 77.13 IU/L, respectively.

The lowest ALP enzyme of treated groups recorded for diabetic group rats fed on 5% mixture powder. While, the highest value recorded was for diabetic group rats fed on 2.5% SPR powder with significant difference ( $P \le 0.05$ ), the mean values were 56.68 and 50.03 U/L, respectively. The obtained data are agreement with **Marchesini** *et al.*, (2001), who reported that enzyme level of transaminases increased in diabetic rats treated with streptozotocin. Increase in their levels indicates their activeness in the absence of insulin. These result in increased availability of amino acids in diabetic as well as increase in glucogenesis and ketogenesis observed in diabetes. Also, they reported that dministration of aqueous sweet potato leaf extract and metformin result in lowering the levels of ALT, AST, and ALP to levels comparable to the control groups. The liver plays an important role in maintenance of normal glucose levels during fasting as well as in the postprandial period and its role in the pathogenesis of type 2 diabetes has attracted much

interest. Indeed, hepatic dysfunction resulting from insulin-resistance syndrome may lead to development of type 2 diabetes.

 

 Table (2): Effect of different levels of sweet potato leaves, roots and their powders, mixture on liver functions level of diabetic rats

| Parameters        | ALT                      | AST                      | ALP                      |
|-------------------|--------------------------|--------------------------|--------------------------|
| Groups            | (U/L)                    | (U/L)                    | (U/L)                    |
| Control group (-) | 32.0 <sup>h</sup> ±1.25  | $20.50^{h} \pm 1.15$     | 34.90 <sup>g</sup> ±1.10 |
| Control group (+) | 90.50 <sup>a</sup> ±1.13 | $85.00^{a} \pm 1.11$     | 77.13 <sup>a</sup> ±1.13 |
| SPL (2.5%)        | 75.00 <sup>b</sup> ±1.20 | $63.00^{b} \pm 1.21$     | $63.80^{b} \pm 1.15$     |
| SPL (5%)          | $67.00^{d} \pm 1.15$     | $57.50^{\circ} \pm 1.14$ | 54.03°±1.21              |
| SPR (2.5%)        | 69.50 <sup>c</sup> ±1.10 | $42.20^{d} \pm 1.15$     | 64.7 <sup>b</sup> ±1.14  |
| SPR (5%)          | 54.50 <sup>e</sup> ±1.23 | $34.90^{e} \pm 1.12$     | $51.74^{d} \pm 1.16$     |
| Mixture (2.5%)    | $42.40^{f} \pm 1.14$     | $28.00^{f} \pm 1.15$     | 43.60 <sup>e</sup> ±1.40 |
| Mixture (5%)      | 39.50 <sup>g</sup> ±1.15 | 25.50 <sup>g</sup> ±1.10 | $38.00^{f} \pm 1.15$     |
| LSD (P≤0.05)      | 2.144                    | 1.425                    | 1.046                    |

SPR= Sweet potato root SPL= Sweet potato leaves.

Each value represents the mean  $\pm$  SD of three replicates.

Mean under the same column bearing different superscript letters are different significantly ( $p \le 0.05$ ).

## Effect of sweet potato root, leaves and their mixture on serum total cholesterol and triglycerides of diabetic rats:

Data given in Table (3) showed the effect of sweet potato root, leaves and their mixture as powder on serum total cholesterol and triglycerides of diabetic rats. It is clear to mention that, there is a significant difference between negative control group and positive control group in total cholesterol levels. The mean values were 84.00 and 167.50 mg/dl, respectively.

The lowest total cholesterol of treated groups recorded for diabetic group rats fed on 5% mixture powder. While, the highest value recorded for diabetic group rats fed on 2.5% SPL powder with a significant difference (P $\leq$ 0.05), the mean values were 85.10 and 118.25 mg/dl, respectively.

In case of triglycerides, data indicated that there are a significant difference between negative control group and positive control group. The mean values were 136.50 and 72.50 mg/dl, respectively.

The lowest triglycerides of treated groups were that recorded for diabetic group rats fed on 5% mixture powder. While, the highest value

was recorded for diabetic group rats fed on 2.5% SPL powder with significant difference ( $P \le 0.05$ ), the mean values were 71.50 and 101.60 mg/dl, respectively. These results are in agreement with **Gad and George**, (2009), they reported that the total cholesterol in diabetic rats increased when compared to the control and metformin treated rats (significantly). They mentioned that possibly due to increase in mobilization of free fatty acids from peripheral fat deposited.

Also, **Rafiu and Luka** (2018) who reported that administration of aqueous sweet potato leaf extract reduced the serum total cholesterol, TG and LDL concentrations while it significantly increased ( $P \le 0.05$ ) the concentration of HDL (good cholesterol). Aqueous sweet potato leaf extract reverse significantly ( $P \le 0.05$ ) these abnormalities observed in lipid metabolism.

| Parameters Groups | Total cholesterol (mg/dl) | Triglycerides (mg/dl)     |
|-------------------|---------------------------|---------------------------|
| Control group (-) | 84.00 <sup>e</sup> ±1.13  | 72.50 <sup>g</sup> ±1.12  |
| Control group (+) | $167.50^{a} \pm 1.15$     | 136.50 <sup>a</sup> ±1.13 |
| SPL (2.5%)        | $118.25^{b} \pm 1.10$     | $101.60^{b} \pm 1.10$     |
| SPL (5%)          | $100.10^{d} \pm 1.14$     | $88.80^{d} \pm 1.15$      |
| SPR (2.5%)        | 105.75 <sup>c</sup> ±1.13 | 93.75 <sup>c</sup> ±1.12  |
| SPR (5%)          | $97.50^{d} \pm 1.16$      | 80.95 <sup>e</sup> ±1.11  |
| Mixture (2.5%)    | $86.05^{e} \pm 1.11$      | 76.25 <sup>f</sup> ±1.14  |
| Mixture (5%)      | $85.10^{e} \pm 1.10$      | 71.50 <sup>g</sup> ±1.13  |
| LSD (P≤0.05)      | 3.350                     | 3.108                     |

 Table (3): Effect of different levels of sweet potato leaves, root and their powders mixture on total cholesterol and triglycerides level of diabetic rats

SPR= Sweet potato root SPL= Sweet potato leaves.

Each value represents the mean  $\pm$  SD of three replicates.

Mean under the same column bearing different superscript letters are different significantly ( $p \le 0.05$ ).

# Effect of sweet potato leaves, roots and their mixture on lipid profile of diabetic rats

Data presented in Table (4) show the effect of sweet potato leaves, roots and their mixture as powder on high density lipoprotein cholesterol (HDL<sub>-C</sub>), low density lipoprotein cholesterol (LDL<sub>-C</sub>) and very low density lipoprotein cholesterol (VLDL<sub>-C</sub>) of diabetic rats. It is evident

that, high density lipoprotein cholesterol (HDL-c) showed significant differences between negative control group and positive control group. The mean values were 50.00 and 33.80 mg/dl, respectively.

The highest HDL-c of treated groups recorded for diabetic group rats fed on 5% powder mixture. While, the lowest value recorded for diabetic group rats fed on 2.5% SPL powder with significant difference (P $\leq$  0.05), the mean values were 51.65 and 37.50 mg/dl, respectively.

In case of low density lipoprotein cholesterol (LDL-c) levels, data indicated that there are significant differences between negative control group and positive control group. The mean values were 19.50 and 106.40 mg/dl, respectively.

The lowest LDL-c of treated groups recorded for diabetic group rats fed on 5% mixture powder. While, the highest value recorded for diabetic group rats fed on 2.5% SPL powder with significant difference (P $\leq$  0.05), the mean values were 19.15 and 60.43 mg/dl, respectively.

On the other hand, there were significant differences between negative control group and positive control group in very high density lipoprotein cholesterol (VLDL-c). The mean values were 14.50 and 27.30 mg/dl, respectively.

The lowest VLDL-c of treated groups was recorded for diabetic group rats fed on 5% mixture powder. While, the highest value was recorded for diabetic group rats fed on 2.5% SPL powder with a significant difference ( $P \le 0.05$ ), the mean values were 14.30 and 27.30 mg/dl, respectively. These results are in agreement with Ludvik *et al.*, (2004) who mention that HDL-c plays a defensive function against atherosclerosis because of its function in reverse cholesterol transport. HDL-c is also connected with metabolism of triglyceride-rich lipoprotein by stimulating lipoprotein lipase.

Also, **Jaleel** *et al.*, (2005) who found that elevated action of lipid markers is connected with insulin resistance, metabolic disorder, and type 2 diabetes mellitus and beneficial effects of methanol extract of sweet potato in diabetic rats.

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| Table (4): Eff | fect of different | i levels of s | weet potato     | leaves, root  | and |
|----------------|-------------------|---------------|-----------------|---------------|-----|
| their          | powders mixtu     | re on lipid j | profile level ( | of diabetic r | at  |

| Parameters<br>Groups | HDL-c (mg/dl)             | LDL ( mg/dl)             | VLDL (mg/dl)             |
|----------------------|---------------------------|--------------------------|--------------------------|
| Control group (-)    | 50.00 <sup>a</sup> ±1.13  | $19.50^{g} \pm 1.11$     | $14.50^{e} \pm 1.14$     |
| Control group (+)    | $33.80^{d} \pm 1.15$      | $106.40^{a} \pm 1.15$    | 27.30 <sup>a</sup> ±1.15 |
| SPL (2.5%)           | $37.50^{d} \pm 1.10$      | 60.43 <sup>b</sup> ±1.13 | $20.32^{b}\pm1.17$       |
| SPL (5%)             | 45.50 <sup>bc</sup> ±1.13 | $38.41^{d} \pm 1.12$     | $16.19^{cd} \pm 1.14$    |
| SPR (2.5%)           | $40.50^{\circ} \pm 1.16$  | $46.50^{\circ} \pm 1.16$ | 18.75 <sup>c</sup> ±1.16 |
| SPR (5%)             | 47.25 <sup>b</sup> ±1.12  | 32.49 <sup>e</sup> ±1.14 | $17.76^{c} \pm 1.10$     |
| Mixture (2.5%)       | $48.70^{b} \pm 1.14$      | $22.10^{f} \pm 1.12$     | $15.25^{d} \pm 1.15$     |
| Mixture (5%)         | 51.65 <sup>a</sup> ±1.11  | 19.15 <sup>g</sup> ±1.10 | $14.30^{e} \pm 1.14$     |
| LSD (P≤0.05)         | 2.640                     | 2.485                    | 1.125                    |

SPR= Sweet potato root SPL= Sweet potato leaves.

Each value represents the mean  $\pm$  SD of three replicates

Mean under the same column bearing different superscript letters are different significantly (p $\leq$ 0.05).

# Effect of sweet potato leaves, root and their mixture on kidney functions of diabetic rats

The effect of sweet potato leaves; root and their mixture as powder on kidney functions (serum uric acid, serum urea and creatinine) of diabetic rats are shown in Table (5). It is clear to notice that the serum uric acid of positive control group recorded higher value when compared with negative control group with a significant difference. The mean values were 10.10 and 6.00 mg/dl, respectively.

On the other hand, the highest serum uric acid level of treated groups recorded for diabetic group rats fed on 2.5% SPL powder. While, the lowest value recorded for diabetic group rats fed on 5% powder mixture with significant difference ( $P \le 0.05$ ), the mean values were 8.30 and 6.85 mg/dl, respectively.

In case of serum urea, data indicated that the positive control group recorded higher value when compared with negative control group with significant differences. The mean values were 36.70 and 21.35 mg/dl, respectively.

On the other hand, the highest serum urea level of treated group recorded for diabetic group rats fed on 2.5% SPL powder. While, the lowest value recorded for diabetic group rats fed on 5 % mixture powder

with significant difference (P $\le 0.05$ ), the mean values were 32.25 and 23.28 mg/dl, respectively.

On the other hand, the value of serum creatinine of positive control group was recorded higher value when compared with negative control group with significant differences. The mean values were 1.65 and 1.00 mg/dl, respectively.

While, the highest serum creatinine level of treated group recorded for diabetic group rats fed on 2.5% SPL powder. While, the lowest value recorded for diabetic group rats fed on 5 % powder mixture with significant difference ( $P \le 0.05$ ), the mean values were 1.43 and 1.05 mg/dl, respectively. These results are in agreement with **Sayed**, (2012), who reported that an increase in serum urea, creatinine, uric acid, and urine albumin was disrupted by diabetes induction in the positive control group. This result is consistent with the fact that streptozitocin induced diabetes leads to diabetic nephropathy.

Also, **Pal** *et al.*, (2015) they mentioned that the kidney functions were also improved in the aqueous fraction of *I. batatas* treated group as nearly 5.99 and 18.9 % decline in serum urea levels on day10 and 30, respectively, 13.5 and 50.1% decline in serum uric acid levels and 8.79 and 21.9% decline in serum creatinine levels were observed on day10 and 30, respectively in the aqueous fraction of *I. batatas* treated group.

| Parameters        | Uric acid               | Urea                     | Creatinine              |  |
|-------------------|-------------------------|--------------------------|-------------------------|--|
| Groups            | (mg/dl)                 | (mg/dl)                  | (mg/dl)                 |  |
| Control group (-) | $6.00^{d} \pm 1.14$     | $21.35^{g}\pm1.12$       | $1.00^{\circ} \pm 1.13$ |  |
| Control group (+) | $10.10^{a} \pm 1.12$    | $36.70^{a} \pm 1.10$     | $1.65^{a} \pm 1.10$     |  |
| SPL (2.5%)        | $8.30^{b} \pm 1.10$     | $32.25^{b} \pm 1.13$     | $1.43^{a} \pm 1.12$     |  |
| SPL (5%)          | $7.70^{b} \pm 1.15$     | $28.55^{d} \pm 1.16$     | $1.21^{b} \pm 1.14$     |  |
| SPR (2.5%)        | $8.00^{b} \pm 1.13$     | $30.60^{\circ} \pm 1.12$ | $1.35^{a} \pm 1.15$     |  |
| SPR (5%)          | $7.00^{\circ} \pm 1.11$ | 25.41 <sup>e</sup> ±1.15 | $1.13^{b} \pm 1.13$     |  |
| Mixture (2.5%)    | 7.25 <sup>c</sup> ±1.12 | $30.70^{\circ} \pm 1.11$ | $1.11^{b} \pm 1.11$     |  |
| Mixture (5%)      | $6.85^{\circ} \pm 1.16$ | $23.28^{f} \pm 1.14$     | $1.05^{b} \pm 1.15$     |  |
| LSD (P≤0.05)      | 1.024                   | 1.615                    | 0.350                   |  |

 

 Table (6): Effect different levels of sweet potato leaves, roots and their powder mixture on kidney functions of diabetic rats

SPR= Sweet potato root SPL= Sweet potato leaves.

Each value represents the mean  $\pm$  SD of three replicates.

Mean under the same column bearing different superscript letters are different significantly (p≤0.05).

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## Journal of Home Economics, Volume 30, Number (1), 2020 التأثيرات المحتملة لتناول أوراق وجذور البطاطا ومخلوطهم معا على الفئران المصابة بالسكرى المستحث بالألوكسان

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الملخص العربي:

اجريت هذه الدراسة لمقارنة تأثير مسحوق أوراق وجذور البطاطا ومخلوطهم معا. تم استخدام ثمانية وأربعين من ذكور فئران الألبينو البالغين في هذه الدراسة ، وكان وزن الفئران (150 ± 10 جم) وتم تقسيمهم إلى ثماني مجموعات كل مجموعه بها سنة فئران منهم مجموعة ضابطة سالبة ومجموعة ضابطة موجبة ، وتم اصابة الفئران بمرض السكر عن طريق الحقن بمادة الألوكسان (بتركيز 150مجم/ كجم). تمت إضافة مسحوق أوراق وجذور البطاطا ومخلوطهم معا بنسبة 2,5 ٪ , 5 ٪ من النظام الغذائي الأساسي. تم تقدير (الكوليستيرول الكلى ، الجلسريدات الثلاثية ، الكولستيرول مرتفع الكثافة، الكولستيرول منخفض الكثافة ، الكولستيرول منخفض الكثافة جدا ، الجلوكوز في الدم ، أنزيمات الكبد في مصل الدم (ALT, AST and ALP) ، وظائف الكلى (الكرياتينين وحمض اليوريك واليوريا). من النتائج التي تم الحصول عليها تبين أن التغذية على مسحوق أوراق وجذور البطاطا ومخلوطهم معا أدى إلى حدوث زيادة كبيرة معنوية(P≤0.05) في مستوى HDL-c، بينما انخفضت مستويات LDL-c, VLDL-c بنسبة عالية مع وجود فرق معنوي. كذلك حدث انخفاض معنوى في كلا من وظائف الكلي ووظائف الكبد وانخفاض معنوى في مستوى الجلوكوز في الدم ، الذي يعكس تأثير علاجي مصاحب للتغذية على مسحوق أوراق وجذور البطاطا ومخلوطهم لعلاج مرض السكري في الفئران. وكانت أفضل نتيجة لتركيز مسحوق مخلوط أوراق وجذور البطاطا بتركيز 5 ٪.

الكلمات المفتاحية: البطاطا ، مرض السكر, الفئران, التحاليل الكيميائية الحيوية.